Sari Edelstein

# *Life Cycle* Nutrition

## An Evidence-Based Approach

SECOND EDITION

# *Life Cycle* Nutrition

## An Evidence-Based Approach

**SECOND EDITION**


Editor

**Sari Edelstein, PhD, RD**

Department of Nutrition
Simmons College
Boston, Massachusetts



JONES & BARTLETT
LEARNING

*World Headquarters*
Jones & Bartlett Learning
5 Wall Street
Burlington, MA 01803
978-443-5000
info@jblearning.com
www.jblearning.com

Jones & Bartlett Learning books and products are available through most bookstores and online booksellers. To contact Jones & Bartlett Learning directly, call 800-832-0034, fax 978-443-8000, or visit our website, www.jblearning.com.

Substantial discounts on bulk quantities of Jones & Bartlett Learning publications are available to corporations, professional associations, and other qualified organizations. For details and specific discount information, contact the special sales department at Jones & Bartlett Learning via the above contact information or send an email to specialsales@jblearning.com.



**Production Credits**
Chief Executive Officer: Ty Field
President: James Homer
Chief Product Officer: Eduardo Moura
Executive Publisher: William Brottmiller
Executive Editor: Rhonda Dearborn
Editorial Assistant: Sean Fabery
Associate Director of Production: Julie C. Bolduc
Production Editors: Jessica Steele Newfell, Louis C. Bruno, Jr.
Senior Marketing Manager: Andrea DeFronzo
Art Development Editor: Joanna Lundeen
Illustrations: diacriTech Publisher Services
VP, Manufacturing and Inventory Control: Therese Connell
Composition: diacriTech Publisher Services
Cover Design: Theresa Day
Photo Research and Permissions Coordinator: Amy Rathburn
Cover Image: © VikaSuh/ShutterStock, Inc.
Printing and Binding: Edwards Brothers Malloy
Cover Printing: Edwards Brothers Malloy

**To order this product, use ISBN: 978-1-284-03667-1**

**Library of Congress Cataloging-in-Publication Data**
Life cycle nutrition : an evidence-based approach / [edited] by Sari Edelstein. —Second edition.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-4496-9430-2 (pbk. : alk. paper)
I. Edelstein, Sari, editor of compilation.
[DNLM: 1. Nutritional Physiological Phenomena. 2. Evidence-Based Medicine. 3. Human Development—physiology. 4. Longitudinal Studies. QU 145]
QP141
612.3'9—dc23 2013038802

6048
Printed in the United States of America
18 17 16 15 14 10 9 8 7 6 5 4 3 2 1

To my wonderful colleagues at Simmons College:
Nancie Herbold, Elizabeth Metallinos-Katsaras, Teresa Fung, Lisa Brown, and Leah Smith


# Brief Contents


# Contents


# Contributors

**Ben Atkinson, MS, RD, CD**
Harborview Medical Center

**Jennifer Blair, RD, CSP, LDN**
The Children's Hospital of Philadelphia

**Claire Blais, RD, CDE, LDN**
Partners Healthcare

**Lisa S. Brown, PhD, RD**
Simmons College

**Jennifer L. Bueche, PhD, RD, CDN**
State University of New York

**Jessica Burfield, RD, LDN**
The Children's Hospital of Philadelphia

**Emily Burritt, MS, RD, CNSC**
Miller Children's Hospital Long Beach

**Victoria Hammer Castellanos, PhD, RD**
University of Scranton

**Cynthia Chandler, RD, LD, CDM**
Sullivan University

**Karen Chapman-Novakofski, RD, LDN, PhD**
University of Illinois at Urbana-Champaign

**Ronnie Chernoff, PhD, RD, FAND, FASPEN**
Central Arkansas Veterans Healthcare System

**Harriet H. Cloud, MS, RD, FADA**
Nutrition Matters

**Rachel Colchamiro, MPH, RD, LDN**
Massachusetts Department of Public Health

**Sharon Collier, RD, LD, M Ed**
Boston Children's Hospital

**Julia Driggers, RD, LDN, CNSC**
The Children's Hospital of Philadelphia

**Karen M. Funderburg, MS, RD, LD**
University of Oklahoma Health Sciences Center

**Stefanie A. Giampa, MS, RD, LDN**
Bearhill Rehabilitation and Nursing Center

**Ellen Glovsky, PhD, RD, LDN**
Northeastern University

**Karlyn Grimes, MS, RD, LDN**
Simmons College

**Laura Harkness, PhD, RD**
PepsiCo

**Edna Harris-Davis, MS, MPH, RD, LD**
Morehouse School of Medicine

**Roschelle Heuberger, PhD, RD**
Central Michigan University

**Pamela S. Hinton, PhD**
University of Missouri

**Christie Hussa, MBA, RD, LDN**
The Children's Hospital of Philadelphia

**Phuong Huynh, MS, RD, CSP**
Children's Hospital and Research Center Oakland

**Sari Kalin, MS**
Blue Cross Blue Shield of Massachusetts

**Jan Kallio, MS, RD, LDN**

**Kristen Kochenour, BS**
WW Hospitality Marketing

**Jeannine Lawrence, PhD, RD, LD**
The University of Alabama

**Anne R. Lee, MS Ed, RD**
Columbia University

**Jessica Brie Leonard, RD**

**Rachelle Lessen, MS, RD, IBCLC**
The Children's Hospital of Philadelphia

**Julie O'Sullivan Maillet, PhD, RD**
Rutgers University

**Alison Mallowe, MA, RD, LDN**
The Children's Hospital of Philadelphia

**Reed Mangels, PhD, RD, LD**
University of Massachusetts Amherst

**Virginia L. Marchant-Schnee, BS**

**Migy K. Mathews, MD**
University of Oklahoma Health Sciences Center

**Elizabeth Metallinos-Katsaras, PhD, RD**
Simmons College

**Yeemay Su Miller, MS, RD**
MOVE Fitness

**Shideh Mofidi, MS, RD, CSP**
Maria Fareri Children's Hospital

**Courtney Moskal, MS, RD**

**Theresa A. Nicklas, DrPH**
Baylor College of Medicine

**Liesje Nieman, RD, CNSD, LDN**
The Children's Hospital of Philadelphia

**Carol E. O'Neil, PhD, MPH, LDN, RD**
Louisiana State University

**Aaron Owens, MS, RD**
Seattle Children's Hospital

**Rebecca Randall, MS, RD, LDN**

**Barbara Robinson, MPH, RD, CNSC, LDN**
Johnson and Wales University

**Jennifer Sabo, RD, LDN, CNSD**
The Children's Hospital of Philadelphia

**Angela Sader, RD, LD, MBA**
SPCR Management

**Judith Sharlin, PhD, RD**
Palm Coast University

**Amy Sheeley, PhD, RD, LDN**
Office of Elderly Affairs, Massachusetts

**Ashley Smith, DVM, MS, RD, LD**
University of North Carolina Hospital

**Sara Snow, MS, RD**
Case Western Reserve University

**Inger Stallmann, MS, RD, LD**
Medical College of Georgia

**Christina Taddei, MSF, RD, LDN**
Plus One Health Management, Inc.

**Ancy Thomas, MS, RD, CSP, LDN**
The Children's Hospital of Philadelphia

**I. David Todres, MD**
Massachusetts General Hospital
Harvard Medical School

**Margaret Udahogora, PhD, RD**
University of Maryland

**Stella Lucia Volpe, PhD, RD, LDN, FACSM**
Drexel University

**Georgianna Walker, MS, RD, LRD**
University of North Dakota



*Life Cycle Nutrition: An Evidence-Based Approach, Second Edition* provides a unique learning experience, reference, and start for students learning about nutrition throughout the life cycle. It also provides a comprehensive reference for those of us already in practice. The book stands alone in its interwoven coverage of public health nutrition with subjects as diverse as media influences on eating, skipping breakfast, sociodemographic moderators of dietary intake, tobacco use and nutritional status, and clinical nutrition. It includes a wide array of diverse topics including parenteral nutrition and biochemical monitoring in neonates, inborn errors of metabolism, and cancer. Contemporary issues such as fruit juice consumption, nutritional needs of athletes, and dietary supplements as ergogenic aids are addressed across the life cycle as well as by using a multidisciplinary approach. This book gives students current information, helps them evaluate emerging information, and prepares them to uncover new information for the public, their clients, and themselves as they journey together through the life cycle.

The book is divided into two sections, the first of which focuses on prenatal to adolescent nutrition. The first chapter covers pregnancy and breastfeeding. Chapter 2 gives students insight into the growth and development of normal infants, along with some of their nutrition "issues" such as food safety and the effect of early diet on health outcomes. As outlined in Chapter 3, toddlers have different issues as they begin to explore their world and express food preferences; they start forming food habits and are influenced by caregiver behaviors. Chapter 4 emphasizes that school-aged children have different needs and are influenced by a wide variety of outside forces including role models as well as television and other media. Although caregivers have a large influence, school-aged children begin to make their own food choices and may be grazers or picky eaters. Adolescents, discussed in Chapter 4, are an understudied group with many nutrition issues; they, too, are influenced not only by media but also by their peers. Adolescents also make many of their own food choices and may skip breakfast or consume fast food and added sugars, often in the form of sweetened beverages. Poor food choices contribute to increasing obesity and the appearance of nutrition-related chronic diseases formerly seen only in adults such as metabolic syndrome and type 2 diabetes. As students learn about the nutritional needs of infants, children, and adolescents, they learn how to help these groups improve their nutritional status.

Chapters 5 through 8 discuss the special nutrition considerations of infants, children, and adolescents. Eating disorders, failure to thrive, food allergies, and the nutrition needs of children with disabilities are covered in these chapters. Pediatric vegetarianism, childhood obesity, and the dietary needs of athletes are highlighted. Very specialized topics such as inborn errors of metabolism and nutrition support of the neonate are included.

Section 2 covers adult nutrition. Chapters 9 through 11 include information on chronic, nutrition-related diseases such as coronary heart disease, hypertension, diabetes, kidney disease, cancer, osteoporosis, HIV/AIDS, and obesity. Evidence analysis for evidence-based practice in these diseases is included as are prevention strategies. Chapter 11 is devoted to physical activity and weight management issues. Chapters 12 and 13 are dedicated to nutritional issues of the elderly. All topics ranging from special nutritional needs to nutritional problems, from activities of daily living to polypharmacy, and from risks of malnutrition to nutrition intervention are included in these chapters. Chapters 14 and 15 discuss professionalism and ethical issues, the final preparation for students to join nutritionists as colleagues.

## New to This Edition

The *Second Edition* of *Life Cycle Nutrition: An Evidence Based Approach* has been carefully updated to mirror current findings and features a wealth of new information in each chapter. Additionally, new case studies were added to each chapter, with answers available in the Instructor's Manual.

Among the changes incorporated into this cutting-edge edition are the following:

### Chapter 1

- Added information on epigenetics
- Expanded the section on lactation

### Chapter 2

- Expanded the section on breast milk composition
- Incorporated WHO Guidelines
- Updated the American Academy of Pediatrics' section on Breastfeeding 2012 Recommendations
- Included information from the Feeding Infants and Toddlers Study (FITS)
- Added a new section on barriers to breastfeeding

### Chapter 3

- Added the American Heart Association Guidelines for Young Children
- Utilized WHO Growth Charts
- Provided the National Institutes of Health approaches to pediatric obesity

### Chapter 4

- Added a full range of Public Health Nutrition program updates
- Updated to Healthy People 2020 recommendations

### Chapter 5

- Added a section on inborn errors of metabolism that covers these disorders:
  - 3-methylcrotonyl-coA carboxylase deficiency
  - methylmalonic acidemia
  - fatty acid oxidation disorders
  - pyruvate dehydrogenase deficiency

### Chapter 6

- Revised the vitamin and mineral sections, including the following areas:
  - role of folate in Neural Tube Defects and Cardiac Problems
  - role of vitamin C in Respiratory Ailments
  - role for vitamin A in Child Health
  - role of phytates in plant foods in reducing zinc absorption
  - changes in the Dietary Reference Intakes (DRIs) for calcium and vitamin D
- Updated the section on autism spectrum disorders

### Chapter 7

- Updated the section on vegetarian diets
  - Incorporated the Institute of Medicine's new Dietary Reference Intakes (DRIs) for calcium and vitamin D
- Added a section on the global rise in childhood obesity
- Updated the section on environmental influences on obese children
- Included dietary trends affecting obese children, including the following:
  - changes in the food environment
  - household food insecurity
  - prenatal influence
  - physical inactivity affecting obesity in children
  - opportunities to intervene and prevent
  - social programs

### Chapter 8

- Fully revised the section on dietary guidelines for athletes
  - Added a new section on the role of vitamin D in athletic performance
  - Enhanced the information about the role of dietary supplements
- Updated the section on pediatric diabetes
  - Incorporated information from the American Diabetes Association's new "Standards of Medical Care in Diabetes"
  - Included information about insulin resistance in pediatric type 2 diabetes
- Revised the section on disordered eating
  - Revised the information on barriers to treatment

### Chapter 9

- Revised to reflect the "Position of the American Dietetic Association: Weight Management"
- Incorporated the Institute of Medicine's new Dietary Reference Intakes (DRIs) for calcium and vitamin D

### Chapter 10

- Revised the information about diseases of older Americans including prevalence, screening guidelines, and diagnosis-related conditions
- Added new material about health programs for elder adults
- Utilized the Healthy People 2020 recommendations

### Chapter 11

- Included data from new obesity evidence-based studies
- Utilized the Healthy People 2020 recommendations
- Incorporated new exercise guidelines for older adults
- Added material about obesity in diverse groups

### Chapter 12

- Added findings from The National Institute on Aging and the Administration on Aging
- Incorporated the Mini-Nutritional Assessment of Older Persons
- Updated the sections on macro- and micronutrient needs
- Added a discussion of the impact of socioeconomics on healthy aging
- Revised the demographics of aging

### Chapter 13

- Utilized current national health statistics
- Provided updated information from the National Institute of Deafness and Other Communication Disorders
- Included references to The Elderly Nutrition Program

### Chapter 14

- Utilized the Academy of Nutrition and Dietetics new position paper "Individualized Nutrition Approaches for Older Adults in Health Care Communities"
- Incorporated the American Health Care Association's "The State of Long-Term Health Care Sector"

### Chapter 15

- Provided updates using the Academy of Nutrition and Dietetics 2008 position paper "Ethical and Legal Issues in Nutrition, Hydration and Feeding"
- Added the Academy of Nutrition and Dietetics "New Code of Ethics for the Profession of Dietetics and Process for Consideration of Ethical Issues"
- Utilized the American Society of Parenteral and Enteral Nutrition 2010 "A.S.P.E.N. Ethics Position Paper"

## Acknowledgments

I thank my graduate assistant at Simmons College, Melissa Przybysc, for her assistance, and I thank the staff at Jones & Bartlett Learning.

# How to Use This Book



This book has many exciting features that not only enhance its usefulness as a teaching tool but also expose future clinicians and scientists to the cutting edge of nutritional sciences.

**Evidence-Based Practice Sidebar.** This section contains articles where readers can transform the written word into a peer-reviewed study or clinical trial. I tell my students that I want to know their informed opinion. But how do they shape these opinions? Only by reading about evidence-based studies and medicine and by conducting studies themselves can students learn the importance of evidence-based practice. With the Evidence Analysis Library, the American Dietetic Association is a leader in presenting practitioners with the concept of evidence-based practice. This text complements this effort and enhances our students' familiarity with this important subject.

**Nitrates**

Infant methemoglobinemia results in cyanosis in infants with few other clinical symptoms and is caused by nitrates in food or water that are converted to methemoglobin-producing nitrites before or after ingestion. The resulting compound, methemoglobin, cannot bind oxygen and results in hypoxemia. Absorbed nitrate that has not been converted to nitrite can be readily excreted in the urine without adverse effects. The greatest risk to infants comes from well water contaminated with nitrates (Greer, Shannon, the Committee of Nutrition, & the Committee on Environmental Health, 2005). It is estimated that 2 million families drink water from private wells that fail to meet federal drinking water standards for nitrate, and 40,000 infants younger than 6 months old live in homes that have nitrate-contaminated water supplies. Breastfed infants whose mothers consume water with high nitrate nitrogen concentrations are not at increased risk because nitrate concentration does not increase in human milk.

Nitrates also occur naturally in plants and may be concentrated in foods such as green beans, carrots, squash, spinach, and beets. Some commercially prepared infant foods are voluntarily monitored for nitrate content, and because of exceedingly high levels in spinach, this product is often labeled as not to be used for infants younger than 3 months of age. Concerns for home-prepared foods are unfounded because there is no nutritional indication for introduction of complementary foods before 6 months. The risk of methemoglobinemia decreases with age as the infant's gastric pH approaches lower levels typical of later childhood and fetal hemoglobin, which more readily oxidizes to methemoglobin, is replaced by adult hemoglobin after 3 months.

**Cultural Diversity Sidebar.** Cultural differences that involve nutrition and health differences and similarities among ethnic groups are highlighted. This is a wonderful opportunity for students to learn more about what I call "diseases that discriminate": obesity, cardiovascular disease, and diabetes are all most common in minority populations. Why? Are differences genetic, or are they related to life-style or to health care? What are the gaps in nutrition research in different groups? How do you work with people from cultures/ethnicities that are different from your own to improve their health or nutritional status? This feature will help students answer these questions, develop professionalism, and improve their practice.

**The Cultural Diversity of Poverty**

Poverty is the most omnipresent of the social risk factors for failure to thrive. One study documented that 13% of patients with poor growth are homeless, which makes access to the medical care needed to prevent and correct malnutrition difficult (Frank & Zeisel, 1988). It is crucial in proper treatment of your patients that you consider their socioeconomic status in designing an individualized care plan.

**Critical Thinking Sidebar.** Found throughout the chapters, the points considered assist the reader in critical thinking concepts presented in different sections of the text. Perhaps the most important thing students can learn is to analyze and evaluate, examine and reason, reflect and decide. Why? So they can solve complex real-world problems, weigh evidence and make conclusions, learn to ask the right questions, and develop informed opinions to share with others.

**CRITICAL Thinking**

Though controversial, flavored milk can be a good source of calcium and may increase compliance in young children. It may be helpful to choose lower fat milk and add flavoring with sugar-free syrups as a way to keep fat and added sugar intake low for toddlers older than age 2 years and still receive the benefits of calcium.

**Learning Points.** These items call out particularly important points.

**Learning Point** Food-related behaviors are established early in life, and how and what an infant or toddler eats in the first years of life can influence later food choices.

**Case Studies.** These sections are provided to demonstrate chapter concepts. Case studies actively involve students in learning and simulate or represent actual problems they will face as professionals. Students can work alone or in groups to develop solutions as they would in the workplace. Thus, case studies help develop knowledge and skills of students in a wide variety of subjects and improve critical thinking, public speaking, and group interaction skills. Instructors can view the Case Study answers in the online Instructor's Manual.

**Case Study 3**

**Infant Nutrition**

Rachelle Lessen, MS, RD, IBCLC

Emory is a 6-week-old former full-term infant. Her mother is 30 years old, healthy, does not smoke, and this is her first baby. Emory has been exclusively breastfeeding since birth. She latched well from the beginning, and mom denies any difficulties or problems with sore nipples. Her output includes 10–11 wet diapers and 2 stools per day. Her mother reports that she breastfeeds more than 12 times per day and that her feedings are very long, typically more than 1 hour. Mom's goal is to breastfeed her for more than 1 year.

Emory's birth weight was 3.487 kg (75th percentile on the WHO growth chart). Her discharge weight from the hospital was 2.98 kg (10% below birth weight). At 1 week, she weighed 3.345 kg, and at 2 weeks she weighed 3.289 kg. Now at 6 weeks she weighs 3.52 kg (<2nd percentile on the WHO growth chart). She has gained 33 g since birth. Her head circumference and length are within normal.

**Questions**

1. What is the primary nutrition concern?
2. What is the most likely cause of the problem?
3. How would you improve her nutritional status while taking into consideration the mother's breastfeeding goals?

**Issues to Debate.** These issues include withholding and withdrawing nutrition, the ethical implications of nutritional care, and right-to-die case law. It is critical that students learn the physiologic, moral, ethical, and legal aspects of these emotionally charged issues. Debate and discussion with others help nutrition students understand these issues as they apply across the life span and how they will interact with other health professionals.

**Issues to Debate**

1. Discuss obstacles to breastfeeding that women encounter and possible public health strategies to overcome these challenges.
2. What are the effects of early feeding on the development of obesity and what can be done to reduce the increasing rates of childhood obesity?
3. Infant formula manufacturers add DHA and AA to their products. This has greatly raised the cost to consumers (including the U.S. government, which is the largest purchaser of formula because of the WIC program), yet studies fail to show long-term benefit of these additions. Discuss the ethical implications of this practice.
4. What are some of the cultural aspects that affect the transitioning from an all-milk infant diet to a diet of family foods?

**Reader Objectives** guide students stepwise through the chapter.

**Reader Objectives**

**After studying this chapter and reflecting on the contents, you should be able to**

1. Describe normal infant nutrition in the first year of life.
2. Compare growth differences between breastfed and formula-fed infants.
3. Describe the impact of early diet on later development of obesity, diabetes, and food allergies.
4. Discuss adequate intake of key nutrients in the first year of life, including energy, protein, fatty acids, iron, zinc, and vitamin D.
5. Describe caregiver behaviors that can affect normal transitioning from an all-milk infant diet to a diet of family foods.
6. Compare and contrast actual complementary feeding patterns with recommended guidelines.

**Key Terms** throughout the chapters assist with new terminology and concepts.

**Development of Growth Charts**

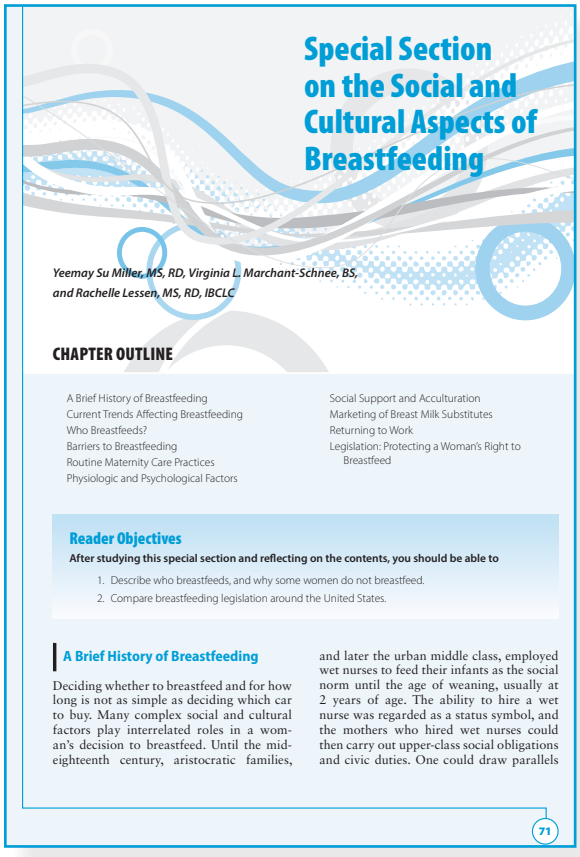
The nutritional status of children is assessed by plotting height and weight on **growth charts** to determine adequacy of nutrient intake, particularly calories and protein. There is considerable evidence to show that growth rates differ for breastfed and formula-fed infants.

**growth charts** Graphs used to assess nutritional status of children by plotting height and weight and comparing these to reference data.

**Chapter Summaries** crystallize the most important elements of the chapters and help bring the chapter contents into perspective.

> **Summary**
>
> In summary, the role for the RD in LTC settings is much more complex than it is in many other care settings. In LTC the dietitian is expected to be proficient in MNT and have expertise in the regulatory environment, food preparation, sanitation, dining, and survey and quality management. The provision of nutrition care in this setting also requires a proactive and system-oriented approach, where residents' quality of life is a primary consideration in every decision, and care should be provided in the most home-like manner possible. The potential for the RD to make a significant difference in the quality of life of residents and to be valued by the facility are limited only by the aspirations of the individual practitioner.

Following some chapters are **Special Sections**, which are designed to heighten curiosity and provide insight into particular issues.



Special Section on the Social and Cultural Aspects of Breastfeeding

*Yeemay Su Miller, MS, RD, Virginia L. Marchant-Schnee, BS, and Rachelle Lessen, MS, RD, IBCLC*

**CHAPTER OUTLINE**

A Brief History of Breastfeeding
Current Trends Affecting Breastfeeding
Who Breastfeeds?
Barriers to Breastfeeding
Routine Maternity Care Practices
Physiologic and Psychological Factors
Social Support and Acculturation
Marketing of Breast Milk Substitutes
Returning to Work
Legislation: Protecting a Woman's Right to Breastfeed

**Reader Objectives**

**After studying this special section and reflecting on the contents, you should be able to**

1. Describe who breastfeeds, and why some women do not breastfeed.
2. Compare breastfeeding legislation around the United States.

**A Brief History of Breastfeeding**

Deciding whether to breastfeed and for how long is not as simple as deciding which car to buy. Many complex social and cultural factors play interrelated roles in a woman's decision to breastfeed. Until the mid-eighteenth century, aristocratic families, and later the urban middle class, employed wet nurses to feed their infants as the social norm until the age of weaning, usually at 2 years of age. The ability to hire a wet nurse was regarded as a status symbol, and the mothers who hired wet nurses could then carry out upper-class social obligations and civic duties. One could draw parallels

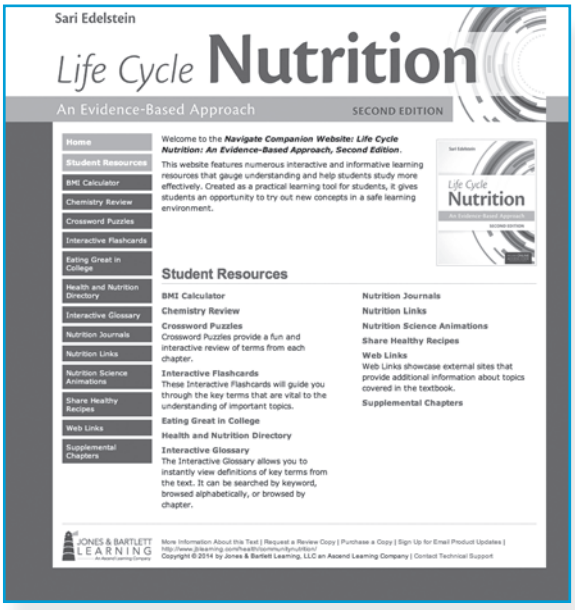
71

For the instructor, the following resources have been provided:

- A **Test Bank** featuring more than 400 multiple choice, true/false, and essay questions
- An **Instructor's Manual** containing answers to the case studies featured in the text
- **PowerPoint Presentations** including more than 15 slides per chapter

For the student, each new copy of this book comes with an access code for the **Navigate Companion Website**, which includes the following features:

- A supplemental chapter on interpreting evidence-based research
- Web links
- An interactive Glossary
- Animated Flashcards
- Crossword puzzles
- And more



Sari Edelstein

Life Cycle Nutrition

An Evidence-Based Approach SECOND EDITION

Home
Student Resources
BMI Calculator
Chemistry Review
Crossword Puzzles
Interactive Flashcards
Eating Great in College
Health and Nutrition Directory
Interactive Glossary
Nutrition Journals
Nutrition Links
Nutrition Science Animations
Share Healthy Recipes
Web Links
Supplemental Chapters

Welcome to the ***Navigate Companion Website: Life Cycle Nutrition: An Evidence-Based Approach, Second Edition***.

This website features numerous interactive and informative learning resources that gauge understanding and help students study more effectively. Created as a practical learning tool for students, it gives students an opportunity to try out new concepts in a safe learning environment.

**Student Resources**

**BMI Calculator**
**Chemistry Review**
**Crossword Puzzles**
Crossword Puzzles provide a fun and interactive review of terms from each chapter.
**Interactive Flashcards**
These Interactive Flashcards will guide you through the key terms that are vital to the understanding of important topics.
**Eating Great in College**
**Health and Nutrition Directory**
**Interactive Glossary**
The Interactive Glossary allows you to instantly view definitions of key terms from the text. It can be searched by keyword, browsed alphabetically, or browsed by chapter.
**Nutrition Journals**
**Nutrition Links**
**Nutrition Science Animations**
**Share Healthy Recipes**
**Web Links**
Web Links showcase external sites that provide additional information about topics covered in the textbook.
**Supplemental Chapters**



**Sari Edelstein, PhD, RD**

*Associate Professor of Nutrition at Simmons College*
**BS Florida State University**
**MS Florida International University**
**PhD University of Florida**

Dr. Sari Edelstein is an Associate Professor in the Nutrition and Dietetics Department at Simmons College. She presently teaches both Food Science and Food Service classes. Before joining the faculty at Simmons College, Dr. Edelstein was in private practice and served as a hospital Food Service Director and Chief Dietitian. She is the author of many research articles, inclusive of topics on ethics, yoga, the glycemic index, and athletic performance, as well as the author or editor of many books.

SECTION I

# Evidence-Based Nutrition in the Life Cycle: Prenatal to the Adolescent

# CHAPTER 1 Nutrition Requirements During Pregnancy

*Lisa S. Brown, PhD, RD*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Describe some of the preexisting conditions that can occur in pregnancy.
2. Discuss the differences in the nutritional needs of pregnancy as compared to the nonpregnant woman.
3. Define problems commonly associated with pregnancy.
4. Relate the impact of lifestyle behaviors on fetal development.

What a woman eats when she is pregnant can have profound and lasting effects on her child's health. The expression "you are what you eat" applies, but in this case, it is this: "You are what your mother eats." During the prenatal period, the fetus has the enormous task of evolving in only 9 short months from a single-celled, fertilized egg to a human infant. To accomplish this, the fetus must have all of the necessary resources available in the proper quantities and at the exact times they are needed. Despite the daunting nature of the task, mothers have been producing healthy infants for thousands of years, demonstrating the amazing adaptability of both the mother and her child. The capacity of the mother's body to create the necessary conditions for fetal growth is one of the great miracles of life. There are limits, however, and the health of the child may suffer in obvious and not so obvious ways if certain thresholds for nutrients are not met.

Although a pregnant body has an amazing ability to compensate for nutrient deficiencies and excesses, a woman cannot provide essential nutrients for her child if she herself is deficient in them. Many factors influence a mother's nutritional status during her pregnancy. The mother's own health before conception, her health during pregnancy, her lifestyle choices, and environmental exposures can all change what and how much she eats and limit precious nutrients available for the growing fetus.

It is important that knowledgeable healthcare providers are available to support the mother-to-be with strategies to help her achieve the most balanced diet possible, thus ensuring the health of both mother and child. This chapter examines normal prenatal nutritional requirements and common factors that may compromise the mother's ability to provide ideal nutrition for her growing fetus.

## Nutritional Status Prior to Pregnancy

The period when a woman is pregnant is often looked at in isolation and is not put in a larger context of the mother's overall health. Although this has been the traditional approach to pregnancy, there is a growing movement to look at human nutrition using the life course approach, which is promoted by the World Health Organization. Using the life course approach, the mother is followed from her own conception through death, and factors that have influenced her health since she herself was in utero are considered when assessing risk of developing chronic diseases for both the mother and her offspring (Darnton-Hill, Nishida, & James, 2004).

### The Fetal Origins Hypothesis

The mother's health before and during pregnancy may be affected by genetics, as well as malnutrition, acute and chronic disease, exposure to environmental toxins, and a number of other factors. The fetal origins hypothesis proposes that certain genes in the fetus may or may not be "turned on" depending on the environment to which the mother is exposed while pregnant (Hampton, 2004). For example, if a mother is exposed to severe food restriction during pregnancy, as was the case in the Dutch Famine during World War II, her developing fetus will adapt genetically to thrive in an environment of severe energy restriction. The child's genetic programming will be sensitized to store fat more efficiently compared with those not exposed to famine in utero. If the environmental conditions the child is born into do not match the famine conditions, the child will accumulate fat more quickly than desired and be at higher risk of obesity, diabetes, and cardiovascular disease. Findings from the Dutch Famine and other cohorts support the

fetal origins hypothesis, and research is ongoing to confirm the process by which it works (Roseboom, de Rooij, & Painter, 2006; Thompson, 2007).

More recently research has focused on the role of epigenetic changes as the mechanism that mediates gene expression in the fetus (Langley-Evans, 2013). *Epigenetics* refers to the control "switches" that determine gene expression and can vary even if the DNA sequence itself does not change. Although human DNA changes slowly, it is believed that we adapt relatively quickly to our environment through the process of epigenetic gene activation and deactivation. Humans are born with many genes that are never activated. The prenatal and early childhood environment interacts with an individual's genetic code to help select which genes will be useful in those specific environmental conditions (Godfrey, Lillycrop, Burdge, Gluckman, & Hanson, 2013).

In the World Health Organization life course approach to disease prevention, the passing of increased risk for chronic disease from mother to child based on prenatal environmental exposures is known as the intergenerational effect. The intergenerational effect is thought to be one reason behind the clustering of chronic disease risk factors in families of lower socioeconomic status. Women in lower socioeconomic classes are more likely to be exposed to extreme environmental conditions and have substandard health care, exacerbating problems. Interventions should seek to maximize the mother's health before and during pregnancy to improve the short- and long-term health of her children. There is also growing evidence that intervention in the first years of the child's life may affect long-term risk through modifying epigenetic programming (Lapillonne & Griffin, 2013).

### Maternal Preconception Weight Status

Many aspects of the mother's health and lifestyle before pregnancy have been shown to affect her subsequent pregnancies with potential to influence the health of her children, but one area of particular concern is the mother's weight before pregnancy. The dramatic increase of overweight and obesity in women in the United States has forced many healthcare providers to focus their counseling on weight management before and during pregnancy. Preconception obesity is associated with a substantial increase in risk for pregnancy complications, such as gestational diabetes and preeclampsia, as well as a significant increase in birth defects (Chu et al., 2007; Hauger, Gibbons, Vik, & Belizán, 2008; Stothard, Tennant, Bell, & Rankin, 2009).

Both maternal obesity (BMI ≥ 30) and maternal overweight status (BMI = 25 to 29.9) have been shown to increase the risk of birth defects. One study from the Centers for Disease Control and Prevention found that babies born to mothers who were overweight at the time of conception had a higher risk for birth defects than those born to normal-weight women, including twice the risk of having babies with heart abnormalities. Mothers who were obese prior to conception were more than three times as likely to deliver babies with spina bifida or the abdominal malformation omphalocele (Watkins, Rasmussen, Honein, Botto, & Moore, 2003). A 2009 meta-analysis and systematic review found that compared to normal-weight women obese women had significantly high odds of giving birth to a baby with a neural tube defect, cardiovascular defects, hydrocephaly, and several other relatively commons birth defects (Stothard et al., 2009).

A mother who is underweight prior to becoming pregnant also puts her baby at higher risk for complications, mainly because of the association between underweight status and malnutrition (Ehrenberg, Dierker, Milluzzi, & Mercer, 2003). As discussed earlier, maternal malnutrition during pregnancy may influence fetal programming, priming the child to be more susceptible to heart disease, diabetes, and high blood pressure later in life. Malnutrition may be caused by illness, food insecurity, or other factors, and both the malnutrition and the underlying cause need to be addressed to maximize positive outcomes for both mother and baby.

Regardless of nutritional status, a BMI of less than 18.5 has been associated with a higher risk of preterm delivery (Hauger et al., 2008). Underweight women should be carefully monitored to ensure that they are meeting their nutritional needs during pregnancy, and weight gain goals should be emphasized. Supplements may need to be customized to ensure that the mother-to-be meets her nutritional requirements. Referrals to social service programs to help the mother obtain food, health insurance, and housing assistance may be necessary if malnutrition is caused by food insecurity.

In some cases, underweight status before pregnancy and/or failure to gain appropriate weight during pregnancy may be a sign of either a preexisting eating disorder or one that has developed

during pregnancy. "Pregorexia" is a recently coined term used to describe the practice of overly restricting energy intake and/or overexercising during pregnancy to minimize weight gain. The pregorexic mother disregards prenatal health guidelines in an effort to control her weight, with potentially dangerous short- and long-term effects for her baby. In addition to placing her baby at risk for poor growth and vitamin deficiencies, one study found that women with eating disorders were significantly more likely than those without to be at risk for fetal growth restriction, preterm labor, anemia, genitourinary tract infections, and labor induction (Bansil et al., 2008).

### Preexisting Conditions

In some cases, the mother may have a preexisting medical condition that may jeopardize the fetus's health long before the child is born. Mothers with multiple sclerosis, type 1 diabetes, and other serious health conditions can have healthy, successful pregnancies, but they need to be carefully monitored by specialists as well as by a healthcare team that is typically led by the obstetrician/gynecologist.

### Public Health Campaigns to Improve Preconception Nutritional Status

Ideally, most women will plan ahead and begin to adopt healthful behaviors compatible with a healthy pregnancy long before they are necessary. In reality, the majority of pregnancies are not planned, and thus, healthcare providers need to take every opportunity to encourage women to adopt healthful practices that will support a healthy pregnancy.

A few social marketing campaigns have been mounted over the years. They were designed to educate women of childbearing age on behaviors related to positive pregnancy outcomes, thus maximizing the health of their children if pregnancy occurs. One example is the March of Dimes campaign to encourage supplementation with folic acid for all women of childbearing age. The campaign had limited success, and many concluded that public health messages urging women to take folic acid supplements were ultimately far less effective in bringing about the kind of changes observed after fortification of the food supply with folic acid (Bower, Miller, Payne, & Serna, 2005). The March of Dimes discontinued this campaign shortly after and is currently focusing its education and prevention efforts more broadly on reducing behaviors in women associated with premature birth.

### General Health and Nutrition Recommendations for Women Preconception

Preconception planning for all women should include advice to begin prenatal multivitamin/mineral supplements even before conception to build stores of valuable nutrients. In addition to a standard prenatal supplement, most women could likely benefit from taking fish oil to build stores of essential fatty acids before becoming pregnant.

Lifestyle interventions should include advising women to stop smoking before they become pregnant. Alcohol and caffeine consumption should be moderated prior to conception, and alcohol should be discontinued altogether if a woman suspects that she is pregnant.

Other good practices to prepare for a healthy pregnancy include evaluating the safely of all medications the mother takes, whether prescription or over-the-counter, to determine whether they are safe to take during pregnancy. Alternate medications that are determined to be safe during pregnancy should be identified, and women should work with their doctors to switch over in the case of prescription medications.

A woman who needs to lose weight prior to conception should be very careful not to overrestrict micronutrients to a point that she becomes deficient. She should be careful not to compromise the health of the fetus when she does become pregnant by following a strict diet plan before becoming pregnant. The focus of preconception weight loss should be on cutting empty-calorie foods and increasing low-calorie, nutrient-dense foods such as fruits and vegetables, whole grains, and low-fat meats and dairy. Increasing exercise may also help the woman lose weight prior to becoming pregnant and have benefits after she becomes pregnant in helping her cope with some of the unpleasant side effects associated with pregnancy.

## Nutrient Needs During Pregnancy

The need for most nutrients is increased during pregnancy to meet the high demands of both the growing fetus and the mother who herself goes through a period of growth to carry the child and prepare for lactation. In this section, we discuss

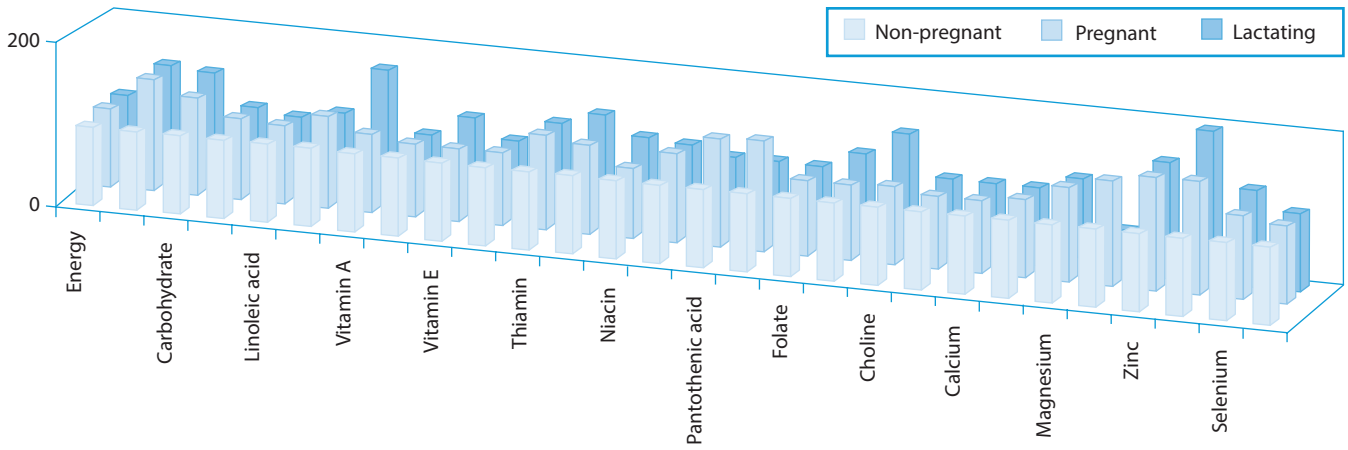



**FIGURE 1.1** Compared nutrient needs

the general need for increased macronutrients and micronutrients (see **Figure 1.1**).

### Total Energy

It often is said that a pregnant woman is "eating for two." Although this is technically correct, expectant mothers often overestimate their need for additional calories, especially early in the pregnancy. For most women, the extra energy needs are easily met by adding a small snack or two during the day. Eating smaller amounts of food more frequently throughout the day also has the benefit of helping with some of the uncomfortable side effects of pregnancy, including nausea and heartburn. The focus should be on increasing the consumption of nutrient-dense foods and minimizing empty-calorie foods that may provide the extra energy needed but do not provide micronutrients that are needed in much higher amounts compared with increased caloric needs.

Before 2002, the advice for pregnant women was to increase their energy intake by approximately 300 calories per day in the second and third trimesters (National Academy of Sciences Institute of Medicine, 1990). In 2002, the Institute of Medicine (IOM) revised the Dietary Reference Intake (DRI) for energy intake during pregnancy. The new recommendation is higher in total calories and more specific in how increased calorie needs should be distributed over the three trimesters of pregnancy. The new recommendation advises no additional calories for the first trimester, adds 340 kilocalories (kcal) for the second trimester, and 452 kcal for the third trimester (Panel on Macronutrients, Institute of Medicine, 2002).

In a comprehensive study released in 2004, Butte, Wong, Treuth, Ellis, and Smith (2004) reported that additional energy needs not only differ by trimester but also should be tailored based on the mother's preconception BMI. Butte and colleagues stated that the additional energy needs during pregnancy by trimester consisted of an additional 150 kcal/day for the underweight woman in the first trimester. In the second trimester, the underweight woman would require a 200 kcal/day increase in usual intake, whereas the normal-weight woman would require 350 kcal/day extra, and the overweight/obese would require 450 kcal/day extra energy. The third trimester would require 300 kcal/day extra for the underweight woman, 500 kcal/day for the normal-weight woman, and 350 kcal/day extra for the overweight/obese woman.

Although the Butt et al. study and the DRI recommendations provide a general guideline for caloric intake during pregnancy, the most accurate way to monitor whether the mother is consuming appropriate energy intake is to monitor her weight gain (see **Table 1.1** and **Table 1.2**). A variety of factors can alter the woman's need for additional calories, with level of physical activity being the most influential, as it is for nonpregnant women. Physical activity during pregnancy is discussed later in this chapter.

### Protein

Healthy fetal development depends on the availability of adequate protein, which provides the basic building blocks necessary for formation of enzymes, antibodies, muscle, and collagen, which are used as the framework for skin, bones, blood vessels, and

**TABLE 1.1 2009 Institute of Medicine Guidelines for Prenatal Weight Gain**

| Weight Category | Recommended Weight Gain |
|---|---|
| Underweight: BMI less than 18.5 | 28–40 pounds |
| Normal weight: BMI 18.5–24.9 | 25–35 pounds |
| Overweight: BMI 25.0–29.9 | 15–25 pounds |
| Obese: BMI greater than 30.0 | 11–20 pounds |
| Twins | 35–45 pounds |

Modified from Institute of Medicine (2009). *Weight Gain During Pregnancy: Reexamining the Guidelines*. National Academies Press; Washington, DC.

**TABLE 1.2 Average Weight Distribution of Weight Gained During Pregnancy**

| Weight | Distribution |
|---|---|
| 7.5 pounds | Average baby's weight |
| 7 pounds | Extra stored protein, fat, and other nutrients |
| 4 pounds | Extra blood |
| 4 pounds | Other additional body fluids |
| 2 pounds | Breast enlargement |
| 2 pounds | Uterine enlargement |
| 2 pounds | Amniotic fluid |
| 1.5 pounds | Placenta |

other body tissue. During pregnancy, the mother must consume adequate protein to meet the needs of her growing fetus, in addition to meeting her own increased needs as she physically grows in size to carry her baby. To accommodate the high demand, the mother's body adapts during pregnancy to conserve protein. Hormones signal the body that she is in a period of anabolism, which causes her body to retain nitrogen for protein synthesis.

The Reference Daily Intake (RDI) for protein for a nonpregnant woman is 0.8 grams per kilogram (g/kg), which comes to approximately 54 g per day for a 150-lb woman. The 2002 RDI for pregnant women recommends 1.1 g/kg of body weight, or an additional 25 g per day to meet the needs of pregnancy. According to the National Health and Nutrition Examination Survey (NHANES), the average daily protein intake for a woman aged 20 to 39 years in the United States is 74 g, and thus, protein needs should easily be met by most American women, even during times of elevated need such as pregnancy (Centers for Disease Control and Prevention [CDC], 2004).

Despite the generally high level of protein intake within the United States, several special populations should be carefully monitored for adequate protein intake and quality during pregnancy: vegetarians, vegans, low-income women experiencing food insecurity, and woman experiencing severe nausea and vomiting.

When evaluating the mother's protein status, several factors should be taken into account. The mother may be consuming an adequate number of calories but taking in insufficient protein, leading to a protein deficiency. Conversely, the mother may consume adequate protein but may still have a protein deficiency if her calorie intake is too low. To meet the increased energy needs of pregnancy, some amino acids may be used for energy, leading to a protein deficiency. Finally, the quality of the mother's protein intake should be accounted for. If the mother does not consume high-quality sources of protein, meaning sources that include all essential amino acids such as meat, eggs, poultry, fish, and dairy, she should be encouraged to consume a variety of plant-based foods to ensure that all essential amino acids are available to the fetus.

A woman who chooses a vegan diet eats no dairy products, meat, fish, or poultry, placing her at higher risk for protein deficiency both before and during pregnancy. She must consume all essential amino acids from plant sources to create the protein necessary for her fetus's growth. Although it is possible for a vegan mother to have a healthy pregnancy, careful planning and monitoring to ensure that she is meeting her increased protein needs is essential. Lacto-ovo vegetarians who do not eat red meat, poultry, or fish should also be screened for protein deficiency, although they are at a much lower level of risk because of consumption of high-quality protein in the form of milk, cheese, yogurt, and eggs.

Although vegetarianism and veganism place a woman at higher risk of protein deficiency because of limited sources of protein that may limit total protein intake, prenatal consumption of plant-based proteins including beans and nuts is associated with lower risk of gestational diabetes and lower fat mass in children into the teen years and should be encouraged in pregnant women

either in combination with animal protein intake or promoted as a good protein source for vegan women (Bao, Bowers, Tobias, Hu, & Zhang, 2013; Yin, Quinn, Dwyer, Ponsonby, & Jones, 2012).

Low-income women also are at high risk of protein deficiency during pregnancy because of potential issues with food insecurity. A woman who is food insecure may lack adequate resources to obtain protein-rich, nutrient-dense foods, which often cost more compared with less nutritious foods. It is important to help food-insecure women identify low-cost protein sources, such as canned tuna, beans, eggs, and limited amounts of meat. Woman experiencing food insecurity also should be referred to nutrition assistance programs such as WIC, the Supplemental Nutrition Assistance Program (SNAP), formerly known as food stamps, and local food pantries.

### Lipids and Fats

The mother-to-be must include enough fat in her diet to meet the needs of her growing baby. Lipids, including sterols, phospholipids, and triglycerides, which are primarily made up of fatty acids, are another basic building material of body tissue and integral to body functioning. Lipids are essential for the formation of cell membranes and hormones and are necessary for proper eye and brain development, especially during the prenatal period and into the first few years of the child's life (Innis & Friesen, 2008).

Fat is also a source of concentrated calories, which may be beneficial to woman at risk of energy malnutrition while pregnant. Women who are not at risk should avoid excess fat because it can easily lead to undesired weight gain; moderation is essential. There is no separate RDI for fat intake during pregnancy, and the recommendation remains 20% to 35% of total calories, the same as for the general population. Fat intake during pregnancy should emphasize sources that provide the essential fatty acids and choline, a component of phospholipids necessary for healthy brain function.

#### Essential Fatty Acids

The essential fatty acids linoleic acid (omega-6) and linolenic acid (omega-3) are necessary for optimal formation of the brain and eyes and also play a key role in the body as precursors of hormone-like substances called eicosanoids. Eicosanoids are used to signal a number of local reactions within the human body necessary for basic functioning. Reactions such as muscle relaxation and blood vessel constriction are signaled by eicosanoids. Immune functions such as the inflammatory response to injury and infection that signals initiation of fever, aggregation of antibodies, and pain are also controlled by eicosanoids (Connor, 2000).

Although omega-6 fatty acids are used to create proinflammatory eicosanoids, omega-3 fatty acids are used to create anti-inflammatory versions. When the body has sufficient access to both essential fatty acids, it is able to create balanced amounts of each type, allowing for optimal immune function. This balance is only possible when the two essential fatty acids are consumed through diet or supplements in adequate amounts. Although omega-6 fatty acids are plentiful in the American diet through meat and vegetables oils, omega-3 fatty acids are relatively deficient. This imbalance would theoretically lead to the ability to mount a proinflammatory response without an equal ability to slow and stop it. Omega-3 fatty acid deficiency has also been linked with lower IQ scores in infants and lower scores of visual acuity, as well as an increased risk of depression in adults; it is suspected to be one potential reason for an increased risk for chronic diseases with an inflammatory component such as cardiovascular disease (Bourre, 2007; Connor, 2000; Horrocks & Yeo, 1999).

The amount of these essential fatty acids available to the fetus is based on how much the mother eats. If a mother-to-be consumes a typical American diet, the fetal tissue will have a high concentration of omega-6. Omega-3 fatty acids are generally deficient in the standard American diet and consumed in a ratio of 1 to 10 with omega-6 fatty acids (Briefel & Johnson, 2004). This deficiency may be even more pronounced in pregnant women who avoid seafood, one of the richest sources of omega-3, because of fears of mercury contamination.

Supplementation with omega-3 fatty acids increases the availability to the fetus and should be recommended to every woman who is currently pregnant or is planning to become pregnant. The mother should be advised to continue to supplement with omega-3 fatty acids during lactation.

The revised 2002 RDI for essential fatty acids recommends an Adequate Intake (AI) of 13 g per day of omega-6 and 1.4 g per day of omega-3. It further states that the ratio of the two fatty acids should be no more than 5:1. Because of a lack of data, the AI for essential fatty acids likely does not represent ideal levels for each nutrient and

will almost certainly change as more information becomes available.

### Choline

When selecting appropriate dietary fat sources, mothers-to-be should be advised to include good sources of choline. Choline is a component of phospholipids and is needed for synthesis of lecithin, a structural component of cell membranes and an essential constituent of the human brain and nervous system. Choline is also necessary to make the neurotransmitter acetylcholine. During fetal development, choline supports the structure and function of the brain and spinal cord. In animal models, choline deficiency during pregnancy in the mother has been associated with impaired memory in her children (Zeisel, 2006).

The AI for pregnant and lactating women is 450 mg per day, increased from 425 mg per day in nonpregnant women of childbearing age. Good sources include whole, reduced-fat, and low-fat milk, liver, eggs, and peanuts. Choline intake in the United States has declined over the past few decades as Americans avoid eating egg yolk to lower cholesterol intake and reduce the amount of milk and dairy products they consume.

### Fiber

Fiber is a very important component of the prenatal diet. The development of the fetus is not dependent on an adequate supply of fiber, but a high-fiber diet significantly increases the comfort of the pregnant mother by helping to reduce constipation, a common side effect of pregnancy. Although the AI for pregnant women is 28 grams per day, according to NHANES 2007–2008, the average intake for a nonpregnant American woman of childbearing age is only approximately 14 g per day (King, Mainous, & Lambourne, 2012).

Although the American diet is generally low in fiber, leading to adverse effects on health, including constipation, hemorrhoids, and diverticulitis, it is even more important that pregnant women get enough fiber because they are at higher risk for these problems. Higher fluid needs, reduced exercise, and hormonal changes within the woman's body designed to allow the baby more room for growth may all contribute to problems with constipation and hemorrhoids during pregnancy. Pregnant women should be given the standard advice to reduce these problems, including increasing noncaffeinated fluids, moderate exercise, and high fiber intake. Fruits, vegetables, beans, whole grains, seeds, and nuts are all good dietary sources of fiber.

### Carbohydrate

Dietary carbohydrate is broken down to form glucose, also known as blood sugar, which is one of the primary energy sources for human growth and activity. The rapid growth of the fetus requires ample amounts of energy in the form of glucose be available to the fetus at all times. The Recommended Dietary Allowance (RDA) for carbohydrate during pregnancy is 175 g per day, increased from 130 g for nonpregnant women. Most Americans eat enough carbohydrate to meet normal and pregnancy requirements with a mean intake of approximately 260 g per day for women of childbearing age (CDC, 2004).

Pregnant women should be advised that a low-carbohydrate diet is dangerous during pregnancy and could place the baby at risk of poor growth. A mild restriction of dietary carbohydrate may be recommended if the mother is diabetic. If a mother enters pregnancy with preexisting diabetes or develops it while she is pregnant, she will need to work closely with her healthcare team to make sure that she provides enough, but not too much, glucose for her baby to ensure optimal growth. Gestational diabetes is discussed in more detail later in this chapter.

### Vitamin A

Vitamin A, a fat-soluble vitamin, and beta-carotene, which can be used in the body as either an antioxidant or a precursor to vitamin A, are critical during fetal development because of their involvement in growth, vision, protein synthesis, and cell differentiation. Beta-carotene is found in fruits and vegetables, and preformed vitamin A can be found in animal products, including fish, meat, and milk. Despite the important role vitamin A plays in the body, the RDA for pregnant women of 770 micrograms (mcg) per day is only slightly higher than the RDA for nonpregnant women. This is because of the high risk of birth defects associated with excessive doses of preformed vitamin A early in pregnancy (Duerbeck & Dowling, 2012).

Although it is unlikely that the mother-to-be will overconsume vitamin A from food sources alone, close attention should be paid to choosing a prenatal vitamin supplement that contains no more than the RDA for vitamin A. To minimize

risks associated with excess intake, a significant percentage of the vitamin A content in the supplement should come from beta-carotene instead of providing it all as preformed vitamin A (Strobel, Tinz, & Biesalski, 2007). Beta-carotene is not converted to vitamin A unless the body determines the need, and thus, it is a safer form to consume.

### Vitamin D

Vitamin D may be obtained through diet and supplements or can also be made by the body when skin is exposed to ultraviolet rays. Vitamin D is necessary to help build and maintain strong bones and teeth and is very important during fetal development for this reason. Recent research shows that babies born during the late summer and early fall are taller and have wider bones (Sayers & Tobias, 2008). There is also mounting evidence that vitamin D plays a key role in preventing common cancers, autoimmune diseases, type 1 diabetes, heart disease, and osteoporosis. Furthermore, studies have shown that vitamin D deficiency is common in the United States, suggesting that most Americans would benefit from supplements (Holick, 2008). One systematic review and meta-analysis found clear associations between vitamin D insufficiency during pregnancy and increased risk of development of gestational diabetes, preeclampsia, and premature birth (Aghajafari et al., 2013). It is unclear whether supplementation prior to or during pregnancy would help prevent these conditions, and research is ongoing.

Fatty fish (e.g., mackerel, sardines, salmon), liver, egg yolks, and fortified milk are all good dietary sources of vitamin D. Vegans, women with lactose intolerance or milk allergies, women who dislike dairy products, and women who avoid the sun may be at particular risk of vitamin D deficiency. The RDA for pregnant and nursing women is currently 200 IU (5 mcg), although this is considered much too low by experts in the field. Michael Holick, MD, PhD, one of the leading researchers on vitamin D, recommends 1,000 IU daily for everyone older than the age of 1 year. Alternatively, if the latitude provides adequate year-round ultraviolet light, vitamin D needs can be met by exposure of the arms and legs to sunlight for 5 to 10 minutes three times a week. Women in the United States living north of Atlanta, Georgia, women with darker skin tones, and women who use a lot of sunblock or who limit their sun exposure should be strongly advised to supplement with additional vitamin D beyond that provided by the prenatal supplement.

### Calcium

Although calcium is also necessary for proper bone formation in conjunction with vitamin D, the RDI for pregnant women is the same as it is for nonpregnant women, 1,000 mg per day for women older than 18 years. Dietary sources include milk, yogurt, and cheese, as well as sardines with bones and some leafy green vegetables. Vegan women and women who consume fewer than three servings of dairy per day should be advised to take a separate calcium supplement during pregnancy to make sure they are getting enough. Alternatives to cow's milk such as soy, rice, and almond milk should be checked to make sure that they are fortified with calcium. These products must be shaken well because the calcium has a tendency to settle on the bottom of the containers. Consuming adequate calcium while pregnant and breastfeeding may not affect the baby's bone density, but it does protect the mother's bones from losing too much of the calcium that she will give to her baby if dietary calcium is not enough.

### B Vitamins

The RDI for most B vitamins is higher for pregnant women compared with nonpregnant women in the same age category. B vitamins are primarily used as cofactors in energy metabolism, and the need for these vitamins is increased proportionately to the increase in energy needs during pregnancy. Deficiency of most B vitamins is rare because of their availability in a wide variety of food sources and their presence in prenatal supplements. Two B vitamins, folate and $B_{12}$, should be given special attention, however, because of their unique roles during fetal development and potential for deficiency in a pregnant woman.

#### Vitamin $B_{12}$

Vitamin $B_{12}$ is essential for the production of red blood cells, the manufacturing of genetic material, and healthy functioning of the nervous system. The RDA is 2.4 mcg in nonpregnant women compared with 2.6 mcg in pregnant women. Deficiencies in pregnant and breastfeeding women may cause neurologic damage in their children. Deficiency of $B_{12}$ at the start of pregnancy may increase risk of birth defects such as neural tube defects and may contribute to preterm delivery (Molloy, Kirke, Brody, Scott, & Mills, 2008). The only natural dietary sources are animal products, including meats, dairy products, eggs, and fish (clams and oily fish are

very high in $B_{12}$), but like other B vitamins, $B_{12}$ is added to commercial dried cereals and included in adequate amounts in prenatal supplements.

### Folate

Folate, and its synthetic form folic acid, is a B vitamin that is used in the manufacturing of neurotransmitters and is particularly important during early pregnancy because of its essential role in synthesizing DNA in the cells. The RDA for folic acid in nonpregnant women of childbearing age is 400 mcg per day and increases to 600 mcg during pregnancy. Good food sources include avocado, bananas, orange juice, dry cereal, asparagus, fruits, green leafy vegetables, and dried beans and peas.

In preconception and early pregnancy, 400 mcg of folic acid in supplement form appears to be adequate for the prevention of neural tube defects, the most common of which are spina bifida and anencephaly. Spina bifida occurs when there is an incomplete closure of the spinal cord and spinal column, and anencephaly is severe underdevelopment of the brain. Neural tube defects occur during the first 28 days of pregnancy, usually before a woman even knows that she is pregnant, making the timing of folate supplementation of particular importance. Additional folate introduced after this critical period will not reverse prior damage done by a lack of the nutrient (Molloy et al., 2008).

As with other nutrients, the fetus has priority for folate over the mother's needs, potentially leaving the mother deficient after delivery unless her intake is sufficient to meet both of their needs. The extra folate in most prenatal vitamins is to prevent the mother from becoming deficient as the pregnancy progresses, both for her own health and to prevent birth defects if a pregnancy occurs within a few months of delivery.

In 1992, the U.S. Public Health Service recommended that all women of childbearing age consume 400 mcg of folic acid daily (CDC, 1992); however, surveys suggested that the overwhelming majority of U.S. women were not doing so. To deal with the public health crisis of neural tube defects, the Food and Drug Administration mandated folic acid supplementation in enriched grain products. Supplementation, which began in 1998, was estimated to increase the daily folate intake of the average American by approximately 100 mcg (Honein, Paulozzi, Mathews, Erickson, & Wong, 2001).

Monitoring has shown that fortification has been effective in reducing the incidence of neural tube defects, which decreased by 70% in the United States since the program began. Although prevention of neural tube defects was the primary justification, folic acid fortification has been effective in reducing cardiovascular events as well. During the same period, stroke and stroke deaths declined by 15% (Yang et al., 2002). Despite concern about potential adverse effects, such as a masking of $B_{12}$ deficiency, no problems have been identified as a result of the fortification, and controversy remains over whether the level should be increased in the United States, similar to the model used in Canada and Europe.

### Iron

Iron is a trace mineral that is vital for fetal growth and development because it plays a key role as a cofactor for enzymes involved in oxidation-reduction reactions, which occur in all cells during metabolism. Iron is also necessary as the component of hemoglobin that allows red blood cells to carry oxygen throughout the body. Perhaps most important, iron is essential for normal neurodevelopment during fetal and early childhood development. Iron deficiency in infancy and early childhood is associated with impaired memory function and changes to temperament that may mimic attention deficient disorder. Memory deficits persist even after normal iron levels are achieved (Georgieff, 2008). Worldwide, inadequate dietary iron intake is the most common nutrient deficiency, and women are at particularly high risk because of a regular loss of iron during monthly menses.

Pregnancy places an even higher demand for iron on the woman's body, as both her own blood volume expands to carry the pregnancy and as her child demands iron for normal development. The RDI for iron during pregnancy is 27 mg per day, which is particularly vital during the second and third trimesters. During the last 3 months, the baby is accumulating iron for use during early life. If adequate iron is not available during the prenatal period and first 6 months of life, studies show that there can be lifelong neurologic effects that cannot be reversed even if iron is supplemented at adequate levels in early childhood (Beard, 2008).

Fetal iron stores are meant to last the child until approximately 6 months of life, when the American Academy of Pediatrics recommends a good dietary source of iron be introduced into the infant's diet to help support the accelerated growth of the child during the first year of life. The best

method for combating iron deficiency in pregnancy is to promote high intakes before pregnancy so there will be at least 300 mg of iron stored as the woman enters pregnancy.

Mothers should be encouraged to take a prenatal vitamin with adequate iron to meet needs, although high amounts of supplemented iron are associated with constipation and upset stomach for many pregnant women. If the woman enters pregnancy iron replete, prenatal supplements do not have to contain as high a level of iron as for a woman who enters pregnancy iron deficient. Lower levels of iron in the multivitamin/mineral will minimize unpleasant side effects. For women who require high amounts of iron during pregnancy, several measures can be suggested to decrease associated discomfort.

It is important to help women find strategies to improve their tolerance of the high iron levels in prenatal supplements because discomfort is a primary reason that mothers stop taking their vitamins. Many women find taking their prenatal vitamin just before bed and/or with food helps them better tolerate the high level of iron. Taking the iron with a source of vitamin C will improve absorption and may help the mother to reduce the amount of extra iron she needs to take. Finally, eating sources of iron that are highly bioavailable such as red meat or snacking on iron-fortified cereal will also help to increase iron intake. Women who snack on dry cereal should be advised to have with it juice or another vitamin C source, instead of dairy products, which inhibit the absorption of iron.

### Magnesium

Magnesium is a cofactor in more than 300 enzymes in the body. Dietary magnesium inadequacy has been demonstrated to be common among American women and is associated with an increased risk of miscarriage, fetal growth retardation, maternal hospitalizations, and preterm delivery (Durlach, 2004). Inadequacy of magnesium has also been identified as a risk factor for the development of both gestational diabetes and type 2 diabetes (Barbagallo, Dominguez, & Resnick, 2007). After birth, it has been associated with an increased risk of sudden infant death syndrome (SIDS) and increased referrals to the neonatal intensive care unit (NICU). Most prenatal vitamins contain only 10% to 25% of the RDA for magnesium, and thus, emphasis should be put on consuming good dietary sources. The best dietary sources include peanuts, bran, wheat germ, nuts, and legumes.

Acute therapeutic intravenous magnesium is sometimes used to treat preeclampsia and premature labor (Thapa & Jha, 2008). Other studies have shown a potential benefit of supplementing magnesium at the RDA level in the reduction of leg cramps in pregnancy, although the association is not conclusive (Young & Jewell, 2002).

### Zinc

The RDA for zinc during pregnancy is 11 mg per day, increased from 8 mg for nonpregnant women. Adequate zinc is extremely important during the first trimester when organs are formed and may play a role in assisting in immune system development (Shah & Sachdev, 2006). Zinc deficiency is common worldwide, and supplementation with the RDA is advised during pregnancy. Because red meat is the best source of zinc, it may be more difficult for vegans and vegetarians to get enough. Furthermore, phytates found in a plant-based diet can interfere with zinc absorption. For vegetarian and vegan women, a supplement that includes 15 mg of zinc is advised throughout the pregnancy.

### Iodine

Although overt deficiency of iodine is uncommon in the United States because of fortification of table salt, iodine deficiency affects more than 2 billion people worldwide and is the leading cause of mental retardation. Women in the United States are at risk, however, of consuming inadequate amounts of iodine as a result of eating variable levels in common foods. Given the nutrient's important role in fetal development, supplementation is recommended for pregnant and lactating women (Pearce, 2007).

The RDI for pregnant women is 220 mcg of iodine per day. Mothers who do not get enough can put their baby at greater risk of mental retardation as well as growth, hearing, and speech problems. Women consuming a low-salt diet during pregnancy, especially those trying to manage edema and/or pregnancy-induced hypertension, are at particular risk of iodine insufficiency.

In a recent study done by scientists at the Boston University Iodine Research Laboratory, researchers determined that a large percentage of prenatal vitamins do not include any iodine (Leung, Pearce, & Braverman, 2009). They found that of 223 prenatal multivitamins, both prescription and those sold

over the counter, in the United States only half of them contained iodine. Of the vitamins that listed iodine, many fell short of the stated amount on the label. Prenatal multivitamins that contain potassium iodide instead of kelp were more likely to contain adequate amounts. Pregnant women are advised to take a prenatal supplement containing iodine, preferably one that uses potassium iodide as the source.

## Fetal Development

A healthy pregnancy lasts an average of 40 weeks, with a normal range of 38 to 42 weeks. There are three distinct stages of fetal development during which the baby must complete specific developmental tasks. The zygote period is the first stage beginning at conception and lasting approximately 2 weeks. The embryonic period goes from 2 to 8 weeks, and the fetal period lasts from 8 weeks until birth.

Weeks of pregnancy are counted from the first day of the woman's last period, and thus, a woman is considered pregnant approximately 2 weeks before she actually conceives the baby. After conception, the fertilized egg, also known as the zygote, makes its way to the uterus, where it is implanted and begins its rapid growth.

### The Embryonic Phase

The embryonic stage begins shortly after implantation of the zygote in the wall of the uterus and lasts until 8 weeks. This period often begins before a woman even knows she is pregnant, but it is a critical stage of fetal development. During this time, the embryo's cells are dividing rapidly to form the basic body systems. By 8 weeks after conception, the embryo is only approximately 1.25 inches long but already has a complete central nervous system, a beating heart, a digestive system, fingers, toes, and the beginning of facial features.

### The Fetal Phase

Fetal development begins after the eighth week. Early in this period the fetus continues to develop all of the necessary body systems and begins to take on a more human appearance. Distinguishable genitals, hair, nails, and vocal chords form. The kidneys begin to process bodily fluids, and the liver begins to function. Bones also begin to harden early in the fetal period.

Although far from ready for birth, by the 28th week of gestation, most babies born can survive outside the womb with intervention to aid breathing until the lungs are more mature.

During the final 12 weeks of gestation, brain and eye development proceed at a fast rate. The fetus also builds bone mass and gains fat stores to help him or her survive outside of the protective environment of the womb. The immune system begins to mature during this time, and lungs develop to a point where the baby can breathe without external intervention after approximately 37 weeks of gestation. Finally, the fetus spends the last several weeks of gestation laying stores of nutrients to help support functioning during early life until he or she can transition to a more varied diet at 6 months of age.

### Critical Periods of Nutrient Intake During Embryonic and Fetal Development

Given the fast rate of growth and the enormous complexity of the task, it is easy to see how deficiency of any nutrient needed during a specific phase of development could affect this delicate process. Although deficiency of almost any micronutrient or macronutrient will certainly affect the course of development, several in particular have been identified with specific birth defects if the nutrient is not present during a very narrow window of time. Several others are suspected to cause less overt problems if the nutrient is deficient during a critical period.

Folic acid deficiency during the first 4 weeks of development when the central nervous system is forming is perhaps the most devastating nutrient deficiency because it may result in neural tube defects. Deficiency of iodine during brain development can also have dramatic and obvious permanent effects, resulting in mental retardation. The effects of other nutrient deficiencies may be less dramatic but may nonetheless have a significant impact on the child's health over the course of the lifetime. For example, a deficiency of vitamin D during the fetal period can result in underminerialized bones, and a deficiency of iron during brain development may lead to permanent alteration of memory function (see the section on each specific nutrient for more information).

Even when there are adequate nutrients available for the child's physical development, during the last several weeks in the womb, the fetus builds stores of nutrients, including fat, vitamin D,

vitamin $B_{12}$, and iron to help the baby through its early development outside of its mother. If nutrient supplies are not adequate in the mother during pregnancy, the infant will need to consume these nutrients in ample amounts after birth, which may tax the immature digestive system.

## Common Problems Associated with Pregnancy

How a woman experiences pregnancy can be very different from woman to woman. Even for the same woman, the experience may be very different from pregnancy to pregnancy. A number of factors contribute to the mother's experience with pregnancy, including how the mother feels about being pregnant; her physical reaction to pregnancy; any health conditions that she has or develops; her physical environment, including availability of food, housing, and health care; and finally other demands that cause stress for the mother (e.g., her job, other kids). This section discusses some of the common physical side effects of pregnancy as well as some of the more serious health problems that may develop as a result of the pregnancy.

### Nausea and Vomiting of Pregnancy and Food Aversions and Cravings

During pregnancy, the mother's body goes through many changes to accommodate the new life growing inside her. Many women experience side effects of these changes that influence their ability to eat as healthfully as they might like. Hormonal shifts caused by the pregnancy are thought to be responsible for the majority of these side effects, but controversy remains regarding whether some common conditions related to pregnancy are physiologic or psychological phenomena (Markl, Strunz-Lehner, Egen-Lappe, Lack, & Hasford, 2008). Regardless of their origin, it is important to help pregnant women struggling with problems such as nausea, vomiting, food aversions, and cravings to minimize the impact of these factors and maximize nutrition for the health of both mother and baby (Keller, Frederking, & Layer, 2008).

#### Nausea and Vomiting of Pregnancy

One of the most common side effects of pregnancy is known as nausea and vomiting of pregnancy (NVP), or more commonly "morning sickness." Ironically, morning sickness is usually not limited to the morning, although that may be when a woman experiences it the most acutely. Up to 80% of pregnant women experience NVP at some point, usually beginning between the fourth and seventh week after their last period and ending for most women by the twentieth week. For 10% of those who have it, however, it remains an ongoing battle throughout the pregnancy (Quinla & Hill, 2003).

The underlying cause of nausea and vomiting of pregnancy remains unclear, but hormonal shifts are hypothesized to be one potential cause. Estrogen, progesterone, and human chorionic gonadotropin (hCG) have all been investigated for possible mechanisms of action (Davis, 2004). Progesterone, for example, which is released during pregnancy, has been suggested to cause delayed gastric motility, causing nausea. One study found that when progesterone was given to nonpregnant women they developed nausea and vomiting similar to NVP (Walsh, Hasler, Nugent, & Owyang, 1996).

The most severe form of NVP is known as hyperemesis gravidarum, which is estimated to affect approximately 1 in every 200 pregnant women. Although there is no standard definition, the condition is characterized by persistent vomiting, dehydration, ketosis, electrolyte disturbances, and weight loss of more than 5% of body weight (Ismail & Kenny, 2007).

Although normal NVP is actually associated with positive outcomes for the baby, hyperemesis can be dangerous to both the mother and the child. NVP has been associated in several studies with improved pregnancy outcomes, including fewer miscarriages, preterm deliveries, and stillbirths, as well as fewer instances of fetal low birth weight, growth retardation, and mortality. One potential explanation for this is that NVP is a mechanism designed to increase the mother's intake of simple carbohydrates, which alleviates her symptoms and supplies increased amounts of glucose to the growing fetus (Furneaux, Langley-Evans, & Langley-Evans, 2001). In contrast, hyperemesis gravidarum has been associated with increased adverse effects for both the mother and fetus. The mother is more likely to experience splenic avulsion, esophageal rupture, Mallory-Weiss tears, pneumothorax, peripheral neuropathy, and preeclampsia. The fetus is at increased risk of fetal growth restriction and mortality (Ismail & Kenny, 2007).

Dietary modifications are the first line of treatment for women with mild nausea and vomiting. Several measures are often used in combination

to help manage symptoms until they pass on their own. Most women find that it helps to eat small, frequent meals. Avoidance of smells and food textures that cause nausea also helps, keeping in mind that what triggers the nausea may be different for each woman. Usually bland-tasting, cold foods that are high in carbohydrates and low in fat are best tolerated. Eating high-carbohydrate foods first thing in the morning often helps and may be most useful even before the woman gets out of bed. Sour and tart liquids such as lemonade often are tolerated better than water. It also may help if the woman avoids the smell of food until mealtime because smells often trigger nausea.

Other treatments include acupressure bands, ginger, and special supplements made specifically for NVP, such as "Pregipops" and "Pregidrops." Supplementation with vitamin $B_6$ has also been associated with relief of symptoms, and several trials have tested both the effectiveness and safety of this treatment (Dror & Allen, 2012; Tiran, 2002).

#### Cravings and Aversions

Although food cravings and their flip side, food aversions, are thought of as common conditions of pregnancy, not all women experience these sensations. One study found that food cravings only occurred in 61% of women, whereas aversions occurred even less frequently, appearing in 54% of pregnant women (Bayley, Dye, Jones, DeBono, & Hill, 2002). There is little understanding of what causes cravings and aversions, and not all experts agree that they are a real phenomenon and not simply a psychological manifestation.

One plausible explanation for why cravings and aversions are experienced by women during pregnancy is as a result of changes in smell and taste. One study found that abnormal smell and/or taste perception was reported by 76% of pregnant women surveyed. Increased smell sensitivity was found to be very common in early pregnancy, occurring in 67% of respondents. Abnormal taste sensitivity was reported by 26% of women in the study, often described as increased bitter sensitivity and decreased salt sensitivity. The results of this study suggest that abnormal smell and/or taste perception is experienced by a large majority of pregnant women and may underlie food aversions and cravings during pregnancy (Nordin, Broman, Olofsson, & Wulff, 2004).

Another theory behind the development of cravings is that some women who deprive themselves when they are not pregnant think of pregnancy as a time to treat themselves to foods they typically avoid and indulge themselves—because after all, they are "eating for two."

There are also several less plausible theories behind the prevalence of food cravings and aversions during pregnancy. One states that food cravings are nature's way of helping the mother meet her nutritional needs. For example, the woman may need sodium to help manage her increased fluid volume during pregnancy, and thus, she may crave salty foods. Although this is an interesting idea, there is no evidence that potential nutrient shortfalls are translated into food cravings in the body.

A similar theory regarding food aversions hypothesizes that they are a protective mechanism during pregnancy designed to steer the mother away from dangerous foods. For example, a woman may have an aversion to drinking coffee when pregnant, which is beneficial because limiting caffeine intake during pregnancy may aid in fetal mental and physical development and decreases risk of infant death. Many women also develop aversions to the taste and smell of cigarettes while pregnant, which increases compliance with smoking cessation during the prenatal period (Pletsch & Kratz, 2004). Although there is no evidence that aversions are developed as a protective mechanism, this remains a persistent myth.

Pregnant women can indulge their cravings so long as they are meeting their needs nutritionally. The craved-for food also should be screened to ensure that it is not contraindicated by special dietary restrictions such as a high-carbohydrate food for a woman with gestational diabetes. Finally, the concept of moderation must be emphasized to keep the mother's weight gain within the IOM guidelines based on her prepregnancy BMI. Mothers-to-be should be advised to keep portions small and to try to eat only healthier versions. For example, a woman who craves ice cream may want to indulge in a reasonable half cup of lite ice cream or frozen yogurt instead of a pint of premium high-calorie ice cream.

#### Reflux

Reflux, also known as heartburn, occurs when acid from the stomach makes its way through the lower esophageal sphincter, causing a burning sensation in the lower esophagus. It is a very common problem during pregnancy and is often one of the first symptoms of pregnancy, appearing within the

first month after conception for many women. Occurring early in pregnancy, reflux is likely caused by hormonal changes because it occurs even before most women have gained any pregnancy weight. Later in pregnancy, the pressure of the baby pushing upward against the mother's stomach may exacerbate the problem (Ali & Egan, 2007).

Most health authorities agree that calcium carbonate supplements such as Tums are safe to use during pregnancy and helpful for alleviating symptoms (Richter, 2005). Other measures encompass standard advice for anyone with reflux, including keeping upright for 1 to 2 hours after eating, eating smaller meals, drinking between meals instead of with the meal to avoid overfilling the stomach, elevating the head of the bed a few inches, and avoiding trigger foods. Although trigger foods differ from person to person, the most common are coffee, acidic foods such as tomato sauce, minty foods, and chocolate. Other novel approaches to managing reflux include drinking water to wash down the acid and chewing sugarless (non-mint-flavored) gum to generate saliva to help wash down the acid.

Reflux is not serious for most women and will go away within a few months post pregnancy. In very rare cases, mothers may need to be prescribed a stronger medication to manage the reflux. This decision should be made based on the mother's level of discomfort.

### Pica

Occasionally a pregnant woman may crave nonfood items such as ice, dirt, clay, paper, and even paint chips, a condition known as pica. It has been theorized, although never proven, that pica may signal a micronutrient deficiency, specifically a zinc, calcium, or iron deficiency (Young, 2010). Expectant mothers may also get the urge to eat flour or cornstarch, which despite being food items are a problem in large amounts. Too much can lead to blocked bowels and crowd out the nutrients the baby needs by causing the mother to feel full. A woman with pica or an unhealthy craving such as cornstarch should resist eating these items and should speak to her doctor right away.

### Preeclampsia

Preeclampsia is a syndrome that is characterized by dangerously high blood pressure and protein in the urine. It is often accompanied by edema, sudden weight gain, headaches, and changes in vision. It occurs only during pregnancy and up to 6 weeks postpartum, usually appearing in the late second or the third trimester. According to the National Heart, Lung and Blood Institute, the condition appears in 5% to 8% of pregnancies in the United States.

Preeclampsia, pregnancy-induced hypertension, and eclampsia (formerly known as toxemia) are closely related conditions. Pregnancy-induced hypertension is generally considered the mildest manifestation, whereas eclampsia is one of the most serious complications of severe preeclampsia. Left untreated, eclamptic seizures can result in coma, brain damage, and possibly maternal or infant death. HELLP syndrome, which stands for hemolysis, elevated liver enzymes, and lowered platelets, is another of the most severe forms of preeclampsia, usually affecting the liver and causing stomach and right shoulder pain. It occurs in 4% to 12% of the women who have preeclampsia. HELLP syndrome is particularly dangerous because it may occur even before the mother develops the classic symptoms of preeclampsia. It often is mistaken for the flu or gallbladder problems (Haram, Svendsen, & Abildgaard, 2009).

Complications of preeclampsia include prematurity, fetal growth restriction, and death. Fetal growth restriction results from reduced blood flow to the placenta, which compromises the supply of nutrients to the baby and can result in a shortage of food and subsequent starvation. As a result, the baby may be small for gestational age. This shortage of nutrients available through the placenta places the baby at serious risk, and premature birth may result naturally or be induced for the protection of both mother and child (Sibai, Dekker, & Kupferminc, 2005). According to the March of Dimes, preeclampsia is a leading cause of premature births in the United States. Worldwide, preeclampsia and other hypertensive disorders of pregnancy are a leading cause of maternal and infant death.

Bed rest is usually recommended after a woman is diagnosed with preeclampsia, and the mother is closely monitored to make sure that her condition does not progress to a more serious stage. Magnesium sulfate is recommended for all women with preeclampsia to reduce risk of progression to eclampsia (Duley, 2003).

There are several known risk factors for preeclampsia, two of which should be of particular concern given changing trends in the profile of pregnant women in the United States over the past few decades: a BMI of 30 or higher and a

maternal age of 40 years and older or 18 years and younger. Other risk factors include a previous history of preeclampsia, a history of chronic high blood pressure, diabetes or kidney disorder, a family history of the disorder, women carrying multiple fetuses, women diagnosed with polycystic ovarian syndrome, and women with lupus or other autoimmune disorders such as rheumatoid arthritis, sarcoidosis, or multiple sclerosis.

Several nutritional interventions have been associated with reduced risk of developing preeclampsia. Increased intake of fiber and calcium has been positively associated with reduced risk in several studies (Hofmeyr, Duley, & Atallah, 2007; Wallis & Saftlas, 2008). Women who are at risk should be encouraged to consume adequate dairy products and fruits and vegetables before and during pregnancy.

The role of nutrient deficiencies has also been examined. One study showed that women with low levels of the mineral selenium were four times more likely to have preeclampsia than women with higher levels (Rayman, Bode, & Redman, 2003). The researchers also found that low selenium was associated with an increased risk of premature delivery. Selenium is found in whole grains, fortified cereal, and Brazil nuts.

### Gestational Diabetes

*Gestational diabetes* is the term used for diabetes diagnosed during pregnancy whether or not it is thought to be directly caused by the pregnancy. It is present in about 5% of all pregnancies and is usually detected between 24 and 28 weeks by an oral glucose tolerance test (Lucas, 2001). In some cases, type 2 diabetes is originally diagnosed during pregnancy. In this case, the diabetes is referred to as gestational diabetes until the mother is no longer pregnant. If the blood sugar fails to return to normal levels after the baby is delivered, the mother will then be diagnosed with type 2 diabetes.

Women of certain ethnic backgrounds appear to have a higher genetic predisposition, increasing rates to as high as 7% to 9% within certain populations. Women who identify themselves as Hispanic, African American, Native American, Asian, Pacific Islander, and Indigenous Australian are at an elevated risk. Women who were obese prior to pregnancy, who have been diagnosed with polycystic ovarian syndrome, and those with thyroid disorders are also at higher risk (Cheng & Caughey, 2008). One analysis suggested that approximately half of the gestational diabetes cases in overweight and obese women in the United States could have been prevented if those women had attained a normal weight prior to pregnancy (Kim et al., 2010).

Under normal conditions, the mother's body adapts during pregnancy to ensure that a constant supply of glucose is available to her baby by becoming slightly insulin resistant. If the mother's body is not able to control this natural phenomenon adequately, she may develop gestational diabetes, creating an overabundance of glucose in her bloodstream. To compensate for the extra supply of glucose, the fetus begins to overproduce insulin to manage his or her own blood sugar level. Insulin is a growth hormone, and a high level of fetal production leads to excessive infant growth and high birth weights.

Untreated gestational diabetes is associated with a higher risk of stillbirth and macrosomia, usually defined as a birth weight of more than 10 lbs. When the infant is abnormally large, the mother is at a high risk for a cesarean section, which carries with it all of the complications of any major surgery (Schmidt et al., 2001). In addition, children born via cesarean section are more likely to develop asthma and allergies, potentially because of a lack of exposure to the normal vaginal flora that helps immunize them (Bager, Wohlfahrt, & Westergaard, 2008).

A macrosomic (large) baby is also at high risk for birth complications such as shoulder dystocia, which occurs when the baby's shoulders are too large to move through the birth canal. Shoulder dystocia can lead to permanent nerve damage for the infant and may be a potentially life-threatening situation. In addition to the physiologic complications associated with delivery of a large infant, there are other potential adverse outcomes. If the baby is overproducing insulin at the time of delivery, there is a serious risk of subsequent low blood sugar for the infant after the umbilical cord is cut and the constant supply of glucose is no longer present. There is a significantly higher rate of neonatal intensive care unit admissions for infants of diabetic mothers who have poor blood sugar control. Prevention is effective in reducing most risks associated with gestational diabetes.

Women who are diagnosed with gestation diabetes can usually manage to keep their blood sugar within a reasonable range with a mild restriction

of carbohydrate in their diet. In cases in which the mother has tried dietary modifications but blood sugars remain above optimal levels, medication may be indicated. Insulin therapy is the accepted treatment for gestational diabetes if a mother fails diet and lifestyle therapy.

Currently, no oral medications are approved for use in pregnant women, although several studies have shown glyburide to be safe and effective for pregnant women and some obstetricians are choosing to prescribe it to pregnant women. Glyburide, a second-generation sulfonylurea, has been compared with insulin treatment in several randomized controlled trials, and glucose control was found to be similar to treatment with injections of insulin. Women taking glyburide have been found to have similar pregnancy outcomes, including a reduced incidence of cesarean delivery rates, preeclampsia, macrosomia, and neonatal low blood sugars (Nicholson et al., 2009). The use of glyburide in pregnant women is not currently endorsed by any major medical authority and is still considered by some to be a potentially unsafe practice. It is gaining popularity in healthcare settings, however, because of mounting evidence of its safety and a high rate of compliance with the treatment by mothers.

Even if the mother's blood sugar returns to normal after the birth, it is important to continue to follow up. These women are at high risk for developing gestational diabetes during subsequent pregnancies. Furthermore, studies show that as many as 70% of all women with gestational diabetes will eventually be diagnosed with type 2 diabetes (Kim, Newton, & Knopp, 2002). Prevention efforts after pregnancy to emphasize after the birth include stressing the importance of maintaining or achieving a healthy weight, regular exercise, and a diet that emphasizes complex carbohydrate.

## The Interaction of Lifestyle and Pregnancy

Lifestyle behaviors such as physical activity, smoking, and alcohol consumption have a strong impact on pregnancy. When a pregnancy is planned, it is easier to modify harmful behaviors ahead of time, but this is often not the case and women are often unexpectedly pregnant. Furthermore, many lifestyle choices are coping mechanisms used to handle stress. Pregnancy may place extra stress on the mother, making it even more difficult to give up harmful practices. It is important to help the mother develop a strong support system to help her cope with the stress of pregnancy as well as stress she may face after delivery to help her give up harmful behaviors and adopt positive ones that will benefit both her child's health and her own. This section discusses some of the common lifestyle choices that may affect pregnancy.

### Physical Activity, Safety, and Energy Needs

There are many good reasons for women to exercise during pregnancy. Exercise during pregnancy helps to reduce some of the common complaints of pregnancy, including backaches, constipation, and bloating and swelling. It improves mood and may help mothers sleep better. It also promotes the strength, muscle tone, and endurance that may be helpful during labor and recovery. The American College of Obstetricians and Gynecologists (ACOG) recommends that pregnant women without medical or obstetric complications engage in 30 minutes or more of moderate exercise a day on most, if not all, days of the week (American College of Obstetricians and Gynecologists [ACOG], 2002).

Expert organizations including the ACOG and the National Academy of Sports Medicine have agreed that most women can safely engage in a level of physical activity similar to their activity level prior to pregnancy, as long as weight gain and infant development are not compromised (ACOG, 2002). Women who maintain a moderate to high level of physical activity during pregnancy will have relative caloric needs higher than less active women who are in the same stage of pregnancy. Moderate to high levels of physical activity have been associated with lower infant birth weights compared with infants of more sedentary women, but this is likely because the higher caloric needs of the active women were not met during pregnancy (Penney, 2008). If the mother is gaining appropriate weight, indicating that she is meeting her caloric needs, the birth weight of the baby should not be affected, regardless of the amount of physical activity.

Some caution should be observed when helping mothers decide on an exercise program during pregnancy. Women with medical or obstetric complications should be encouraged to avoid vigorous physical activity. Contraindications to exercise during pregnancy put forth by ACOG include pregnancy-induced hypertension, preterm rupture of membranes, preterm labor during the prior or

current pregnancy, incompetent cervix or cerclage placement, persistent second- or third-trimester bleeding, placenta previa, and intrauterine growth retardation. Relative contraindications include chronic hypertension, thyroid function abnormality, cardiac disease, vascular disease, and pulmonary disease.

Pregnant women should be advised to avoid contact sports such as soccer and high-impact sports and those that require advanced balance, which may be affected later in pregnancy. Examples include horseback riding, skiing, and gymnastics.

Pregnant women also should be warned to be careful when engaging in flexibility exercises. During pregnancy, a hormone called relaxin is produced that is designed to help loosen joints so the baby has room to grow and to make labor easier. The relaxing of ligaments caused by the release of relaxin increases risk of injury, especially during flexibility exercises, and thus, women should be advised not to overdo stretching.

### Food Safety

Food safety should be a concern for every American at all times, but pregnant women, young children, individuals with compromised immune systems, and older individuals need to take extra precautions to avoid exposure to foodborne pathogens.

Infection with a foodborne illness may cause harm to both the mother and her fetus and should be avoided if possible. Women become more susceptible to infection with the hepatitis E virus, *Coxiella burnetii*, *Listeria monocytogenes*, and *Toxoplasma gondii*, which are intracellular pathogens that take advantage of hormonal shifts during pregnancy that downregulate cell-mediated immune functions (Smith, 1999).

All pregnant women in the United States are currently advised to avoid foods that are considered health risks for pregnant women and developing babies. These include any meat, seafood, or egg item that is raw or undercooked and any unpasteurized food, including milk, cheeses made from unpasteurized milk such as brie and feta, and unpasteurized juice. Women are advised to wash all fruits and vegetables thoroughly before eating them and to avoid any that cannot be properly washed or are known to carry foodborne illness, such as alfalfa sprouts.

Special precautions have been set forth by the U.S. Food and Drug Administration's Center for Food Safety and Applied Nutrition (CFSAN) to reduce risk of contamination with *L. monocytogenes* (U.S. Department of Agriculture, 2003). *L. monocytogenes* is of particular concern because it can grow at refrigerator temperatures, whereas most other foodborne bacteria do not. Measures to reduce exposure include avoiding unpasteurized foods and heating ready-to-eat foods until they are steaming to kill the bacteria. For example, it is recommended that hot dogs and luncheon meats should be eaten only if they are reheated until steaming hot.

CFSAN recommends that pregnant women avoid the following foods:

- *Sushi or sashimi*: Raw fish (including oysters, clams, mussels, and scallops) or foods made with raw fish are more likely to contain parasites or bacteria than are foods made from cooked fish.
- *Swordfish, tilefish, king mackerel, and shark:* These fish can contain high levels of methylmercury, a metal that can be harmful to the unborn baby. Other cooked fish/seafood is considered safe, but women should try to vary their seafood. Up to 12 ounces (2 average meals) a week of a variety of fish and shellfish that are lower in mercury appear to be safe during pregnancy. Five of the most commonly eaten fish that are low in mercury are shrimp, canned light tuna, salmon, pollock, and catfish. Albacore ("white") tuna has more mercury than canned light tuna.
- *Raw sprouts (including alfalfa, clover, radish, and mung bean):* Bacteria can get into sprout seeds through cracks in the shell before the sprouts are grown. After this occurs, these bacteria are nearly impossible to wash out. Sprouts grown in the home are also risky if eaten raw. Many outbreaks have been linked to contaminated seed. If pathogenic bacteria are present in or on the seed, they can grow to high levels during sprouting—even under clean conditions.
- *Unpasteurized or untreated juice:* These are normally in the refrigerated sections of grocery stores, health food stores, cider mills, or farm markets.

Mothers also should be advised to wash their hands regularly.

### Alcohol

No level of alcohol consumption is considered safe during pregnancy. Alcohol can pass freely through the placenta, and thus, when the mother drinks, so does her unborn child. Consumption of alcohol during pregnancy is associated with higher risk for birth defects and miscarriage. At high levels, alcohol may cause fetal alcohol syndrome (FAS), which is the leading cause of preventable mental retardation (Chiriboga, 2003).

FAS is characterized by mental retardation, malformations of the skeletal system, malformation of the heart and brain, growth problems, central nervous system problems, poor motor skills, increased mortality, and problems with learning, memory, social interaction, attention span, problem solving, speech and/or hearing. Children with FAS can often be identified by facial features that are characteristic of babies with FAS. Characteristic features include small eyes, a short or upturned nose, flat cheeks, and thin lips. These features fade as the child grows up, but other effects do not (National Organization on Fetal Alcohol Syndrome, 2006).

It is important for mothers to understand that although FAS is usually caused by binge drinking or regular alcohol consumption during pregnancy, any alcohol intake during pregnancy may contribute to fetal damage and place the child at risk of fetal alcohol spectrum disorder (FASD). The term *FASD* is used to encompass the full spectrum of birth defects that are caused by prenatal alcohol exposure. FASD is usually split into two levels of damage: fetal alcohol syndrome, which represents the most severe damage and "fetal alcohol effects," which describes lesser damage caused by more moderate drinking.

According to the Fetal Alcohol Spectrum Disorders Center for Excellence, fetal alcohol effects are manifestations of moderate drinking during pregnancy. Physical and neurological deficiencies may be a result of fetal alcohol effects. The effect that fetal alcohol exposure has on the developing brain explains common mental and behavioral impairments characteristic of fetal alcohol exposure, such as learning disabilities, poor school performance, poor impulse control, and problems with memory, attention, and judgment.

### Illegal Drug Use

Just as with alcohol, there is no safe level of illegal drug consumption during pregnancy. Illegal drug use during pregnancy may result in miscarriage, low birth weight, premature labor, placental abruption, and fetal death. Mothers suspected of using illegal drugs during pregnancy should be referred to their doctor, who should prescribe appropriate treatment (Floyd et al., 2008).

### Cigarette Smoking

According to the American Lung Association, smoking during pregnancy is estimated to account for 20% to 30% of low-birth-weight babies, up to 14% of preterm deliveries, and about 10% of all infant deaths. Furthermore, even exposure to second-hand smoke can increase risk. Smoking during pregnancy has also been associated with an increased risk for colds, lung problems, learning disabilities, and physical growth problems for exposed children after birth.

It is strongly advised that pregnant women quit smoking for the duration of the pregnancy. Studies show that approximately half of all women are able to do this, but half do not. One study looked at the profile of women who did not quit smoking and found that they were more likely to have been heavy smokers prior to pregnancy and had more psychosocial problems compared with women who quit (Pickett, Wilkinson, & Wakschlag, 2009). If a woman is not able to quit smoking, she should be encouraged to cut her usual amount to the lowest possible level.

Helping the mother to deal with the stressors in her life may help her quit smoking. In addition to putting her in contact with food, housing, and counseling resources, there are public health resources available to help women stop smoking in addition to help she may get from her doctor. The American Lung Association also suggests the following ideas for pregnant women who wish to quit smoking: ask for help from healthcare providers, family, and friends. Make a list of reasons for wanting to quit, for herself as well as for the baby. Set a quit date—the sooner the better. If a woman is not ready to set a date, she can begin to cut down on smoking, and then she can make a plan to stop all smoking in the near future. Try the four Ds: delay, deep breathe, drink water, and do something else. If a woman slips and goes back to smoking, she should first find out what caused the slip, and then she can keep trying to quit again until she succeeds. The only failure is if she stops trying.

### Caffeine

Caffeine is one of the most widely consumed pharmacologically active substances in the world. It is found in various drinks, chocolate, and some medications. Caffeine is an important concern during pregnancy because it crosses the placenta to the baby whose metabolism is still maturing and cannot fully process it. Some studies have shown that there may be an increased risk of miscarriage among women who consume more than 300 mg a day, the equivalent of a medium cup of coffee at most fast-food coffee shops. Other potential adverse outcomes include preterm labor and low-birth-weight babies (Kuczkowski, 2009).

Although most doctors advise women that they can drink caffeine in moderation while pregnant, keeping under 300 mg per day, it is safer to avoid caffeine altogether if possible. If a woman must have caffeine, she should be advised to limit herself to one cup per day of coffee, tea, or soda. To reduce the common side effects of headache, ideally women would gradually reduce their intake prior to pregnancy. A stepwise reduction during pregnancy will also help to manage headaches that will likely result from sudden stoppage of caffeine consumption.

## Summary

Pregnancy is a critical time of human development, and anything that compromises the fetal environment may have important and lasting effects on the child's future health. It is important as a society that we prioritize helping women understand the impact that their lifestyle choices have on their children. Maximizing the health of the pregnant mother will assure her child the best start in life possible.

Nutrition is a vital component of fetal development because the baby cannot build with materials he or she does not have. Limiting exposure to damaging substances such as nicotine, caffeine, foodborne bacteria, and alcohol will also aid in the child's development. Helping women deal with unpleasant side effects of pregnancy as well as more serious ones should be a focus of prenatal support. Finally, encouraging healthful lifestyle practices during pregnancy such as moderate exercise and healthy eating will affect not only the child's long-term health, but potentially the mother's as well.

## Case Study 1

### Nutrition Prior to Pregnancy

Janet is a 25-year-old normal-weight, healthy woman planning to become pregnant within the next 6 months.

#### Questions

1. How will her need for protein, fat, and carbohydrate be different at 5 months pregnant compared to her current needs?
2. There is one vitamin and one mineral that she needs to pay special attention to making sure she gets enough of *before* she becomes pregnant. What are they and why does she need them before she is pregnant? What will happen if she is low on them and becomes pregnant?
3. If she were obese and she became pregnant, does that pose any special risks for both her and her baby?
4. If she were underweight and she became pregnant, does that pose any special risks for her and her baby?
5. Briefly discuss at least five other vitamins and minerals that she needs to get more of during pregnancy compared to before she is pregnant. Why does she need more of each one and what would happen if she were deficient in it?

## Case Study 2

### Gestational Diabetes

Nadine is a 37-year-old African American woman. She is 28 weeks gestation and has three children already. Her routine glucose screen at 24 weeks was high; she has not yet had a follow-up glucose tolerance test to determine whether she has gestational diabetes. Her weight is 244 lbs; height is 5 foot 6 inches. Her mother has type 2 diabetes. Her first baby weighed 9 lbs, and her second baby weighed 9 lbs 4 oz. She works in a hectic job as a day care provider. She often skips meals.

#### Questions

1. How likely is it that Nadine will be diagnosed with gestational diabetes? What are her risk factors?
2. Assuming Nadine is diagnosed with GDM, what recommendations would you make in terms of her nutrition management?
3. Following is a 24-hour recall for Nadine. Help Nadine modify her diet to better control her blood sugar.

   Sample 24-Hour Recall

   | | |
   |---|---|
   | 8:30 a.m. | 1½ cups orange juice, 2 slices toast (white bread) |
   | 11:00 a.m. | Mars candy bar |
   | 12:30 p.m. | 2 slices pepperoni pizza (14-inch) |
   | 5:30 p.m. | Banana chips (20 items) |
   | 8:45 p.m. | Red beans and rice (2 cups) |

4. Discuss potential negative outcomes for Nadine and her baby if she is diagnosed with gestational diabetes and she remains untreated.

## Case Study 3

### Weight Gain During Pregnancy

Maria is a 27-year-old woman with a 2-year-old child. Maria is now 14 weeks pregnant with her second child. She weighs 160 lbs and her height is 4 foot 11 inches.

Maria gained 80 lbs with her first pregnancy and has never lost most of the weight. She is at home all day with her child while her husband works two jobs. Maria drinks 4 cups of coffee a day and 3 cans of diet cola because she is so tired all the time. She also drinks fruit punch with her son when they have meals. She has not been taking her prenatal vitamin because she feels that it upsets her stomach and constipates her. She and her son often eat at fast-food restaurants or consume packaged food such as boxed macaroni and cheese or frozen chicken nuggets because that is what her son likes to eat. She eats very few fruits and vegetables and drinks no milk, although she eats some American cheese.

Maria would like to eat better but doesn't know what she should do. She also has heard that exercise during pregnancy is a good idea because it will help keep her back and legs from aching and she would like to try it.

#### Questions

1. What recommendations would you make in terms of lifestyle management for Maria?
2. How would you modify her diet?
3. Based on the IOM guidelines, how much weight should Maria gain during this pregnancy? How should the weight gain break down by trimester? What recommendations can you give her to slow her weight gain?
4. Assuming that Maria and her family are at the federal poverty line, what nutrition programs are they eligible for?

## Case Study 4

### Vegetarian Pregnancy

Arunima is a 25-year-old woman who was born in India. She is 8 weeks pregnant with her first child. For the past month, she has been very nauseous, although not vomiting, and has not been eating much. She weighs 110 lbs and her height is 5 foot 4 inches. She has lost 2 lbs since she became pregnant. She does not work outside the home; her husband works as a store manager.

She is a strict vegetarian (vegan).

#### Questions

1. Discuss interventions that may help manage Arunima's nausea.
2. At 12 weeks the nausea passes and Arunima is hungry! What recommendations would you make in terms of nutrition management for Arunima? As a vegan, what elements of her diet do you need to be concerned about? How can you modify her diet to make sure she gets everything she needs?

## References


American College of Obstetricians and Gynecologists. (2002). ACOG Committee opinion no. 267: Exercise during pregnancy and the postpartum period. *Obstetrics and Gynecology*, *99*(1), 171–173.

Aghajafari, F., Nagulesapillai, T., Ronksley, P. E., Tough, S. C., O'Beirne, M., & Rabi, D. M. (2013). Association between maternal serum 25-hydroxyvitamin D level and pregnancy and neonatal outcomes: Systematic review and meta-analysis of observational studies. *British Medical Journal*, *346*, f1169.

Ali, R. A., & Egan, L. J. (2007). Gastroesophageal reflux disease in pregnancy. Best Practice *and Research* Clinical Gastroenterology, *21*(5), 793–806.

American College of Obstetricians and Gynecologists. (2002). ACOG Committee opinion no. 267: Exercise during pregnancy and the postpartum period. *Obstetrics and Gynecology,* 99(1), 171–173.

Bager, P., Wohlfahrt, J., & Westergaard, T. (2008). Caesarean delivery and risk of atopy and allergic disease: Meta-analyses. *Clinical and Experimental Allergy*, *38*(4), 634–642.

Bansil, P., Kuklina, E. V., Whiteman, M. K., Kourtis, A. P., Posner, S. F., Johnson, C. H., & Jamieson, D. J. (2008). Eating disorders among delivery hospitalizations: Prevalence and outcomes. *Journal of Women's Health (Larchmt)*, *17*(9), 1523–1528.

Bao, W., Bowers, K., Tobias, D. K., Hu, F. B., & Zhang, C. (2013). Prepregnancy dietary protein intake, major dietary protein sources, and the risk of gestational diabetes mellitus: A prospective cohort study. *Diabetes Care*, *36*(7), 2001–2008.

Barbagallo, M., Dominguez, L. J., & Resnick, L. M. (2007). Magnesium metabolism in hypertension and type 2 diabetes mellitus. *American Journal of Therapeutics*, *14*(4), 375–385.

Bayley, T. M., Dye, L., Jones, S., DeBono, M., & Hill, A. J. (2002). Food cravings and aversions during pregnancy: Relationships with nausea and vomiting. *Appetite*, *38*(1), 45–51.

Beard, J. L. (2008). Why iron deficiency is important in infant development. *Journal of Nutrition*, *138*(12), 2534–2536.

Bourre, J. M. (2007). Dietary omega-3 fatty acids for women. *Biomedicine and Pharmacotherapy*, *61*(2–3), 105–112.

Bower, C., Miller, M., Payne, J., & Serna, P. (2005). Promotion of folate for the prevention of neural tube defects: Who benefits? *Paediatric and Perinatal Epidemiology*, *19*(6), 435–444.

Briefel, R. R., & Johnson, C. L. (2004). Secular trends in dietary intake in the United States. *Annual Review of Nutrition*, *24*, 401–431.

Butte, N. F., Wong, W. W., Treuth, M. S., Ellis, K. J., & Smith, E. (2004). Energy requirements during pregnancy based on total energy expenditure and energy deposition. *American Journal of Clinical Nutrition*, *79*(6), 1078–1087.

Centers for Disease Control and Prevention. (1992). Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects. *Morbidity and Mortality Weekly Report*, *41*, 1–8.

Centers for Disease Control and Prevention. (2004). Trends in intake of energy and macronutrients—United States, 1971–2000. *Morbidity and Mortality Weekly Report*, *53*(04), 80–82. Retrieved from www.cdc.gov/mmwr/preview/mmwrhtml/mm5304a3.htm

Cheng, Y. W., & Caughey, A. B. (2008). Gestational diabetes: Diagnosis and management. *Journal of Perinatology*, *28*(10), 657–664.

Chiriboga, C. A. (2003). Fetal alcohol and drug effects. *Neurologist*, *9*(6), 267–279.

Chu, S. Y., Callaghan, W. M., Kim, S. Y., Schmid, C. H., Lau, J., England, L. J., & Dietz, P. M. (2007). Maternal obesity and risk of gestational diabetes mellitus. *Diabetes Care*, *30*(8), 2070–2076.

Connor, W. E. (2000). Importance of n-3 fatty acids in health and disease. *American Journal of Clinical Nutrition*, *71*(1 Suppl.), 171S–175S.

Darnton-Hill, I., Nishida, C., & James, W. P. (2004). A life course approach to diet, nutrition and the prevention of chronic diseases. *Public Health and Nutrition*, *7*(1A), 101–121.

Davis, M. (2004). Nausea and vomiting of pregnancy: An evidence-based review. *Journal of Perinatal and Neonatal Nursing*, *18*(4), 312–328.

Dror, D. K., & Allen, L. H. (2012). Interventions with vitamins $B_6$, $B_{12}$ and C in pregnancy. *Paediatric and Perinatal Epidemiology*, *26*(Suppl. 1), 55–74.

Duley, L. (2003). Pre-eclampsia and the hypertensive disorders of pregnancy. *British Medical Bulletin*, *67*, 161–176.

Duerbeck, N. B., & Dowling, D. D. (2012). Vitamin A: Too much of a good thing? *Obstetrics and Gynecological Survey*, *67*(2), 122–128.

Durlach, J. (2004). New data on the importance of gestational Mg deficiency. *Journal of the American College of Nutrition*, *23*(6), 694S–700S.

Ehrenberg, H. M., Dierker, L., Milluzzi, C., & Mercer, B. M. (2003). Low maternal weight, failure to thrive in pregnancy, and adverse pregnancy outcomes. *American Journal of Obstetrics and Gynecology*, *189*(6), 1726–1730.

Floyd, R. L., Jack, B. W., Cefalo, R., Atrash, H., Mahoney, J., Herron, A., Husten, C., & Sokol, R. J. (2008). The clinical content of preconception care: Alcohol, tobacco, and illicit drug exposures. *American Journal of Obstetrics and Gynecology*, *199*(6 Suppl. 2), S333–S339.

Furneaux, E. C., Langley-Evans, A. J., & Langley-Evans, S. C. (2001). Nausea and vomiting of pregnancy: Endocrine basis and contribution to pregnancy outcome. *Obstetrics and Gynecological Survey*, *56*(12), 775–782.

Georgieff, M. K. (2008). The role of iron in neurodevelopment: Fetal iron deficiency and the developing hippocampus. *Biochemical Society Transcripts*, *36*(Pt. 6), 1267–1271.

Godfrey, K. M., Lillycrop, K. A., Burdge, G. C., Gluckman, P. D., & Hanson, M. A. (2013). Non-imprinted epigenetics in fetal and postnatal development and growth. *Nestlé Nutrition Institute Workshop Series*, *71*, 57–63.

Hampton, T. (2004). Fetal environment may have profound long-term consequences for health. *Journal of the American Medical Association*, *292*, 1285–1286.

Haram, K., Svendsen, E., & Abildgaard, U. (2009). The HELLP syndrome: Clinical issues and management: A review. *BMC Pregnancy and Childbirth*, *9*(1), 8.

Hauger, M. S., Gibbons, L., Vik, T., & Belizán, J. M. (2008). Prepregnancy weight status and the risk of adverse pregnancy outcome. *Acta Obstetricia et Gynecologica Scandinavica*, *87*(9), 953–959.

Hofmeyr, G. J., Duley, L., & Atallah, A. (2007). Dietary calcium supplementation for prevention of pre-eclampsia and related problems: A systematic review and commentary. *BJOG*, *114*(8), 933–943.

Holick, M. F. (2008). The vitamin D deficiency pandemic and consequences for nonskeletal health: Mechanisms of action. *Molecular Aspects of Medicine*, *29*(6), 361–368.

Honein, M. A., Paulozzi, L. J., Mathews, T. J., Erickson, J. D., & Wong, L. C. (2001). Impact of folic acid fortification of the U.S. food supply on the occurrence of neural tube defects. *Journal of the American Medical Association*, *285*, 2981–2986.

Horrocks, L. A., & Yeo, Y. K. (1999). Health benefits of docosahexaenoic acid (DHA). *Pharmacological Research*, *40*(3), 211–225.

Innis, S. M., & Friesen, R. W. (2008). Essential n-3 fatty acids in pregnant women and early visual acuity maturation in term infants. *American Journal of Clinical Nutrition*, *87*(3), 548–557.

Ismail, S. K., & Kenny, L. (2007). Review on hyperemesis gravidarum. *Best Practices and Research Clinical Gastroenterology*, *21*(5), 755–769.

Keller, J., Frederking, D., & Layer, P. (2008). The spectrum and treatment of gastrointestinal disorders during pregnancy. *Nature Clinical Practice of Gastroenterology and Hepatology*, *5*(8), 430–443.

Kim, C., Newton, K. M., & Knopp, R. H. (2002). Gestational diabetes and the incidence of type 2 diabetes: A systematic review. *Diabetes Care*, *25*(10), 1862–1868.

Kim, S. Y., England, L., Wilson, H. G., Bish, C., Satten, G. A., & Dietz, P. (2010). Percentage of gestational diabetes mellitus attributable to overweight and obesity. *American Journal of Public Health*, *100*(6), 1047–1052.

King, D. E., Mainous, A. G., 3rd, & Lambourne, C. A. (2012). Trends in dietary fiber intake in the United States, 1999–2008. *Journal of the Academy of Nutrition and Dietetics*, *112*(5), 642–648.

Kuczkowski, K. M. (2009). Caffeine in pregnancy. *Archives of Gynecology and Obstetrics*, *280*(5), 695–698.

Langley-Evans, S. C. (2013). Fetal programming of CVD and renal disease: Animal models and mechanistic considerations. *Proceedings of the Nutrition Society*, 1–9. doi:10.1017/S0029665112003035

Lapillonne, A., & Griffin, I. J. (2013). Feeding preterm infants today for later metabolic and cardiovascular outcomes. *Journal of Pediatrics*, *162*(3 Suppl.), S7–16.

Leung, A. M., Pearce, E. N., & Braverman, L. E. (2009). Iodine content of prenatal multivitamins in the United States. *New England Journal of Medicine*, *360*(9), 939–940.

Lucas, M. J. (2001). Diabetes complicating pregnancy. *Obstetrics and Gynecology Clinics of North America*, *28*(3), 513–536.

Markl, G. E., Strunz-Lehner, C., Egen-Lappe, V., Lack, N., & Hasford, J. (2008). The association of psychosocial factors with nausea and vomiting during pregnancy. *Journal of Psychosomatic Obstetrics and Gynecology*, *29*(1), 17–22.

Molloy, A. M., Kirke, P. N., Brody, L. C., Scott, J. M., & Mills, J. L. (2008). Effects of folate and vitamin $B_{12}$ deficiencies during pregnancy on fetal, infant, and child development. *Food and Nutrition Bulletin*, *29*(2 Suppl.), S101–S111; discussion S112–S115.

National Academy of Sciences Institute of Medicine. (1990). *Nutrition during pregnancy*. Washington, DC: National Academy of Sciences Press.

National Organization on Fetal Alcohol Syndrome. (2006). FASD Identification. Retrieved from www.nofas.org/wp-content/uploads/2012/05/identification.pdf

Nicholson, W., Bolen, S., Witkop, C. T., Neale, D., Wilson, L., & Bass, E. (2009). Benefits and risks of oral diabetes agents compared with insulin in women with gestational diabetes: A systematic review. *Obstetrics and Gynecology*, *113*(1), 193–205.

Nordin, S., Broman, D. A., Olofsson, J. K., & Wulff, M. (2004). A longitudinal descriptive study of self-reported abnormal smell and taste perception in pregnant women. *Chemical Senses*, *29*(5), 391–402.

Panel on Macronutrients, Institute of Medicine. (2002). *Report on Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Washington, DC: Institute of Medicine.

Pearce, E. N. (2007). National trends in iodine nutrition: Is everyone getting enough? *Thyroid*, *17*(9), 823–827.

Penney, D. S. (2008). The effect of vigorous exercise during pregnancy. *Journal of Midwifery and Women's Health*, *53*(2), 155–159.

Pickett, K. E., Wilkinson, R. G., & Wakschlag, L. S. (2009). The psychosocial context of pregnancy smoking and quitting in the Millennium Cohort Study. *Journal of Epidemiology and Community Health*, 63, 474–480. doi:10.1136/jech.2008.082594

Pletsch, P. K., & Kratz, A. T. (2004). Why do women stop smoking during pregnancy? Cigarettes taste and smell bad. *Health Care for Women International*, *25*(7), 671–679.

Quinla, J. D., & Hill, D. A. (2003). Nausea and vomiting of pregnancy. *American Family Physician*, *68*(1), 121–128.

Rayman, M. P., Bode, P., & Redman, C. W. (2003). Low selenium status is associated with the occurrence of the pregnancy disease preeclampsia in women from the United Kingdom. *American Journal of Obstetrics and Gynecology*, *189*, 1343–1349.

Richter, J. E. (2005). Review article: The management of heartburn in pregnancy. *Alimentary* Pharmacology *and Therapeutics*, *22*(9), 749–757.

Roseboom, T., de Rooij, S., & Painter, R. (2006). The Dutch famine and its long-term consequences for adult health. *Early Human Development*, *82*(8), 485–491.

Sayers, A., & Tobias, J. H. (2008). Estimated maternal ultraviolet B exposure levels in pregnancy influence skeletal development of the child. *Journal of Clinical Endocrinology and Metabolism, 94*(3), 765–771.

Schmidt, M. I., Duncan, B. B., Reichelt, A. J., Branchtein, L., Matos, M. C., Costa e Forti, A., … for the Brazilian Gestational Diabetes Study Group. (2001). Gestational diabetes mellitus diagnosed with a 2-h 75-g oral glucose tolerance test and adverse pregnancy outcomes. *Diabetes Care*, *24*(7), 1151–1155.

Shah, D., & Sachdev, H. P. (2006). Zinc deficiency in pregnancy and fetal outcome. *Nutrition Reviews*, *64*(1), 15–30.

Sibai, B., Dekker, G., & Kupferminc, M. (2005). Pre-eclampsia. *Lancet*, *365*(9461), 785–799.

Smith, J. L. (1999). Foodborne infections during pregnancy. *Journal of Food Protection*, *62*(7), 818–829.

Stothard, K. J., Tennant, P. W., Bell, R., & Rankin, J. (2009). Maternal overweight and obesity and the risk of congenital anomalies: A systematic review and meta-analysis. *Journal of the American Medical Association*, *301*(6), 636–650.

Strobel, M., Tinz, J., & Biesalski, H. K. (2007). The importance of beta-carotene as a source of vitamin A with special regard to pregnant and breastfeeding women. *European Journal of Nutrition*, *46*(Suppl. 1), I1–I20.

Thapa, K., & Jha, R. (2008). Magnesium sulphate: A life saving drug. *Journal of the Nepal Medical Association*, *47*(171), 104–108.

Thompson, J. N. (2007). Fetal nutrition and adult hypertension, diabetes, obesity, and coronary artery disease. *Neonatal Network*, *26*(4), 235–240.

Tiran, D. (2002). Nausea and vomiting in pregnancy: Safety and efficacy of self-administered complementary therapies. *Complementary Therapies in Nursing and Midwifery, 8*(4), 191–196.

U.S. Department of Agriculture, Food Safety and Inspection Service. (2003). *Interpretive Summary: Quantitative Assessment of the Relative Risk to Public Health from Foodborne* Listeria monocytogenes *Among Selected Categories of Ready-to-Eat Foods*. Retrieved from www.fda.gov/downloads/Food/FoodScienceResearch/UCM197329.pdf

Wallis, A. B., & Saftlas, A. F. (2008). A gram of prevention: A modest increase in fiber consumption may reduce risk of preeclampsia. *American Journal of Hypertension*, *21*(8), 849–850.

Walsh, J. W., Hasler, W. L., Nugent, C. E., & Owyang, C. (1996). Progesterone and estrogen are potential mediators of gastric slow-wave dysrhythmias in nausea of pregnancy. *American Journal of Physiology*, *270*(3 Pt. 1), G506–G514.

Watkins, M. L., Rasmussen, S. A., Honein, M. A., Botto, L. D., & Moore, C. A. (2003). Maternal obesity and risk for birth defects. *Pediatrics*, *111*, 1152–1158.

Yang, Q., Botto, L. D., Erickson, J. D., Berry, R. J., Sambell, C., Johansen, H., & Friedman, J. M. (2006). Improvement in stroke mortality in Canada and the United States, 1990 to 2002. *Circulation*, *113*(10), 1335–1343.

Yin, J., Quinn, S., Dwyer, T., Ponsonby, A. L., & Jones, G. (2012). Maternal diet, breastfeeding and adolescent body composition: A 16-year prospective study. *European Journal of Clinical Nutrition*, *66*(12), 1329–1334.

Young, G. L., & Jewell, D. (2002). Interventions for leg cramps in pregnancy. *Cochrane Database of Systematic Reviews*, (1), CD000121.

Young, S. L. (2010). Pica in pregnancy: New ideas about an old condition. *Annual Reviews in Nutrition*, *30*, 403–422.

Zeisel, S. H. (2006). The fetal origins of memory: The role of dietary choline in optimal brain development. *Journal of Pediatrics*, *149*(5 Suppl.), S131–S136.

# Special Section on Lactation Nutrition

*Lisa S. Brown, PhD, RD*



## CHAPTER OUTLINE



### Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Discuss the pros and cons of lactation.
2. Identify breastfeeding trends.

Breast milk is the only food that has evolved along with humans and so contains a highly specific balance of nutrients and non-nutrient substances designed to promote optimal infant health (Marcobal et al., 2010). Exclusive breastfeeding of infants up to 6 months of age and continued breastfeeding until 1 year is thought to be one of the most basic public health interventions to reduce infectious disease in infants and obesity and chronic disease later in life (American Academy of Pediatrics, 2012). According to Healthy People 2020, currently only approximately 33% of U.S. infants are exclusively breastfed through the first 3 months of life, and that number drops to 14% at 6 months (Healthy People 2020, n.d.).

Breastfeeding has many benefits to both infant and mother. Breastfeeding has been associated with lower risk of short-term acute infant infections including ear infections and vomiting/diarrheal illnesses. It also

is associated with lower risk of more dangerous infant illnesses including pneumonia and lower respiratory infection as well as sudden infant death syndrome (SIDS) (Meyers, 2009). Breastfeeding is associated with lower risk of immune-mediated problems in early childhood including asthma, atopic dermatitis, and type 1 diabetes. Longer-term impacts on health include observations that people who were breastfed in infancy have lower blood pressure and blood cholesterol later in life as well as lower risk of obesity and type 2 diabetes (Robinson & Fall, 2012).

For the mother, breastfeeding is associated with reduced risk of later development of breast and ovarian cancers and type 2 diabetes. Not breastfeeding and early cessation of breastfeeding is associated with higher risk of postpartum depression. Some studies have shown an association with breastfeeding and lower risk of osteoporosis for the mother, but this is still not proven (Ip et al., 2007).

## Breastfeeding Trends

Beginning in the twentieth century, American women had the option of choosing to feed their infants an artificial form of nutrition that later became known as "formula." Several innovations in the nineteenth century contributed to the widespread availability of artificial infant food including changes in the design of baby bottles, refrigeration technology to keep formula safe for later consumption, and food manufacturing technology that allowed for mass production of breast milk alternatives. Breastfeeding saw a steady decline through most of the twentieth century and by the 1950s and 1960s was seen as something only poor and uneducated women did (Stevens, Patrick, & Pickler, 2009). Many misconceptions regarding breastfeeding helped fuel the popularity of formula, among them the idea that formula was healthier for the infant. It was commonly thought that a mother's nutrition was the primary determinant of breast milk composition, and therefore a mother who did not eat a perfect diet did not produce adequate milk to support the growth of her children.

Over the past few decades it has been clearly shown with few exceptions that breast milk is generally appropriate and is usually better suited to promote the health and growth needs of infants compared to artificial feeding. In fact, breast milk not only contains ideal nutrient composition for optimal digestion, but also is created in such a way that it contains both nutritive and non-nutritive components that protect the infant from microbes and assist with immune development (Rautava & Walker, 2009). Breast milk also contains other components such as hormones that may assist with appetite regulation (Savino, Fissore, Liguori, & Oggero, 2009).

## Maternal Nutrition and Breastfeeding

### Macronutrients

The Recommended Dietary Allowances (RDAs) for most nutrients during lactation is similar to the RDAs for pregnant women. Calorie intake is set at an additional 330 kilocalories (kcal) during the first 6 months of breastfeeding and 400 kcal during the second 6 months. Carbohydrate needs increase from pregnancy requirements from 175 g per day to 210 g per day to support the high energy requirement of the infant. The RDA for protein remains at 71 g per day, the same level set for pregnant women.

The recommendation for fat intake remains at 20–35% of total calories, and as in pregnancy, consumption of essential fatty acids and specifically omega-3 fatty acids continue to be emphasized. The mother's intake of omega-3 fatty acids directly affects her milk. International studies show that essential fatty acid content in breast milk is highly correlated with local dietary patterns (Boersma, Offringa, Muskiet, Chase, & Simmons, 1991). Populations that eat more fish tend to have higher amounts of omega-3s in breast milk. If a breastfeeding mother does not eat fish at least once or twice a week, it may be beneficial for her to consume fish oil supplementation to increase the omega-3 fatty acids available to her baby.

Another nutrient that deserves special attention during lactation is water. The RDA is set for 3.8 liters (L) or 128 ounces (oz) per day. Because of the additional loss of fluid in breast milk, fluid needs are increased overall. Water and other forms of liquid in drink form or contained within food count toward the fluid needs. Caffeinated drinks are considered safe in amounts similar to those of pregnancy but should be eliminated if they appear

to affect the baby's sleep pattern. Alcoholic drinks may be consumed by a breastfeeding mother, but she should refrain from feeding her baby for a few hours following her last drink because the ethanol transfers into the milk. Waiting recommendations are similar to those for driving after having an alcoholic beverage.

### Micronutrients

Although macronutrient composition of breast milk is relatively unaffected by the nutritional intake of the mother, some micronutrients are more dependent on maternal consumption than others are. The most affected nutrients are those that are not stored in the mother's body in large quantities such as water-soluble B vitamins and vitamin C as well as minerals including zinc, selenium, and iodine (Bates & Prentice, 1994). For most micronutrients, the Reference Daily Intakes (RDIs) are similar to those for pregnant women with the notable exception of iron, which returns to the amount recommended for nonpregnant women. During exclusive breastfeeding, menses are suppressed, leading to limited iron loss. If the new mother is anemic post pregnancy, iron intake should be increased to correct the anemia. It is generally suggested that the mother continue to take her prenatal vitamin while she is breastfeeding to assist with meeting her increased needs for these nutrients.

In very rare cases, a micronutrient deficiency may develop in the mother that eventually affects her milk composition. Several case reports have found vitamin $B_{12}$ deficiencies in exclusively breastfed infants that were related to the mother's $B_{12}$ deficiency developed as a result of a previous gastric bypass surgery (Doyle, Langevin, & Zipursky, 1989; Grange & Finlay, 1994). Other case reports have connected $B_{12}$ deficiencies in exclusively breastfed infants to strict vegan mothers who did not consume supplemental $B_{12}$ from either vitamin pills or fortified vegan products (Reghu, Hosdurga, Sandhu, & Spray, 2005). Although these cases are extremely rare, they highlight the importance of dietitians and other medical professionals carefully monitoring the nutritional status of both mother and infant when the mother is on a highly restrictive diet or has potential for malabsorption of nutrients. A mother may maintain a highly restrictive diet by personal choice, as is the case with a strict vegan, or may be restricted as a result of allergies or other medical reasons. She may have an underlying eating disorder or a condition that affects her ability to absorb nutrients properly, such as having had a gastric bypass or a poorly treated medical condition related to nutrient absorption such as celiac disease.

## Summary

In summary, breastfeeding is strongly recommended as the ideal source of infant nutrition and as an important health promotion activity for both mother and child. The mother's nutrition does not need to be perfect during breastfeeding, but it is good for her to maintain a similar eating and micronutrient regimen to when she was pregnant. Mothers at high risk for nutrient deficiencies because of overly restrictive diets or malabsorption issues should be carefully monitored if they choose to exclusively breastfeed.

## References


American Academy of Pediatrics. (2012). Breastfeeding and the use of human milk. Section on Breastfeeding. *Pediatrics*, *129*(3), e827–841.

Bates, C. J., & Prentice, A. (1994). Breast milk as a source of vitamins, essential minerals and trace elements. *Pharmacology and Therapeutics*, *62*, 193–220.

Boersma, E. R., Offringa, P. J., Muskiet, F. A., Chase, W. M., & Simmons, I. J. (1991). Vitamin E, lipid fractions, and fatty acid composition of colostrum, transitional milk, and mature milk: An international comparative study. *American Journal of Clinical Nutrition*, *53*(5), 1197–1204.

Doyle, J. J., Langevin, A. M., & Zipursky, A. (1989). Nutritional vitamin $B_{12}$ deficiency in infancy: Three case reports and a review of the literature. *Pediatric Hematology-Oncology*, *6*(2), 161–172.

Grange, D. K., & Finlay, J. L. (1994). Nutritional vitamin $B_{12}$ deficiency in a breastfed infant following maternal gastric bypass. *Pediatric Hematology-Oncology*, 11(3), 311–318.

Healthy People 2020. (n.d.). *Maternal, infant, and child health*. Retrieved from www.healthypeople.gov/2020/topicsobjectives2020/objectiveslist.aspx?topicId=26

Ip, S., Chung, M., Raman, G., Chew, P., Magula, N., DeVine, D., … Lau, J. (2007). Breastfeeding and maternal and infant health outcomes in developed countries. *Evidence Report/Technology Assessment* (Full Report), (153), 1–186.

Marcobal, A., Barboza, M., Froehlich, J. W., Block, D. E., German, J. B., Lebrilla, C. B., & Mills, D. A. (2010). Consumption of human milk oligosaccharides by gut-related microbes. *Journal of Agricultural and Food Chemistry*, *58*(9), 5334–5340.

Meyers, D. (2009). Breastfeeding and health outcomes. *Breastfeeding Medicine*, *4*(s1), S-13–S-15.

Rautava, S., & Walker, W. A. (2009). Academy of Breastfeeding Medicine founder's lecture 2008: Breastfeeding—an extra-uterine link between mother and child. *Breastfeeding Medicine*, *4*(1), 3–10.

Reghu, A., Hosdurga, S., Sandhu, B., & Spray, C. (2005). Vitamin $B_{12}$ deficiency presenting as oedema in infants of vegetarian mothers. *European Journal of Pediatrics*, *164*(4), 257–258.

Robinson, S., & Fall, C. (2012). Infant nutrition and later health: A review of current evidence. *Nutrients*, *4*(8), 859–874.

Savino, F., Fissore, M. F., Liguori, S. A., & Oggero, R. (2009). Can hormones contained in mothers' milk account for the beneficial effect of breast-feeding on obesity in children? *Clinical Endocrinology* (Oxford), *71*(6), 757–765.

Stevens, E. E., Patrick, T. E., & Pickler, R. (2009). A history of infant feeding. *Journal of Perinatal Education*, *18*(2), 32–39.

# CHAPTER 2 Normal Infant Nutrition

*Jennifer L. Bueche, PhD, RD, CDN, and Rachelle Lessen, MS, RD, IBCLC*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Describe normal infant nutrition in the first year of life.
2. Compare growth differences between breastfed and formula-fed infants.
3. Describe the impact of early diet on later development of obesity, diabetes, and food allergies.
4. Discuss adequate intake of key nutrients in the first year of life, including energy, protein, fatty acids, iron, zinc, and vitamin D.
5. Describe caregiver behaviors that can affect normal transitioning from an all-milk infant diet to a diet of family foods.
6. Compare and contrast actual complementary feeding patterns with recommended guidelines.

The physiologic changes that occur during pregnancy and lactation affect nutritional requirements. These nutritional requirements include both macronutrients and micronutrients, which in turn affect the health of both mother and baby. After birth, early childhood is an important time for the development of food preferences and eating patterns. Establishment of lifelong eating habits begins in infancy and is based on a complex integration of physiologic and psychological events, including food preferences, food availability, parental modeling, praise or reward for food consumption, and peer behaviors (Stang, 2006).

## Normal Infant Nutrition

### Nutrition Recommendations for Normal Infant Feeding

Normal infant feeding is breastfeeding. Human milk is designed to support the normal growth and development of human infants. The infant's nutritional and immunologic needs are best supported by the specific components of maternal milk. In its 2012 Policy Statement the American Academy of Pediatrics (AAP) recommends exclusive breastfeeding for 6 months, followed by continued breastfeeding for 1 year or longer as complementary foods are introduced (American Academy of Pediatrics [AAP] Section on Breastfeeding, 2012) (see **Table 2.1**).

### Breast Milk Composition

Human milk is an ever-changing fluid that meets the nutritional needs of the growing infant. It varies by stage of lactation, time of day, maternal nutrition, and during a given feeding. Colostrum is the first milk that is usually available in small quantities during the first 4 days of life. It is yellow colored as a result of high levels of beta-carotene. It is high in protein, fat-soluble vitamins, minerals, and immune factors. It is lower in fat and lactose than later mature milk and therefore easier to digest. Colostrum contains bifidus factor to enhance the establishment of friendly gut bacteria *Lactobacillus bifidus* and aids in the passage of meconium from the newborn gut to reduce the risk of hyperbilirubinemia.

**TABLE 2.1 Infant Feeding Recommendations**

- Breast milk or infant formula exclusively for the first 6 months of life
- Introduction of soft or puréed foods at 6 months
- Self-feeding of soft finger foods when developmentally ready
- Cup feeding with water, milk, or juice after 6 months
- Continue breastfeeding for at least 1 year and as long as desired by mother and baby
- If not breastfeeding, formula feeding until 1 year of age
- Introduction of whole cow's milk after 12 months

Data from World Health Organization. (2003). *Global strategy for infant and young child feeding* [pamphlet]. Geneva, Switzerland: Author; and American Academy of Pediatrics Section on Breastfeeding. (2012). Breastfeeding and the use of human milk. *Pediatrics, 129*, e827–e841.

Approximately half of the calories in mature human milk come from fat. This gives the growing infant a readily available source of energy for growth requirements. The fat composition of human milk varies based on the maternal diet. The concentration of fat in milk increases during the feeding with the highest percentage occurring when the breast has been emptied. The protein in human milk is easy to digest and well absorbed. The whey-to-casein ratio changes during the course of lactation. Early milk is predominantly whey protein (80:20), whereas later milk has 50:50 whey-to-casein ratio. The total protein in human milk is the lowest of all mammals because of the slower growth of humans compared to other mammals. The primary carbohydrate in human milk is lactose, which is found in all mammalian milk. It is the least variable of all the macronutrients. It is secreted by the mammary gland and is unrelated to maternal intake. Carbohydrates provide approximately 40% of the energy in human milk. Vitamins in human milk vary with the stage of lactation, maternal stores, and dietary intake. The mineral content of human milk changes over time but is not influenced by maternal stores or intake.

The unique components of human milk offer newborn infants important immunologic protection at a time when they are most vulnerable to infection. Human milk contains immune cells, immunoglobulins, lactoferrin, cytokines, nucleotides, glycans, oligosaccharides, hormones, and bioactive peptides to aid the infant's immature immune system. Many of these components enhance infant immunity by either destroying or neutralizing pathogens or by preventing pathogens from binding to mucousal surfaces (Hanson, 2004).

### Formula Feeding

When breastfeeding is not chosen by the mother or is not possible because of maternal or infant factors, and in situations where the infant needs to be supplemented with a breast milk substitute, infant formula is an acceptable alternative for the first year of life. Recent studies have reported that there are significant risks associated with not breastfeeding (see **Table 2.2**). Infants who are exclusively formula fed from birth have increased risk of many acute and chronic illnesses compared to infants who are exclusively breastfed. The risk of otitis media is reduced by 23% for any breastfeeding compared to no breastfeeding and exclusive breastfeeding for either 3 or 6 months reduces the risk by 50% (Ip et al., 2007).

Studies have shown a 72% reduction in the risk for hospitalizations from lower respiratory tract infections for infants exclusively breastfed longer than 4 months compared to formula feeding (Ip et al., 2007). Both exclusivity and duration

**TABLE 2.2 Risks Associated with Not Breastfeeding**

- Increased incidence and severity of infection: otitis media, lower respiratory tract infection, urinary tract infection, diarrhea, bacterial meningitis, sepsis
- Increased rate of sudden infant death syndrome (SIDS), necrotizing enterocolitis (NEC), postneonatal deaths
- Increased risk of atopic dermatitis, leukemia, lymphoma, Hodgkin's disease, asthma, diabetes
- Decreased cognitive development
- Increased obesity
- Pathogen contamination in formula
- Manufacturing errors and warehouse contamination of formula
- Adulteration of formula

Data from Ip, S., Chung, M., Raman, G., Chew, P., Magula, N., DeVine, D., … Lau, J. (2007, April). *Breastfeeding and maternal and infant health outcomes in developed countries*. Evidence report/technology assessment No. 153 (Prepared by Tufts-New England Medical Center Evidence-Based Practice Center, under Contract No. 290-02-0022). AHRQ Publication No. 07-E007. Rockville, MD: Agency for Healthcare Research and Quality; and American Academy of Pediatrics Section on Breastfeeding. (2012). Breastfeeding and the use of human milk. *Pediatrics, 129*, e827–e841.

of breastfeeding have been noted to affect health outcomes. Not breastfeeding increases the infant's risk of dying. Sudden infant death syndrome (SIDS) risk is reduced by half with exclusive breastfeeding at 1 month (Vennemann et al., 2009), and any breastfeeding reduces the risk of SIDS by 36% (Ip et al., 2007). Compared to never breastfeeding, children who ever breastfed had 0.79 times reduction in postneonatal mortality from all causes, and longer breastfeeding was associated with lower risk (Chen & Rogan, 2004). If 90% of U.S. families breastfed their infants exclusively for 6 months as recommended, 911 deaths per year would be prevented (Bartick & Reinhold, 2010).

There are additional long-term outcomes associated with mode of infant feeding. Breastfeeding is associated with a reduced risk of atopic dermatitis, allergies, asthma, childhood cancer, and diabetes (Ip et al., 2007). Infants who have never been breastfed are at higher risk for later childhood obesity than are infants who have ever been breastfed. Increased breastfeeding duration is associated with lower rates of childhood obesity.

### Contraindications to Breastfeeding

There are a small number of health concerns related to the mother or the infant that may require temporary or permanent cessation of breastfeeding. Whenever alternatives to breastfeeding are considered, the benefits of breastfeeding should be compared to the risks posed by the specific condition or concern.

Infants with galactosemia, maple syrup urine disease (MSUD), or phenylketonuria (PKU) require a specialized formula to meet their nutritional needs. Infants with galactosemia cannot tolerate breast milk as a result of their metabolic disorder (AAP Section on Breastfeeding, 2012). Some breast milk may be allowed with careful monitoring for infants with PKU and MSUD (World Health Organization [WHO], 2009). Mothers who are HIV positive are advised to avoid breastfeeding and to use an acceptable substitute (WHO, 2009). If a mother has a herpes simplex virus type I lesion on the breast, direct contact between the lesion and the infant's mouth should be avoided. The baby can safely breastfeed on the unaffected breast (WHO, 2009). A mother with untreated tuberculosis should not have direct contact with her baby but may express her milk for the baby's feedings because tuberculosis is not passed through her milk (WHO, 2009).

Certain maternal medications may be of concern for breastfeeding infants, causing side effects such as drowsiness or respiratory depression and should be avoided or minimized (WHO, 2009). Mothers should be encouraged to avoid substances that have been demonstrated to have harmful effects on the baby, such as illicit drugs, opioids, and benzodiazepines (AAP Section on Breastfeeding, 2012; WHO, 2009). Although not absolutely contraindicated, it is recommended that lactating mothers wait 2 hours after ingesting no more than 2 oz liquor, 8 oz wine, or 2 beers (AAP Section on Breastfeeding, 2012; WHO, 2009) before breastfeeding. Maternal smoking should be strongly discouraged, not only because of infant respiratory effects and increased rates of SIDS but also because it is a risk factor for low milk supply and poor weight gain (AAP Section on Breastfeeding, 2012).

### Expression, Handling, and Storage of Human Milk

Mothers need to be instructed on establishing and maintaining lactation if they are separated from their babies. The first 2 weeks are critical in establishing an adequate milk supply, and mothers need support and guidance (Meier, 2001; Spatz, 2004). Mothers who cannot directly breastfeed their infant need to initiate pumping within hours of birth. They should pump and/or hand express both breasts every 2 to 3 hours, eight times daily and save this milk for the baby's feedings. Mothers need encouragement and support to establish adequate milk production during this critical period when their infants are too sick or premature to feed directly at the breast. Guidelines for storage of human milk for home use in healthy term infants are available from the Academy of Breastfeeding Medicine (ABM) and the Centers for Disease Control and Prevention (CDC).

### Donor Human Milk

In general, a healthy term newborn does not require any supplementation in the first 24–48 hours of life and should remain with the mother skin-to-skin to facilitate frequent breastfeeding (Academy of Breastfeeding Medicine Protocol Committee, 2009). If mother and baby are separated, or the mother has inadequate milk production, or the baby is not able to transfer available milk from the breast, supplementary feeding may be necessary. It is important to feed the baby appropriately while maximizing maternal milk production.

Whereas a mother's own milk is the standard food for infants and young children, there are

situations when a mother cannot provide sufficient milk for her child. In such cases, human milk from screened donors that is pasteurized is the next best option, particularly for high-risk or ill infants (AAP Section on Breastfeeding, 2012; WHO, 2003). The Human Milk Banking Association of North America (HMBANA) has developed guidelines for establishing and operating human milk banks to ensure the safety of the milk. There are currently 11 operational human milk banks in the United States and Canada and 5 that are in the developmental stages.

Holder pasteurization (62°C for 30 minutes) is used to eliminate the threat of pathogen contaminants. All milk is cultured to ensure that there is no bacterial growth after pasteurization. Milk is frozen and shipped to users in a frozen state. Most bioactive components are still active in human milk after pasteurization, and donor milk has been shown to reduce the incidence of necrotizing enterocolitis, sepsis, and infection and to result in shorter hospital stays. Donor milk improves outcomes in a variety of conditions including bowel surgery, failure to thrive, formula intolerance, allergies, leukemia, and HIV.

## Early Feeding

### Colic

Infantile colic is characterized by paroxysms of uncontrolled crying or fussing in an otherwise healthy and well-nourished infant. The crying and fussing behavior can be described by the rule of threes: It starts at 3 weeks of age, there is more than 3 hours of crying a day for at least 3 days a week, and it lasts for more than 3 weeks. Colic resolves spontaneously without any further sequelae. Some attribute this behavior to psychogenic causes from tension in the maternal–infant bond and to maternal smoking or alcohol consumption, whereas other models focus on possible allergens in breast milk or infant formula as the causative agent (Schach & Haight, 2002).

Colic can be particularly distressing for not only the infant, but for the parents as well. Colic occurs in both breastfed and formula-fed infants. Breastfeeding is not protective against colic, and it is estimated that at 6 weeks the overall prevalence of colic in all infants is 24% (Clifford, Campbell, Speechley, & Gorodzinsky, 2002). Colic appears earlier and resolves earlier in formula-fed infants. Lucas and St. James-Robert (1998) reported that at 2 weeks 43% of formula-fed infants showed signs of intense crying and colic behavior but only 16% of breastfed infants demonstrated these symptoms. By 6 weeks, distressed behavior was seen in 31% of breastfed infants but only in 12% of formula-fed infants.

In searching for a remedy for this stressful condition, attempts to modify the infant's diet may be implemented. Cow's milk protein may contribute to the etiology of colic, and removal of the protein may alleviate the symptoms. Use of a hypoallergenic formula for non-breastfed infants may be recommended (Canadian Paediatric Society, 2003). Switching to a low-lactose formula or a formula with fiber was not found to be helpful in reducing colic symptoms. A hypoallergenic maternal diet for breastfeeding mothers may reduce colicky symptoms in some infants. For some, removal of all cow's milk from the mother's diet can provide relief for the colicky breastfed infant.

#### Research About Colic

Studies have failed to show a clear link between dietary factors in either breast milk or formula as the cause of infant colic. Evidence suggests that dietary interventions may be helpful for some infants, especially those with a family history of atopic disease and severe colic. Most of the studies on the impact of dietary changes on colic have had a small sample size and were not blinded or controlled. Colic always improves over time, and any intervention is susceptible to a placebo effect affecting the outcome. Speculations as to the origins of colic abound, and treatment options are limited.

## Food Safety

### Safe Handling of Infant Formula

Infant formula should be prepared with careful attention to manufacturer's instructions for use and storage. Bottle-fed infants are at an increased risk for exposure to food-borne pathogens, particularly if the bottles are left at room temperature for several hours. Freshly expressed, but not previously frozen, breast milk contains live white cells that destroy pathogens and can remain at room temperature for up to 8 hours before feeding. All bottles, nipples, and other feeding equipment should be properly cleaned and disinfected between uses.

Powdered infant formula products are not sterile and can be a source of illness and infection in infants. At greatest risk are neonates in the first 28 days of life, premature infants, low-birth-weight infants, and immunocompromised infants. Intrinsic contamination of powdered infant formula products with

*Cronobacter sakazakii* and *Salmonella* has been a cause of significant disease, resulting in severe developmental sequelae and death (Centers for Disease Control and Prevention, 2002). The Infant Formula Act of 1980 (revised 1986) requires formula makers to use "good manufacturing practice" but does not guarantee or require sterility (Baker, 2002). Formula manufacturers are urged to develop a sterile powdered product for high-risk infants. Even low concentrations of *C. sakazakii* in powdered infant formula can cause serious harm because of the potential for exponential multiplication during preparation and holding at ambient temperatures.

Good hygienic practices, such as hand washing, using sanitized containers, and preparing only the amount needed for one feeding and using immediately, have been recommended to minimize risk (European Food Safety Authority, 2004). The WHO (WHO in collaboration with Food and Agriculture Organization of the United Nations, 2007) recommends boiling water no longer than 30 minutes and using water no cooler than 70°C (158°F) to prepare powdered infant formula. The prepared formula should be consumed immediately or refrigerated and consumed within 24 hours to prevent growth of *C. sakazakii*. The U.S. Food and Drug Administration (FDA) issued warnings regarding the use of powdered infant formula in neonatal intensive care units.

### Nitrates

Infant methemoglobinemia results in cyanosis in infants with few other clinical symptoms and is caused by nitrates in food or water that are converted to methemoglobin-producing nitrites before or after ingestion. The resulting compound, methemoglobin, cannot bind oxygen and results in hypoxemia. Absorbed nitrate that has not been converted to nitrite can be readily excreted in the urine without adverse effects. The greatest risk to infants comes from well water contaminated with nitrates (Greer, Shannon, the Committee of Nutrition, & the Committee on Environmental Health, 2005). It is estimated that 2 million families drink water from private wells that fail to meet federal drinking water standards for nitrate, and 40,000 infants younger than 6 months old live in homes that have nitrate-contaminated water supplies. Breastfed infants whose mothers consume water with high nitrate nitrogen concentrations are not at increased risk because nitrate concentration does not increase in human milk.

Nitrates also occur naturally in plants and may be concentrated in foods such as green beans, carrots, squash, spinach, and beets. Some commercially prepared infant foods are voluntarily monitored for nitrate content, and because of exceedingly high levels in spinach, this product is often labeled as not to be used for infants younger than 3 months of age. Concerns for home-prepared foods are unfounded because there is no nutritional indication for introduction of complementary foods before 6 months. The risk of methemoglobinemia decreases with age as the infant's gastric pH approaches lower levels typical of later childhood and fetal hemoglobin, which more readily oxidizes to methemoglobin, is replaced by adult hemoglobin after 3 months.

### Risks of Infant Formula and Bottle-Feeding

Infant feeding methods other than direct breastfeeding may be associated with increased risk of adverse outcomes for infants. Bottle-feeding of either expressed breast milk or formula may be of concern because of bisphenol A (BPA), a synthetic compound used in the production of hard, clear polycarbonate plastic bottles. BPA mimics the effects of estrogen, and exposure early in life is concerning because of the potential effects of BPA on the brain, behavior, and prostate gland of fetuses, infants, and children. Major manufacturers of infant feeding bottles and cups have voluntarily limited the use of BPA; however, BPA continues to be used in liners of infant formula cans (see www.fda.gov/NewsEvents/PublicHealthFocus/ucm064437.htm). Furthermore, inherent risks are associated with formula feeding because of manufacturing errors and contamination during processing. Formulas have been recalled for bad odors and contamination with beetle parts. Adverse outcomes including neurologic and cardiac effects, hospitalizations, and death have resulted from formula lacking important nutrients, including sodium chloride and thiamine, or from toxic levels of aluminum or mercury.

### Safe Handling of Complementary Foods

Infants are at risk of exposure to food-borne pathogens when complementary foods are not prepared using safe food-handling techniques. Contamination of food with microbes is recognized as the leading cause of diarrheal disease and ill health in infants. A wide range of symptoms, including diarrhea, vomiting, abdominal pain, fever, and jaundice, occurs with potentially severe and life-threatening consequences.

Two particular areas of food preparation are of concern because they allow the survival and growth of pathogens to disease-causing levels. The first is preparation of food several hours before consumption along with storage at ambient temperatures, which favors the growth of pathogens and/or toxins. The second concern is insufficient cooling of foods or inadequate reheating to reduce or eliminate pathogens (Motarjemi, 2000). General food safety guidelines for both commercially prepared and homemade infant food should be followed.

## Growth

### Adequate Growth in Infancy

In general, if a mother is well nourished and is exclusively breastfeeding, her milk will provide adequate nutrition for her infant to grow at an appropriate rate (see **Table 2.3**). Human milk has unique nutritional characteristics, such as a high ratio of whey to casein, a high proportion of nonprotein nitrogen, and fatty acids essential for brain and retinal development. It is not known at what point human milk is no longer sufficient for sustained growth, but it is unlikely that complementary foods are required before 6 months of age. Although it is commonly believed that insufficient calories and protein in human milk limit growth, it is probably more likely that other factors, such as iron and zinc, affect growth. This applies to infants in both disadvantaged and affluent populations (Dewey, 2001).

Feeding mode has been found to affect weight gain and body composition during the period of exclusive or predominant milk feeding in early infancy. Infant feeding practices are one of the few modifiable risk factors for childhood obesity. Excess weight gain in the first year of life has been found to be positively associated with the development of subsequent obesity (Druet et al., 2012; Griffiths, Smeeth, Sherburne-Hawkins, Cole, & Dezateux, 2009). The effects of breastfeeding initiation and duration on weight gain from birth to 3 years were examined in a prospective cohort study of 10,533 children in the U.K. Millennium Cohort Study (Griffiths et al., 2009). The researchers found that children not breastfed or breastfed shorter than 4 months gained weight more quickly and were both heavier and fatter at 3 years than were children breastfed longer than 4 months. The choice to bottle-feed rather than breastfeed contributes to accelerated infant growth and an increased risk for developing childhood obesity (Johnson, Wright, & Cameron, 2012).

**TABLE 2.3** Signs that Breastfeeding is Going Well

- Breastfeeding 8–12 times per day
- More than 6 wet diapers daily
- More than 3 yellow seedy stools daily
- Mother is not having any nipple pain
- Baby is gaining appropriately according to the WHO growth chart

Data from U.S. Department of Health and Human Services, Office on Women's Health. (2011, January). *Your guide to breastfeeding*. Washington, DC: Author. Retrieved from www.womenshealth.gov/publications/our-publications/breastfeeding-guide/BreastfeedingGuide-General-English.pdf

### Development of Growth Charts

The nutritional status of children is assessed by plotting height and weight on **growth charts** to determine adequacy of nutrient intake, particularly calories and protein. There is considerable evidence to show that growth rates differ for breastfed and formula-fed infants.

**growth charts** Graphs used to assess nutritional status of children by plotting height and weight and comparing these to reference data.

The World Health Organization conducted a 6-year multicenter international study to develop new growth charts derived from the growth of exclusively or predominantly breastfed infants based on the assumption that optimal infant growth occurs in infants from healthy populations who are exclusively breastfed for the first 6 months of life with continued breastfeeding until 2 years of age. The WHO Child Growth Standards (de Onis, Garza, Onyango, & Borghi, 2007) show how every child in the world should grow when free of disease and when their mothers follow healthy practices such as breastfeeding and not smoking. The standards depict normal human growth under optimal environmental conditions and can be used to assess children everywhere, regardless of ethnicity, socioeconomic status, and type of feeding (de Onis et al., 2007). These charts, released in 2006, are growth standards, as opposed to the previous charts from the CDC from 2000 that were growth references describing how certain children grew in a particular place and time. The difference is significant because the former describes the growth of healthy children in optimal environmental and health conditions, whereas the latter describes the growth patterns of a sample of children in the United States from 1963 to 1994 that lacked racial diversity and included infants who were mostly formula fed (Grummer-Strawn, Reinold, & Krebs, 2010).

In 2010, the CDC recommended that the WHO international growth charts be used for children younger than 24 months based on the recognition that breastfeeding is the recommended standard for infant feeding and that the healthy breastfed infant is the standard against which all infants

are compared. Because of the normally slower growth of breastfed infants from 3–18 months, the formula-fed infant gaining weight more rapidly on the WHO chart will be identified as at risk for the development of overweight at an earlier age (Grummer-Strawn, Reinold, & Krebs, 2010). See **Figure 2.1** and **Figure 2.2** for proper growth velocity for boys and girls birth to 1 year old.

## Nutrient Requirements

### Energy

It is difficult to estimate energy requirements for infants and young children. The 1985 Food and Agriculture Organization/WHO/United Nations Organization (FAO/WHO/UNO)

| | Percentiles (Weight Increments in Grams) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Interval** | **1st** | **3rd** | **5th** | **15th** | **25th** | **50th** | **75th** | **85th** | **95th** | **97th** | **99th** |
| 0–4 weeks | 280 | 388 | 446 | 602 | 697 | 879 | 1066 | 1171 | 1348 | 1418 | 1551 |
| 4 weeks–2 months | 410 | 519 | 578 | 734 | 829 | 1011 | 1198 | 1301 | 1476 | 1545 | 1677 |
| 2–3 months | 233 | 321 | 369 | 494 | 571 | 718 | 869 | 952 | 1094 | 1150 | 1256 |
| 3–4 months | 133 | 214 | 259 | 376 | 448 | 585 | 726 | 804 | 937 | 990 | 1090 |
| 4–5 months | 51 | 130 | 172 | 286 | 355 | 489 | 627 | 703 | 833 | 885 | 983 |
| 5–6 months | –24 | 52 | 93 | 203 | 271 | 401 | 537 | 611 | 739 | 790 | 886 |
| 6–7 months | –79 | –4 | 37 | 146 | 214 | 344 | 480 | 555 | 684 | 734 | 832 |
| 7–8 months | –119 | –44 | –2 | 109 | 178 | 311 | 450 | 526 | 659 | 711 | 811 |
| 8–9 months | –155 | –81 | –40 | 70 | 139 | 273 | 412 | 489 | 623 | 675 | 776 |
| 9–10 months | –184 | –110 | –70 | 41 | 110 | 245 | 385 | 464 | 598 | 652 | 754 |
| 10–11 months | –206 | –131 | –89 | 24 | 95 | 233 | 378 | 459 | 598 | 653 | 759 |
| 11–12 months | –222 | –145 | –102 | 15 | 88 | 232 | 383 | 467 | 612 | 670 | 781 |

**FIGURE 2.1** WHO weight gain goals: Girls monthly weight velocity

Courtesy of the World Health Organization.

| | Percentiles (Weight Increments in Grams) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Interval** | **1st** | **3rd** | **5th** | **15th** | **25th** | **50th** | **75th** | **85th** | **95th** | **97th** | **99th** |
| 0–4 weeks | 182 | 369 | 460 | 681 | 805 | 1023 | 1229 | 1336 | 1509 | 1575 | 1697 |
| 4 weeks–2 months | 528 | 648 | 713 | 886 | 992 | 1196 | 1408 | 1524 | 1724 | 1803 | 1955 |
| 2–3 months | 307 | 397 | 446 | 577 | 658 | 815 | 980 | 1071 | 1228 | 1290 | 1410 |
| 3–4 months | 160 | 241 | 285 | 403 | 476 | 617 | 764 | 845 | 985 | 1041 | 1147 |
| 4–5 months | 70 | 150 | 194 | 311 | 383 | 522 | 666 | 746 | 883 | 937 | 1041 |
| 5–6 months | –17 | 61 | 103 | 217 | 287 | 422 | 563 | 640 | 773 | 826 | 927 |
| 6–7 months | –76 | 0 | 42 | 154 | 223 | 357 | 496 | 573 | 706 | 758 | 859 |
| 7–8 months | –118 | –43 | –1 | 111 | 181 | 316 | 457 | 535 | 671 | 724 | 827 |
| 8–9 months | –153 | –77 | –36 | 77 | 148 | 285 | 429 | 508 | 646 | 701 | 806 |
| 9–10 months | –183 | –108 | –66 | 48 | 120 | 259 | 405 | 486 | 627 | 683 | 790 |
| 10–11 months | –209 | –132 | –89 | 27 | 100 | 243 | 394 | 478 | 623 | 680 | 791 |
| 11–12 months | –229 | –150 | –106 | 15 | 91 | 239 | 397 | 484 | 635 | 695 | 811 |

**FIGURE 2.2** WHO weight gain goals: Boys monthly weight velocity

Courtesy of the World Health Organization.

recommendations for energy intake were derived from the observed intakes of healthy thriving children. This assumes that the natural *ad libitum* feeding of infants and toddlers reflects desirable intake. However, the observed energy intake of infants and toddlers may not be optimal and may reflect outside influences such as type of feedings and caregiver behaviors. The FAO/WHO/UNO recommendations for energy were based on data compiled from the literature predating 1940 and up to 1980 and included an extra 5% allowance for presumed underestimation of energy intake (Institute of Medicine [IOM], 2002).

Experts questioned the validity of the 1985 FAO/WHO/UNO recommendations for energy intake, and in 1996 experts concluded that the guidelines were too high. Energy requirements for infants and toddlers based on actual energy expenditure and energy deposition or growth, rather than observed intake, would more accurately reflect true energy needs (Butte, 2005). Energy requirements of infants and young children need to support a rate of growth and body composition consistent with good health. Satisfactory growth is an indicator that energy needs are being met. Researchers in Scotland using the doubly labeled water method found that the average energy intake among healthy, exclusively breastfed infants with normal weight gain was 91 kcal/kg at 4 months and 82 kcal/kg at 6 months (Nielsen, 2011).

Butte, Wong, Hopkinson, Heinz, and colleagues (2000) used data obtained from doubly labeled water studies on infants aged 3 to 24 months to define energy requirements in the first 2 years of life based on total energy expenditure and energy deposition. Energy expenditure of infants is calculated from basal metabolic rate, thermic effect of feeding, thermoregulation, and physical activity. They were able to demonstrate that total energy expenditure was greater in older infants than in younger infants, greater for males than for females, and greater for formula-fed infants than for breastfed infants. After adjusting for body weight and fat-free mass, only feeding mode, not gender or age, influenced total energy expenditure. Energy expenditure was 12% higher in formula-fed infants at 3 months and 7% higher at 6 months than in breastfed infants (Butte, 2005). The energy cost of growth is important in early infancy when energy deposition contributes significantly to energy requirements. At 1 month of age, 40% of energy requirements are utilized for growth. This drops dramatically to 6% at 6 months and even further to 2% to 3% of total energy requirements in late infancy.

### Protein

It is not difficult to meet the protein needs of infants. Exclusively breastfed infants receive adequate protein for at least the first 6 months of life. The most recent recommended Adequate Intake (AI) of protein for infants from birth to 6 months is 1.5 g/kg/day and reflects the observed mean intake of infants who are fed mostly human milk (IOM, 2002). This value is calculated from various studies in which the volume of human milk consumed is measured by test weighing, and the average protein content of human milk was determined using values from several studies.

DuPont (2003) reported that total protein in breast milk varies greatly during the course of lactation, providing from less than 2.0 g/kg/day in the first weeks of life to approximately 1.15 g/kg/day at 4 months, less than the AI recommendations of the IOM. Dewey, Cohen, Rivera, Canahuati, and Brown (1996) reported that protein intake of breastfed infants decreases from 2.0 g/kg/day at 1 month to 1.0 g/kg/day at 6 months as protein concentration in milk decreases and average breast milk intake increases slightly. According to Dewey and colleagues, estimated daily protein intake of a 6-month-old breastfed infant is 8.0 to 8.4 g/day, lower than the calculated AI of 9.1 g/day.

Protein content of infant formula is greater than protein in human milk, but no study has shown that the amount of protein in human milk has deleterious effects. Multiple studies have shown that infants fed human milk have improved immune function and fewer illnesses than do formula-fed infants. The casein and whey in infant formula are different from those present in human milk; therefore, the digestibility, absorption, and functionality of these proteins differ. The FAO/WHO (1989) states that digestibility and comparative protein quality need to be considered when determining the amount of protein to be included in infant formula based on various protein sources. Protein requirements for formula-fed infants may be greater because of their less efficient utilization and retention of protein compared to that of breastfed infants. Dewey, Cohen, and colleagues (1996) found that adding extra protein to the diet of 4- to 6-month-old exclusively breastfed infants

did not improve weight or length gains despite an additional 20% protein in their diet, and no differences were found in growth rate based on protein intake.

The amount of protein required for growth is highest in early infancy and decreases over time. At 1 month of age 64% of protein intake is used for growth, decreasing to 24% at 6 to 12 months. Daily increments in body protein gains in male breastfed infants decreased from 1.0 g/kg/day at 1 month to 0.2 g/kg/day at 6 months. Protein needs for growth in early infancy are influenced by birth weight as well. Infants with higher birth weights generally grow at a slower rate and would require less protein than do infants born at a lower birth weight, who experience faster rates of weight gain (Dewey, Beaton, Fjeld, Lonnerdal, & Reeds, 1996). Protein requirements (both total protein and protein per kilogram of body weight) for infants older than 6 months are lower than the requirements for younger infants. High-protein follow-up formulas are not indicated or necessary for infants consuming a variety of foods (Dewey, 2000b).

Calculations for protein requirements for infants ages 7 through 12 months are based on the relationship between protein intake and nitrogen balance. Studies examining protein losses, requirements for maintenance, and protein deposition were used to derive the Dietary Reference Intake (DRI) and Recommended Dietary Allowance (RDA) for older infants of 1.5 g/kg/day, which do not differ greatly from the AI for younger infants. Higher protein intake would be indicated for a child requiring catch-up growth or recovery from an infection. Infants older than 6 months may receive a significant portion of their protein needs from complementary foods.

An adequate growth rate has traditionally been used as the determinant for sufficient protein intake. Dewey, Beaton, and colleagues (1996) reported that other measures of protein intake, such as immune function and behavioral development, may become compromised long before growth falters. Observed differences in growth rates between infants who are breastfed and formula-fed raise the question of whether maximal growth rate is synonymous with optimal growth rate. Higher intakes of protein in formula-fed infants have been a cause for concern because the liver and kidneys need to metabolize and excrete the increased levels of plasma amino acids and urea nitrogen, which could have long-term consequences on immature organs.

### Fatty Acids

Both human milk and currently available infant formula contain generous amounts of the essential fatty acids linoleic acid and alpha-linolenic acid. Cow's milk contains very little, and infants and toddlers who drink cow's milk often have low levels of these fatty acids. Corn, soybean, or safflower oil can be added to the diet of a child who has been weaned from breast milk or formula to provide these nutrients (Butte et al., 2004).

In addition to the essential fatty acids linoleic acid and alpha-linolenic acid, there is growing concern that infants also need **long-chain polyunsaturated fatty acids (LCPUFAs)** in their diets. The fetus and newborn infant are not capable of converting adequate amounts of linoleic acid and alpha-linolenic acid to docosahexaenoic acid (DHA) and arachidonic acid (AA) until 16 weeks after term age and therefore rely on an exogenous source from the mother or from milk to meet their needs for LCPUFAs (Hadders-Algra, 2010).

**long-chain polyunsaturated fatty acids (LCPUFAs)** A category of dietary fats important in brain and retinal growth; normally present in human milk, they are now added to infant formula.

These fats, particularly AA and DHA, are vital for neural development and visual acuity. Infants fed formula containing only linoleic and alpha-linolenic acid, the precursors for AA and DHA, can synthesize only a limited amount of long-chain fatty acids. When compared with breastfed infants, formula-fed infants had less mature neurophysiologic maturation and brain function (Khedr, Farghaly, Amry Sel, & Osman, 2004). These LCPUFAs are concentrated in the phospholipid bilayer of biologically active brain and retinal neural membranes during the periods of rapid brain and retinal growth from the last trimester of pregnancy until 2 years of age. During this critical period of rapid growth and maturation, the quantity and quality of the LCPUFAs may influence the efficiency of nerve cell signaling and have long-lasting effects on brain function.

In 1998, an expert panel for the FDA in the United States and a working group for the Canadian authorities did not recommend the addition of LCPUFAs to infant formula because of the uncertainties related to product safety and efficacy (Koo, 2003). In February 2002, term infant formula with added DHA and AA became readily available, and shortly thereafter DHA and AA were added to preterm infant formula. The rationale for adding these LCPUFAs to infant formula is based on the observation that they are present in large quantities

in the brains and retinas of breastfed children and are present in breast milk. Maternal plasma and human milk DHA levels as well as the infant's plasma levels can be increased by adding DHA to the maternal diet at levels of 200 mg/day or greater. However, studies have not shown marked improvement in infant outcome related to visual function and neurodevelopment in breastfed infants whose mothers received 200 mg/day DHA supplementation (Jensen et al., 2005).

A comparison of the multitude of studies on LCPUFA supplementation and visual acuity and neurodevelopmental outcomes in infants is hindered by the fact that many of the neurodevelopment tests were never designed to test normal healthy infants and lack predictive ability for long-term neurodevelopment outcomes. Although DHA and AA supplementation no doubt raises plasma levels in both infants and breastfeeding mothers, it remains highly controversial whether there is any functional benefit in visual acuity or neurodevelopment. A meta-analysis of available evidence on the effects of LCPUFA supplementation in infant formula shows that although there is a beneficial effect on short-term neurodevelopmental outcomes in full-term infants, there are no long-term effects beyond 4 months of age (Hadders-Algra, 2010). The safety of these additives is an additional concern, although there have been few reported adverse events. The oils used for the supplementation are new food products from Martek Biosciences. They have been approved by the FDA as "Generally Recognized as Safe" and are approved for use in infant formula and baby food.

The DHA used to supplement infant formula is an oil derived from microalgae, and the AA is an oil derived from soil fungi. An imbalance of omega-3 to omega-6 fatty acids could have biological effects such as prolonged bleeding time and diminished growth, and close monitoring of infants consuming infant formula with added LCPUFAs through scientific studies is indicated and indeed required by the FDA. Although families will embrace a new product promising to deliver a formula that is closer to human milk and is good for the brain and eyes, the cost of these products is a burden on the family budget and on public funding for nutrition programs. Justification of increased costs of up to 25% may prove difficult without substantial scientific evidence of improved clinical outcomes in vision and intelligence.

## Iron

**Iron-deficiency anemia** is the most common childhood nutritional deficiency worldwide, with consequences of delays in motor and cognitive development caused by irreversible brain injury. Developmental deficits occur when iron deficiency becomes severe and chronic enough to result in anemia. Although iron supplementation increases iron stores, poor developmental outcomes may persist, with lower scores on mental and motor tests and functional impairment in school-aged children.

**iron-deficiency anemia** The most common childhood deficiency resulting in motor and cognitive developmental deficits.

In the 1930s, in the United States a high prevalence of nutritional iron-deficiency anemia was first noted in infants and the prevalence has decreased dramatically since the 1960s, when it was acknowledged as a public health problem. Interventions, including an increase in breastfeeding, the start of the **Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)** in 1972, and education of physicians and the public, resulted in a dramatic decrease in iron-deficiency anemia across all socioeconomic groups in the United States. The interventions were so successful that in the 1980s it was suggested that routine screening of all infants be replaced by selective screening of high-risk patients.

**The Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)** A program that serves to safeguard the health of low-income women, infants, and children up to age 5 years who are at nutritional risk by providing nutritious foods to supplement diets, information on healthy eating, and referrals to health care.

It now appears that the prevalence of iron-deficiency anemia is increasing in 1- to 3-year-olds (Kazal, 2002). Data from the 2001 Pediatric Nutrition Surveillance System indicated that 16.6% of 6- to 11-month-old infants and 15.3% of 12- to 17-month old children were anemic. The subjects in this national sample were largely from low-income populations, where 82% were enrolled in WIC (Altucher, Rasmussen, Barden, & Habicht, 2005). It is not unusual for children ages 1 to 3 years to have low intakes of iron. Most 12-month-olds receive 100% of the daily requirement for iron, but this declines to less than recommended intake by 18 months, likely because of cessation of breastfeeding, switching from iron-fortified infant formula to cow's milk, and reduced intake of iron-fortified cereals (Kazal, 2002). Juice intake can decrease a child's appetite for other more nutritional solid foods, further contributing to iron deficiency.

Healthy, normal-birth-weight, full-term infants receive adequate iron from human milk for approximately the first 6 to 9 months of life. Reserves at birth are a critical factor for anemia. Delaying clamping of the umbilical cord for 30–120 seconds at birth improves iron status at 2–3 months (Hutton & Hassan, 2007; Van Rheenen & Brabin, 2004). Total body iron is fairly stable in infants from birth through age 4 months as stored iron is gradually used to support growth. Between 4 and 12 months there is a significant increase in iron requirements as body size increases. These needs cannot be met through the iron available in human milk. The concentration of iron in human milk is 0.2 to 0.4 mg/L and remains stable throughout lactation. Although the absolute amount of iron in human milk is low, efficiency of iron absorption from human milk is quite high, at about 50%. Once iron stores are depleted, iron-related physiologic functions may become compromised, with both cognitive and motor deficits even in the absence of anemia. After age 2 years when growth velocity decreases, iron stores start to accumulate and the risk for deficiency decreases (AAP, 2004).

The risk for anemia is much greater in low-birth-weight infants. In a study of low-birth-weight infants born in Honduras, infants with birth weights less than 3,000 g were at risk for anemia at 6 months even when iron-fortified complementary foods were introduced between ages 4 and 6 months (Dewey, Peerson, & Brown, 1998). It is recommended that low-birth-weight breastfed infants receive iron drops beginning at ages 2 to 3 months.

Fomon (2001) stated that infants who are exclusively or predominantly breastfed are at risk of becoming iron deficient by 8 to 9 months of age. Whereas Fomon recommended beginning iron supplementation of breastfed infants at an early age to prevent depletion of body stores, the AAP notes that iron deficiency is rare in breastfed infants as a result of increased absorption and the absence of microscopic blood loss in the intestinal tract that may occur with whole cow's milk. Supplementation of healthy term breastfed infants with iron to prevent deficiency is controversial. Unnecessary supplementation can increase the prevalence of gastrointestinal infection, sepsis, and cancer because iron is essential for the growth of microorganisms and malignant cells. Gastrointestinal effects such as nausea, vomiting, constipation, and abdominal pain have been reported by individuals on iron supplementation. Routine iron supplementation of breastfed infants with normal hemoglobin levels resulted in increased diarrhea and poor linear growth, possibly as the result of the pro-oxidant effect of iron on the intestinal mucosa. Reduction in zinc absorption leading to poor growth can occur from excessive iron intake (Dewey et al., 2002). Ermis, Demirel, Demircan, and Gurel (2002) found that supplementation of breastfed infants from ages 5 to 9 months with iron at a dose of 2 mg/kg every other day prevented iron deficiency and iron-deficiency anemia. Every-other-day dosing improved compliance and reduced unpleasant side effects.

Domellof, Lonnerdal, Abrams, and Hernall (2002) found that regulation of iron absorption in breastfed infants undergoes developmental changes from ages 6 to 9 months that enhance the ability of the infant to adapt to a low-iron diet. Unlike iron absorption in adults, which increases in states of iron depletion, iron absorption in infants was found to be directly related to the dietary intake rather than to iron status. Iron status as measured by serum ferritin was improved in infants given iron supplementation, but there was a significant inverse relationship between dietary iron provided and absorption of iron from human milk. Unsupplemented infants absorbed 37% of the iron in human milk compared with only 17% absorption in breastfed infants supplemented with 1 mg/kg/day. Supplemental iron drops had a greater effect on decreasing iron absorption from human milk than absorption of iron from complementary foods. Domellof and colleagues concluded that breastfeeding with the addition of complementary foods containing adequate iron likely provides sufficient iron for some, but not all, healthy 9-month-old infants, possibly because of up-regulation of iron in response to low dietary intake, thus avoiding iron deficiency.

Iron absorption from foods varies greatly from less than 1% to 50% of available iron. About 4% of the iron in fortified infant formula is absorbed versus 50% of the iron in human milk. The AAP estimates that for infants consuming iron-fortified formula there is an 8% risk for iron deficiency and less than 1% risk for iron-deficiency anemia. Infants drinking cow's milk have a 30% to 40% risk of iron deficiency by ages 9 to 12 months. Exclusively breastfed infants have a 20% risk of iron deficiency by 9 to 12 months

of age. Formula-fed infants should not be switched to whole cow's milk until after 1 year of age, and there is no medical indication for low-iron formulas. The AAP has recommended that the manufacture of low-iron formulas be discontinued because there is no scientific evidence to support the claim that iron-fortified formulas increase gastrointestinal distress in infants.

The main sources of iron from complementary foods in the infant diet are iron-fortified cereal and meats. Absorption of iron from infant cereal is only about 4%. The form of iron used in dry infant cereals is an insoluble iron salt or metallic iron powder that is used to reduce oxidative rancidity, and these forms have low bioavailability. Meat is a much better source of iron because the iron is in the heme form, with an absorption efficiency of 10% to 20%. Nonheme iron from plant foods and fortified products is less well absorbed. Plant-based foods have a high phytic acid, polyphenol, and/or dietary fiber content that can inhibit absorption of micronutrients. Ascorbic acid counteracts the effects of phytate on iron absorption by preventing it from binding with available iron.

Estimates for absorption of iron depend on the amount of animal or fish protein in a meal relative to plant-based foods. Consumption of meat, fish, or poultry enhances the absorption of nonheme iron from plant-based foods. Vitamin C in the form of fresh fruits such as cantaloupe, kiwi, or strawberries or vegetables such as broccoli and kale consumed at the same time as nonheme iron enhances absorption. Tea, bran, and milk inhibit nonheme iron absorption. Heme iron is absorbed in the intestines intact and is not affected by inhibitors of nonheme iron.

Engelmann, Sandstrom, and Michaelsen (1996) studied the effect of meat intake on hemoglobin levels in breastfed infants. When healthy 8-month-old partially breastfed infants were fed a high meat intake of 27 g/day for 2 months, they had only minimal decreases in hemoglobin of 0.6 g/L compared with a similar group of breastfed infants with a low meat intake of 10 g/day who had decreases in hemoglobin of 4.9 g/L. The group with the low meat intake had overall greater intakes of iron (3.4 versus 3.1 mg/day) but lower intakes of iron from meat (0.1 versus 0.4 mg/day), suggesting that animal muscle protein has an iron absorption–enhancing effect and can minimize decreases in hemoglobin that are typically observed from ages 8 to 10 months in breastfed infants.

Heath, Tuttle, Simons, Cleghorn, and Parnell (2002) found that 9- to 18-month-old New Zealand breastfed infants experienced low rates of iron-deficiency anemia of 7% even while their intake of breast milk and infant formula declined. Their intake of highly bioavailable iron in the form of meat, poultry, and fish increased from 0 g/day at 9 months to 21 g/day at 12 months and 32 g/day at 18 months. Their intake of vitamin C was also high, at 52 to 96 mg/day, likely further enhancing iron absorption.

Kattelmann, Ho, and Specker (2001) found that the age of introduction of complementary foods to formula-fed infants did not affect iron status parameters. Infants introduced to complementary foods early, at 3 to 4 months of age, had greater iron intakes at age 6 months but no difference in hemoglobin levels at age 12, 24, or 36 months than infants with later introduction to complementary foods at age 6 months. These infants were all formula fed and received at least the RDA for iron for the first 6 months of life.

### Zinc

The AI for zinc for infants from birth to age 6 months reflects the usual zinc intake of infants fed exclusively human milk. Zinc is crucial for growth and development (Otten, Hellwig, & Meyers, 2006). Human milk is sufficient to meet the infant's requirements for the first 6 months of life (Brown, Engle-Stone, Krebs, & Peerson, 2009; IOM, 2001). Zinc deficiency is prevalent in undernourished children and is linked to reduced activity and play with subsequent poor developmental outcomes. **Zinc deficiency** is associated with poor growth as well as diarrheal disease. Globally, there is a widespread prevalence of zinc deficiency, often along with iron deficiency, in infants and young children (Krebs et al., 2006). It is estimated that 12- to 24-month-olds only meet 50% to 60% of the DRI/RDA for zinc (Krebs, 2000). Meeks Gardner and colleagues (2005) found that when poor, undernourished Jamaican children ages 9 to 30 months were supplemented with 10 mg zinc daily and also participated in a weekly program to improve mother–child interactions, the developmental quotient and hand and eye coordination improved. Diarrheal morbidity

**zinc deficiency** Prevalent in undernourished children and associated with poor growth, poor developmental outcomes, and diarrheal disease.

was reduced, but there were no improvements in the children's growth.

Zinc concentration in human milk is low, but bioavailability is high. Neonatal stores are likely sufficient to maintain zinc homeostasis until 6 months of age. The young infant has a relatively high zinc requirement to support the rapid growth of early infancy. Zinc concentration in human milk decreases throughout lactation but continues to be an important source of zinc beyond 6 months (Otten et al., 2006). There is greater bioavailability of zinc in human milk than in cow's milk. The concentration of zinc in human milk decreases rapidly from 4 mg/L at 2 weeks to 2 mg/L at 2 months and to 1.2 mg/L at 6 months (Krebs, Reidinger, Robertson, & Hambridge, 1994). Despite an increased intake in volume over the first 6 months, this steep decline in zinc concentration in human milk results in a decline in zinc intake. Zinc concentration in human milk of well-nourished mothers is resistant to changes in the maternal diet. Although zinc supplementation is associated with improved growth, exclusively breastfed infants grow well without additional zinc. Dewey and associates (1998) found that when breastfed children received complementary foods fortified with zinc to double their average zinc intake there was no significant increase in weight or length.

Zinc absorption is greater from a diet high in animal protein, and the best source of zinc is red meat (Krebs et al., 2006). Meat provides adequate amounts to meet requirements of breastfed infants 7 to 12 months of age. Plant-based foods containing phytic acid bind with zinc in the intestines and reduce absorption. Vegetarians who rely on a plant-based diet may need to increase their zinc intake by 50% because of decreased bioavailability of zinc from phytic acid. Complementary foods based on unrefined cereals and legumes have a high phytate-to-zinc ratio and can compromise zinc status, whereas rice has a lower phytate-to-zinc ratio (Gibson & Holtz, 2000). Offering infants complementary foods of animal origin such as red meat and fish improves zinc intake and bioavailability. Supplementation with a combination of micronutrients can lead to problems of interaction and limitations of absorption. A zinc supplement given in water interferes with absorption of iron but not when both are added to food (Rossander-Hulten, Brune, Sandstrom, Lonnerdal, & Hallberg, 1991).

### Vitamin D

**rickets** Vitamin D deficiency resulting in growth deficits, developmental delay, failure to thrive, short stature, tetany, seizures, and skeletal deformities.

**Rickets** was almost universally seen in African American infants living in the northern United States at the turn of twentieth century. With the discovery of vitamin D and a public health campaign to fortify infant foods and supplement breastfed infants with cod liver oil, rickets were nearly eradicated (Rajakumar & Thomas, 2005). Once again, nutritional rickets is a public health concern. Breastfed infants are at risk for vitamin D deficiency because of limited amounts of vitamin D in breast milk and the current trend to limit sun exposure (Fomon, 2001). Vitamin D–deficiency rickets can cause significant morbidity, including delays in growth and motor development, failure to thrive, short stature, tetany, seizures, and skeletal deformities. A review of published reports from 1986 to 2001 found 166 cases of rickets in children in North Carolina, Texas, Georgia, and the mid-Atlantic region (Weisberg, Scablon, Li, & Cogswell, 2004). Most cases (83%) were African American, and 96% were breastfed. Only 5% of the breastfed infants received vitamin D supplementation, and most were weaned from the breast to a diet low in vitamin D and calcium.

Dark-skinned infants who are exclusively breastfed are at particular risk because of differences in skin melanin content and the ability to convert UV light to previtamin D3. From 1990 to 1999, in North Carolina 30 cases of nutritional rickets were seen in African American children who were breastfeeding without supplemental vitamin D, even though infants living in sunny southern states were believed to be at low risk (Kreiter et al., 2000). Infants born to African American women in the southeastern United States were found to have lower levels of vitamin D at birth compared to Caucasian infants and are at greater risk of developing rickets (Basile, Taylor, Wagner, Quinones, & Hollis, 2007). Vitamin D deficiency was found to be common among unsupplemented breastfed infants living in Iowa. It was more common in winter, when 78% of unsupplemented breastfed infants were found to be deficient. Deficiency was also observed in summer months and was more common in dark-skinned infants. Deficiency dropped with age but was reported to be 12% at 15 months of age without supplementation (Ziegler, Hollis, Melson, & Jeter, 2006). Vitamin D–deficiency rickets is common in infants

in Pakistan, Saudi Arabia, and the United Arab Emirates where breastfeeding women have limited sun exposure and a diet low in vitamin D. Infants are not routinely supplemented with vitamin D while breastfeeding, and many mothers avoid consumption of fortified dairy products. Despite abundant sunshine, only rural women who spent more time working outdoors had adequate serum levels of vitamin D (Dawodu, Adarwal, Hossain, Kochiyil, & Zayed, 2003).

Guidelines from the AAP, based on evidence from new clinical trials, recommend a daily intake of 400 IU/day of vitamin D for all infants, children, and adolescents beginning the first few days of life to prevent vitamin D–deficiency rickets and potentially to maintain innate immunity and prevent diseases such as diabetes and cancer (Wagner, Greer, and the Section on Breastfeeding and Committee on Nutrition, 2008).

Vitamin D synthesis occurs in the skin from exposure to ultraviolet B light from sunlight. Dietary sources include fish liver oils, fatty fish, and foods fortified with vitamin D, particularly cow's milk, infant formula, and breakfast cereals. Sunlight exposure may not be sufficient at higher latitudes, during winter months, or with sunscreen use. Individuals with dark skin pigmentation have limited vitamin D synthesis with sunlight exposure. Human milk typically contains 25 IU/L of vitamin D, not enough to meet the recommended requirement.

Maternal vitamin D status has a direct effect on the vitamin D content in human milk. Traditionally, sunlight exposure provided adequate vitamin D for both mothers and breastfeeding infants. For instance, in light-skinned individuals 10 to 15 minutes of total body peak sunlight exposure endogenously produce and release into circulation 20,000 IU of vitamin D. The daily recommended intake for vitamin D for lactating women is 400 IU, an amount unlikely to provide for optimal vitamin D levels in human milk. Wagner, Hulsey, Fanning, Ebeling, and Hollis (2006) found that by supplementing lactating mothers with 6,400 IU/day of vitamin D, they were able to increase maternal vitamin D levels sufficiently to increase the amount of vitamin D in human milk from 82 to 873 IU/L and to significantly improve circulating vitamin D in both the mother and the breastfed infant.

Supplementing breastfeeding mothers with high-dose vitamin D does not adversely affect the level of calcium in maternal milk and does not result in toxicity for mother or baby (Basile, Taylor, Wagner, Horst, & Hollis, 2006). In the Middle East, where severe vitamin D deficiency is a significant health problem, supplementing mothers with high-dose vitamin D and their vitamin D–deficient infants with 400 IU/day vitamin D for 3 months was associated with a three-fold increase in infants' serum vitamin D levels and a 64% reduction in the prevalence of vitamin D deficiency (Saadi et al., 2009).

### Supplemental Nutrients

Human milk continues to provide many nutrients in the second half of the first year of life during the period of **complementary feeding**. Human milk intake of infants ages 9 to 11 months meets the estimated needs for vitamin C, folate, vitamin $B_{12}$, selenium, and iodine. After 6 months, complementary foods are needed to provide 12% of vitamin A; 25–50% of copper; 50–75% thiamin, niacin, and manganese; and up to 98% of iron and zinc (Gibson & Holtz, 2000).

**complementary feeding**
The period that begins with the introduction of the first nonmilk food and ends with the cessation of breastfeeding or formula feeding.

Despite the fact that most young children receive adequate vitamins and minerals from their diet, many families supplement their child's diet with additional vitamins and minerals. Eichenberger Gilmore, Hong, Broffitt, and Levy (2005) studied trends in children younger than 2 years in Iowa and found that by 24 months 31.7% used some type of supplement. In the first 6 months of life, 3.5% to 6.3% of non-breastfed infants received supplements compared with 18.3% to 29.2% of breastfed infants. After 6 months of age, use of multiple vitamin supplements and multiple vitamin supplements with minerals increased with age. Diet alone provided AI of most vitamins and minerals, and with additional supplementation intake of vitamin A exceeded the recommended upper limit. Hypervitaminosis A is a concern because reports associate excessive vitamin A intake with decreased bone mineral density and increased risk of fracture. The long-term adverse effects of high intakes of vitamin A during early life are not clear.

Milner, Stein, McCarter, and Moon (2004) reported that early use of multivitamins increased the risk of developing food allergies and asthma. There was an association between infant multivitamin supplementation within the first 6 months of life and an increased risk of developing asthma by 3 years of age among black children. In addition, multivitamin supplementation in the first 6 months

of life was associated with increased risk for food allergies by 3 years of age in both formula-fed and breastfed infants. Early infancy may be a sensitive time for exposure to exogenous stimuli that influence the differentiation of naive T cells into either proinflammatory or anti-inflammatory cells. Vitamins may be a potent stimulus for the differentiation of T cells that promote allergic response when encountering specific antigens. Recommendations for routine vitamin supplementation may need reevaluation in light of these findings.

Fortified foods provide a significant portion of nutrient needs, reflecting a limited intake of nutritious foods such as fruits and vegetables (Fox, Reidy, Novak, & Ziegler, 2006). As infants move into the toddler stage, a decrease in naturally occurring vitamin A from vegetables is replaced by vitamin A from cereals and supplements. At the same time, two of three of the leading sources of vitamin C are fortified juices and fortified sweetened beverages rather than fruits and vegetables. Consumption of a wide variety of foods to meet nutrient needs, rather than reliance on fortified foods and supplements, is optimal. There is potential for excess intake and toxicity when vitamin A, zinc, and folate are consumed through fortified foods and supplements.

### Vegetarianism

A vegetarian diet during infancy and childhood can be adequate in all essential nutrients, with normal growth and nutritional status expected unless the diet is severely restricted. A breastfed infant of a well-nourished vegetarian mother receives adequate nutrition, particularly if the mother pays close attention to her own intake of iron, vitamins $B_{12}$ and D, and DHA. Women who consume three or more servings of dairy products receive sufficient vitamins D and $B_{12}$ from their diet. Women following a vegan diet need to supplement with foods fortified with vitamin $B_{12}$ such as nutritional yeast or soy milk. Vegan infants who are not breastfed need to receive soy infant formula until 1 year of age. Soy milk, rice milk, or homemade formulas based on grains, nuts, vegetables, or vegetable juice do not provide adequate nutrition for infants younger than 1 year old and should not be used to replace breast milk or commercial infant formula (American Dietetic Association, 2003; Mangels & Driggers, 2012).

A variety of protein-rich vegetarian foods is available for the older infant and toddler, including tofu, legumes, soy or dairy yogurt, and cheese, eggs, and cottage cheese. These foods can be easily puréed or mashed for increased acceptance when the child is first introduced to complementary foods. Later, soft-cooked beans, bean spreads or nut butters on toast, and chunks of tofu, cheese, and soy burgers can be offered as finger foods. Fat is an important source of energy and should not be restricted in children younger than 2 years of age. Vegan infants need a supplementary source of vitamin $B_{12}$ when they are weaned from breast milk or infant formula.

## Complementary Feeding

### Transitioning from All Milk to Family Foods

Complementary feeding is defined as the period extending from the first introduction of nonmilk feeds to the cessation of breastfeeding or formula feeding (Weaver, 2000). Any food that provides energy and displaces breast milk is considered a complementary food. A gradual progression from an exclusive milk diet to a variety of complementary foods allows the infant's gastrointestinal function to accommodate new types of foods, including starch, sucrose, and fiber. In many other mammals there are abrupt and well-defined changes to the intestinal mucosa and enzyme activity as weaning occurs. However, in humans these changes are less obvious and more gradual. Human milk contains many bioactive substances, including digestive enzymes such as bile salt–stimulated lipase, amylase, and protease, that may be involved in the digestion of complementary foods.

The timing of complementary feeding varies according to cultural practices and the personal beliefs of the mother as well as guidance she receives from her pediatrician. In some instances, pediatricians may suggest adding cereal to the infant's diet before age 4 months when a mother is concerned about the baby's sleeping patterns or growth. Child nutrition experts agree that there is no reason to introduce complementary foods before 4 months of age. At least 60 countries have policies in place to introduce complementary foods after 6 months of age. Earlier introduction of complementary foods can affect immune function and immunotolerance, development of chronic disease, and risk of atopy.

At birth the gut of a full-term infant may be anatomically and functionally mature, but subtle immaturities in luminal digestion, mucosal absorption,

and protective function could predispose the infant to gastrointestinal and systemic disease during the first 6 months of life. It is not known exactly when the period of immature immune function ends and when it is safe to feed foreign proteins. Early introduction can cause protein-induced enteropathies, leading to mucosal inflammation, villous atrophy, diarrhea, and failure to thrive (Muraro et al., 2004). The question of when the normal-term infant is developmentally ready to discontinue **exclusive breastfeeding** and begin the intake of solid and semisolid complementary foods is an important one. There probably is not a single optimal age for introduction of complementary foods but rather optimal ages that are determined by factors such as infant birth weight, maternal nutrition while breastfeeding, and environmental conditions.

**exclusive breastfeeding**
Infant receiving only breast milk and no other foods or drinks for the first 6 months.

### Feeding Guidelines

From 1979 until 2001, the WHO recommended that normal full-term infants be exclusively breastfed for 4 to 6 months. Later, it was found that discontinuing exclusive breastfeeding before 6 months increased infant morbidity and mortality, and the WHO recommendations were revised to encourage exclusive breastfeeding for 6 months. Early introduction of complementary foods and exposure to pathogens in food could result in symptomatic infection and illness in the infant and reduced sucking at the breast, followed by a decrease in the amount of milk and immune substances consumed as well as decreased maternal milk production from reduced demand. Exclusive breastfeeding for 6 months results in greater immunologic protection and limits exposure to pathogens at an early age when the immune system is immature. Energy and nutrients that are valuable for normal growth and development can be used for their intended purpose rather than diverted for immunologic function.

Malnutrition is the leading cause of death in children younger than 1 year of age worldwide. Inappropriate feeding practices include early cessation of exclusive breastfeeding, introducing complementary foods too early or too late, and providing nutritionally inadequate or unsafe foods. Malnourished children who survive are frequently sick and may suffer lifelong consequences. Overweight and obesity are increasing at alarming rates throughout the world and are associated with poor feeding practices that often begin in early childhood. Global strategies for infant and young child feeding can affect social and economic development.

The WHO feeding guidelines (WHO, 2003) are for generally healthy breastfed infants. These guidelines target primarily low-income countries where most children are breastfed and safe low-cost alternatives to breast milk are not readily available. No information is included for feeding premature infants or children with infections or other acute or chronic diseases that could affect nutritional status. No information is provided on feeding non-breastfed infants. An important goal of the WHO is to improve complementary feeding practices in terms of timeliness, quality, quantity, and safety to ensure adequate global nutrition. It is difficult to make recommendations regarding the optimal age for introduction of complementary foods. Ideally, the appropriate time to introduce complementary foods and the optimal duration of breastfeeding consider infant outcomes, such as growth, behavioral development, micronutrient status, the risks of infection, allergy, and impaired intestinal function as well as those of the mother, such as general health and nutritional status and return to fertility.

The American Academy of Pediatrics Section on Breastfeeding (2012) recommends that infants be exclusively breastfed for approximately 6 months, followed by continued breastfeeding for 1 year or longer as complementary foods are introduced. Aware that complementary feeding occurs prior to 6 months in some families because of family preference and the infant's developmental status, it is advised that breastfeeding continue when complementary foods are introduced. This may be important when gluten-containing cereals are introduced because breastfeeding is immunoprotective and may reduce the risk of developing celiac disease (Silano, Agostoni, & Guandalini, 2010).

The AAP Section on Breastfeeding (2005) recommends that complementary foods rich in iron be introduced gradually around 6 months of age, but because of the unique needs of individual infants the need to introduce complementary foods could occur as early as 4 months or as late as 8 months of age. Exclusive breastfeeding in the first 6 months is defined by the AAP as consumption of human milk with no supplementation of any type (no water, no juice, no nonhuman milk, and no foods) except for vitamins, minerals, and medications. During the first 6 months, water, juice, or other

liquids are unnecessary, even in hot climates, and can introduce contaminants or allergens.

Exclusive breastfeeding provides protection from many acute and chronic diseases. Breastfeeding is also more likely to continue for at least the first year of life when infants are exclusively breastfed for the first 6 months. For optimal health benefits, breastfeeding should continue for 12 months or longer. The recommendations of the WHO are to breastfeed for at least 2 years, and the AAP states that there is no upper limit to the duration of breastfeeding and no evidence of psychological or developmental harm from breastfeeding into the third year of life or longer (AAP Section on Breastfeeding, 2005). Indeed, in late infancy breast milk provides significant amounts of energy and micronutrients and is a key source of PUFAs crucial for brain development and neurologic function (Villalpando, 2000).

### Complementary Foods and Growth

Introducing complementary foods to the exclusively breastfed infant before 6 months does not increase total calorie intake or improve growth. A breastfed infant who receives complementary foods at 4 months decreases his or her intake of human milk to maintain the same level of calories. Dewey (2000a) expressed concern that the improper introduction of complementary foods has the potential to adversely affect breast milk intake and breastfeeding duration. Providing excess energy in the form of complementary foods can reduce the intake of breast milk. Breastfed infants reduce their intake of human milk as non–breast milk foods and fluids are introduced. Although it appears that the timing of breastfeeding in relation to complementary foods (e.g., offering complementary foods before or after breastfeeding) does not seem to affect overall breast milk intake, there is a paucity of information on the effect of the introduction of complementary foods on breastfeeding.

Complementary foods displace human milk, and the infant receives fewer immune factors and is at greater risk for infection. No significant improvement in weight or length was observed by Dewey (2001) in infants up to 12 months of age when they received complementary foods at 4 months compared with infants who received exclusive human milk until 6 months.

Despite recommendations to exclusively breastfeed for the first 6 months of life, this practice may be uncommon. In the developing world, exclusive breastfeeding rates for 6 months are 37%, ranging from 22% in West and Central Africa to 45% in South Asia (Alipui, 2012). In the United States, where the national goal is for 25% of mothers to exclusively breastfeed for 6 months, overall 16.3% of mothers meet this goal (CDC, 2012).

Carruth, Skinner, Houck, and Moran (2000) found that in a study of 94 mothers, 60 added solid food by 4 months and 8 were feeding cereal in a bottle. The median age for introduction of cereal was 4 months and for juice 4½ months. Thirteen mothers introduced cereal, juice, or fruit as early as 2 months. In looking at the growth of the children who had early solids, there was no association between the age of introduction of complementary foods and change in weight or length from 2 to 8 months or from 12 to 24 months. These results were similar to the WHO data (WHO Working Group on the Growth Reference Protocol & WHO Task Force on Methods for the Natural Regulation of Fertility, 2002) showing only minor differences in growth among infants receiving complementary foods at different ages. The WHO data were based on a unique longitudinal seven-country study of predominantly breastfed infants. The study found little evidence of risk or benefit related to growth based on timing of introduction or types or frequency of complementary foods in healthy infants living in environments without major economic restraints and with low rates of illness.

The amount of energy and nutrients needed from complementary foods depends on the amount of breast milk or formula the infant is consuming. Although it is possible for an infant to receive adequate nutrition for the first year solely from iron-fortified formula, all infants need complementary foods for exposure to novel tastes and textures and to develop appropriate feeding skills. A variety of flavors and foods is important in the first 2 years of life and may increase the likelihood that children will try new foods.

**● Learning Point** Food-related behaviors are established early in life, and how and what an infant or toddler eats in the first years of life can influence later food choices.

### Meal Patterns and Nutrient Intakes

During infancy and early childhood, lifelong patterns of eating are formed that can influence later eating habits and overall health. As the

child makes the transition from an all-milk diet to sharing foods from the family table, meeting nutritional guidelines may become more problematic for families. Families strive to follow nutrition recommendations to provide the best for their children, particularly in the year after birth when the infant is eating mostly jarred baby foods. As the infant matures and begins to incorporate more of the family foods into his or her diet, less than optimal nutrition becomes evident. Transitioning from baby food to table food has been associated with a decreased intake of vitamin A, iron, and folate at meals as baby food fruits, vegetables, and meats are not replaced with equivalent table foods.

Children imitate what others around them are eating, and if no one else eats fruits or vegetables, the young child will quickly abandon these foods. When other family members drink sweetened carbonated beverages and eat high-calorie low-nutrient-dense foods, such as french fries, donuts, and potato chips, the young child will happily join in. As rates of obesity, excess weight, and type 2 diabetes are increasing in children, it is evident that the influence of early diet needs to be addressed. At a surprisingly young age, many infants and toddlers do not receive a variety of fruits and vegetables and instead are consuming high-calorie, high-fat, salty snacks and sweetened drinks. It is the parent's responsibility to offer nutritious foods to infants and toddlers. Although a preference for sweet foods is innate, repeated exposure at an early age and observing other family members eating these foods increase the likelihood that young children will develop preferences for these foods. The disappearance of the family meal and the increase in the number of meals eaten outside the home and from fast-food establishments have concerned experts as the prevalence of snacking and the quantity and quality of foods consumed by children and adolescents in recent years have changed.

**Learning Point** Breastfeeding exposes the infant to a variety of flavors from the very beginning through mother's milk. Repeated exposures to a new food may be necessary before it is accepted.

## Food Trends and Preferences

Designed to describe infant and toddler feeding patterns and food group consumption patterns, the **Feeding Infants and Toddlers Study (FITS)** was a cross-sectional telephone study using a national random sample of 3,022 infants and toddlers between 4 and 24 months of age in 2002 and of 3,273 infants and toddlers 0–47 months in 2008 along with a subsample for 2-day dietary recalls. The sample size was sufficiently large to categorize data by age groups: 4 to 6 months, 7 to 11 months, and 12 to 24 months. Conducted first in 2002 and again in 2008, the study consisted of up to three telephone interviews to collect data on growth, development, and feeding patterns. The studies had large sample sizes and were representative of ethnicity of the general population. They also included a large proportion of breastfed infants. Subsequent to the FITS 2002 study, childhood obesity emerged as a major public health concern. Efforts to curb obesity included focusing on the quality of foods consumed by preschoolers; therefore, the upper age of FITS 2008 was extended to 4 years to better assess the food environment of the preschool population (Briefel, 2010).The FITS survey collected data on food choices and their nutritional impact, feeding practices and patterns, and infant and toddler growth and developmental milestones (Devaney, Kalb, et al., 2004). The study was sponsored by Gerber Products Company and provided in-depth nutritional information about feeding behaviors of infants and toddlers and compared intakes with the newly developed DRIs. The data from 2008 were compared to the data from 2002 to identify shifts in food consumption patterns over time. Positive trends were observed related to breastfeeding practices. The percentage of infants currently breastfeeding increased from 26% in 2002 to 43% in 2008 for 4- to 5.9-month-old infants, from 27% to 37% for infants 6–8.9 months, and from 21% to 37% for infants 9–11.9 months (Siega-Riz et al., 2010).

**Feeding Infants and Toddlers Study (FITS)** This study was conducted on a large random sample of infants and toddlers to collect data on food choices and feeding practices and their impact on growth and development.

Overall, FITS found that most infants and toddlers in the United States were receiving adequate nutrition without getting excessive amounts of nutrients. Inadequacies were noted in FITS 2008, where 12% of infants 6–11 months had inadequate iron intakes and 6% had inadequate zinc intake (Butte et al., 2010). The mean energy intakes were lower in FITS 2008 than in FITS 2002 because the consumption of many desserts and sweets was reduced. Energy intake reported

by parents was often greater than those recommended by the new DRI standard for energy, called the Estimated Energy Requirement (EER). The mean energy intake exceeded the EER by 10% for infants ages 4–6 months, by 23% for infants ages 7–12 months, and by 31% for ages 12–24 months (Devaney, Ziegler, Pac, Karwe, & Barr, 2004). For infants younger than 6 months of age, the largest discrepancies were for those receiving complementary foods in addition to breast milk or formula. The FITS infants and toddlers consumed more energy than recommended on average for all age groups. Overreporting of food intake by caregivers is possible because parents may perceive the amount of food consumed reflects their success as providers. Parents may also have difficulty accurately assessing food intake because of spillage and a discrepancy between foods offered and foods consumed. However, with 10% of 2- to 5-year-old children considered overweight, it is plausible that overfeeding is occurring in infants and toddlers. Early exposures to fruits and vegetables or to foods high in energy, sugar, and fat at this critical time period can influence food preferences and dietary habits later in life.

In 2002, complementary foods continued to be introduced at ages earlier than recommended by experts, and 29% of all infants were introduced to infant cereals or puréed foods before 4 months of age (Briefel, Reidy, Karwe, & Devaney, 2004). Improvements were noted in 2008 as fewer children were introduced to complementary food before 6 months (65% vs. 50% for infant cereal; 35% vs. 17% for baby food fruit, and 31% vs. 24% for baby food vegetables, for 2002 and 2008, respectively). Although it is recommended that foods rich in iron be introduced as complementary foods at 6 months, fewer children were consuming baby food meats and iron-fortified cereals in 2008 compared to 2002 (Siega-Riz et al., 2010).

There was no significant difference in the mean age of introduction of complementary foods or cow's milk according to income level or ethnicity or in children ever breastfed compared with those never breastfed. The contribution of commercial baby foods and beverages to energy consumption peaks at ages 7 to 8 months and declines as table food intake increases (Briefel, Reidy, Karwe, Jankowski, & Hendricks, 2004).

The FITS study revealed disturbing trends regarding the food consumption of infants and toddlers. Early food preferences can predict future eating behaviors, and an alarming percentage of young children have already developed suboptimal food consumption patterns. Not surprisingly, food consumption patterns in infants and toddlers reflect the typical eating patterns of older children and adults, such as diets lacking in fruits and vegetables with high intakes of readily available low-nutrient snacks and beverages. Daily consumption of fruits and vegetables is the cornerstone of a healthy diet, and a wide variety is encouraged. In 2008, there continued to be a substantial proportion of infants and toddlers who did not receive any fruits or vegetables in a given day. Among 6- to 8.9-month-olds, 35% did not have any fruit or vegetable, and among 9- to 11.9-month-olds 64% did not have any vegetables and 19% did not have any fruit (Siega-Riz et al., 2010). Fewer than 10% of infants and toddlers consumed dark leafy vegetables at any age. Intake of deep yellow vegetables decreased as infants transitioned from commercial baby food to table food. Commercial baby food was the main source of fruits and vegetables until 9 months of age when children were offered a greater percentage of cooked vegetables and fresh fruits. Bananas were the most commonly consumed fruit, followed by apples, and few children received citrus, melon, or berries.

Establishment of healthy patterns of beverage consumption including milk with meals is important for adequate calcium intake during the years of active bone growth in childhood and adolescence. Water is a better choice for quenching thirst than sweetened juice drinks or carbonated beverages are. Consumption of sweetened beverages changed little from 2002 to 2008. Sweetened beverages were consumed by 5% of 6- to 8.9-month-olds and 10% of 9- to 11.9-month-olds. During the second year, this increased to one-third of all toddlers drinking some form of sweetened beverage. FITS found that colas, fruit-flavored carbonated drinks, and carbonated mineral water were consumed by an increasing percentage of children from ages 4 through 24 months. Substitution of fruit drinks or carbonated beverages for milk at lunch, dinner, and snacks was evident after 15 months (Skinner, Ziegler, & Ponza, 2004). Fruit juice is not a necessary component of the diet of infants and toddlers and, if used at all, should be introduced after 6 months of age and limited to 8 ounces per day (Kleinman, 2000a). Fruit juice consumption has decreased overall but continues to be common among all age groups. Introduction before

6 months decreased from 19% in 2002 to 7% in 2008. However, at 1 year more than half of all toddlers drank fruit juice. Adverse gastrointestinal reactions to pear or apple juice are possible because of poor absorption of fructose and sorbitol (Fomon, 2001). Offering juice in a bottle after teeth have erupted can increase the risk of dental caries and should be avoided.

According to the AAP, limited amounts of 100% juice amounting to 4 to 6 ounces per day can be offered after 6 months of age, yet Skinner, Ziegler, and Ponza (2004) found from FITS that 22% of infants were introduced to juice earlier. Juice consumption increases dramatically with age. Ten percent of toddlers ages 15 to 24 months consume more than 14 ounces of juice per day. Fruit drinks are also popular, and 5% of toddlers consume more than 16 ounces of fruit drinks a day. The AAP does not provide recommendations for limitations on fruit drinks or carbonated beverages but states that they are not equivalent to 100% juice and should not be considered a fruit serving. Most fruit juices and fruit drinks contain added vitamin C and provide 20% to 30% of the daily vitamin C requirements, but apple juice, the most commonly consumed juice, contains little vitamin A and folate, nutrients commonly obtained from fruits and vegetables. Overfeeding juice and juice drinks can displace more nutritious beverage options such as milk and water and can be associated with excessive calorie intake and risk of obesity in older children.

Desserts and sweets are introduced at a surprisingly early age, with 5% of 4- to 6-month-olds already consuming a dessert, sweet, or sweetened beverage daily (Siega-Riz et al., 2010). These numbers increased dramatically after age 9 months, when nearly half were consuming one or more foods in this category. Parents should offer age-appropriate finger foods such as soft fresh fruits, diced canned fruit, well-cooked vegetables, and easily dissolvable fortified grains such as unsweetened ready-to-eat cereals.

No controlled studies address the practical aspects of introducing foods for the first time. Although feeding guidelines for parents abound, there is no evidence for a benefit of introducing one particular food first or at any particular rate. The AAP suggests that when complementary food introduction is initiated after 6 months of age, the order of the specific food introduction is not critical. Mixing cereal with breast milk may enhance acceptance of solid foods by breastfed infants. Foods commonly consumed by infants at 1 year of age include cereals and fruits. FITS data showed that infant cereal was the most common source of grains in young infants, but even by ages 7 to 8 months many infants were consuming ready-to-eat cereals, crackers, pretzels, rice cakes, breads, and rolls (Fox, Pac, Devaney, & Jankowski, 2004). After 9 to 11 months, the number of infants receiving infant cereal declined. This was replaced by other noninfant ready-to-eat and cooked cereals, including presweetened cereals. Many presweetened cereals are comparable in vitamins and minerals with unsweetened ready-to-eat cereals, but their use in this age group may lead to preference for sweetened foods.

Infants rarely consume meat. Krebs (2000) suggested that meat intake for breastfed infants at 6 months would adequately support both iron and zinc requirements in this age group. Introduction of red meat is desirable by age 6 months because of the high bioavailability of iron (Fomon, 2001). Offering plain single meats promotes the goals of complementary feeding, which is to gradually increase the variety of flavors and textures in the diet. The formula-fed infant is less reliant on complementary foods for iron and zinc. The addition of cereal to complement the intake of protein and energy from formula is considered adequate (Wharton, 2000). In FITS, few infants were receiving any type of meat, and often the meat appeared in commercially prepared baby food dinners. Fewer than 5% of infants in any age group received plain baby food meats. After 9 to 11 months of age, non–baby food meats were offered, with chicken or turkey the most common, followed by hot dogs, sausages, and cold cuts. Fewer children received beef, fish, and pork and there was a decline in consumption of these foods by infants and toddlers from 2002 to 2008. By 12 months of age, less nutritious high-fat deli meats were the second most commonly consumed source of meat. Popular nonmeat protein sources include cheese, eggs, and yogurt (Skinner, Ziegler, Pac, & Devaney, 2004). Few children were fed dried beans, peas, vegetarian meat substitutes, and foods such as chili, rice and beans, and other bean mixtures. Peanut butter, seeds, and nuts were rarely offered before 2 years of age, likely because of concern regarding the development of allergies.

Cow's milk and cow's milk products make a significant contribution to nutritional intake during the period of complementary feeding.

If breastfeeding continues into the second year of life and the diet contains a reasonable amount of animal protein in the form of meat, fish, poultry, or eggs, most infants thrive without the addition of dairy products to their diet (Michaelsen, 2000). In FITS, nearly all children younger than 24 months of age consumed some form of milk daily. Infant formula was consumed by 82% of 7- to 8-month-olds in 2002 and 75% in 2008 and decreased as cow's milk was introduced. More than 90% of infant formula consumed was iron fortified, and about 10% consumed soy-based formula.

Of concern in the 2008 survey was the use of cow's milk by 5% of infants age 6 to 8.9 months and 17% of infants 9 to 11.9 months (Siega-Riz et al., 2010). AAP recommendations are to wait until after the child's first birthday before introducing whole cow's milk. Furthermore, it was found that 14–33% of toddlers ages 12 to 23.9 months were drinking reduced-fat milk. Although, the AAP suggests waiting until 2 years to transition to reduced-fat milk, the American Heart Association recommends 2% milk in the second year of life. Cow's milk has an undesirably high renal solute load compared with infant formula and is a significant concern for children at risk of dehydration. Iron in cow's milk is low and poorly absorbed, and feeding non-heat-treated cow's milk can cause microscopic gastrointestinal bleeding in infants, resulting in loss of iron and anemia. Cow's milk is low in essential fatty acids, zinc, vitamin C, and niacin and is high in saturated fats. Recommendations to delay cow's milk introduction until 12 months of age are mainly focused on prevention of iron-deficiency anemia (Michaelsen, 2000).

## Feeding Skills and Neuromuscular Development

### Reflexes

A normal progression of sucking and feeding reflexes is necessary for the child to advance from a milk-only diet to consumption of foods from the family diet. Swallowing is present in early fetal life at the end of the first trimester. The fetus has ample opportunities to practice by swallowing amniotic fluid even before the development of the **sucking reflex**, which appears by the middle of the second trimester. The sucking reflex is quite strong in the newborn and can be easily elicited by stroking the infant's lips, cheeks, or inside the mouth. By about 3 months of age, sucking becomes less automatic and more voluntary. The **gag reflex** is present in the third trimester and is stimulated by contact of the posterior two-thirds of the tongue. This reflex gradually diminishes to one-fourth of the posterior tongue by 6 months of age. The **rooting reflex**, which assists the infant to locate the breast and nipple by turning the head side to side and opening the mouth wide when the skin surrounding the mouth is stroked, disappears by 3 months of age.

**sucking reflex** Strong reflex present in the newborn elicited by stroking the infant's lips, cheeks, or inside the mouth.

**gag reflex** Reflex stimulated by contact with the tongue that gradually diminishes over the first 6 months.

**rooting reflex** Reflex present from birth to age 3 months that assists the infant to locate the breast and nipple by turning the head side to side and opening the mouth wide when the mouth is stroked.

### Advanced Motor Skills

Infants need new oral motor skills to transition from a full liquid milk-based diet to a more solid diet of complementary foods. Disappearance of the rooting and sucking reflexes and the accompanying changes in anatomy help prepare the infant for this transition. Phasic biting, resulting in the rhythmic opening and closing of the jaw when the gums are stimulated, disappears between 3 and 4 months of age. Between 6 and 9 months it becomes possible for the infant to receive a bolus of food without reflexively pushing it out of the mouth. By 12 months of age, rotary chewing is well established, along with sustained controlled biting that permits the infant to consume a variety of foods (Kleinman, 2000c).

During the first 2 years of life, there is increasing head and torso control that permits a child to achieve developmental milestones required for proper self-feeding abilities. Finger coordination to permit self-finger feeding usually is adequate by 6 to 7 months of age. The infant must be able to sufficiently stabilize the head and balance the trunk before he or she can sit without support and use arm and hand movements for self-feeding. Carruth, Ziegler, Gordon, and Hendricks (2004) found that one-third of 4- to 6-month-old infants and 99% of 9- to 11-month-olds can sit alone without support. Stability of the trunk is crucial in the process of progressing to complementary foods, and by 6 months most infants have achieved greater strength in the trunk, shoulder, and neck muscles. There is a wide range of ages when feeding skills emerge, and it is crucial that caregivers allow ample opportunities

for appropriate exploratory activities. Offering the child a variety of nutritious foods and allowing him or her to self-feed when appropriate skill has sufficiently developed will not jeopardize adequate nutrient intake.

Beginning at 6 months, most infants are ready for puréed, mashed, and semisolid foods. By 7 months, soft foods that can be pressed down by the infant's tongue can be introduced, and at 9 months the infant can handle foods that can be compressed by the gums. Teeth are not necessary for chewing soft lumpy foods. The ability to handle advanced textures increases day by day, and children require multiple opportunities to practice new feeding skills. By 8 months, they can progress to finger foods they can pick up and feed themselves. By 12 months of age, most children can transition to the same diet as the rest of the family, keeping in mind the need for calorically and nutrient-dense foods because of the smaller portion size. Infants possessing self-feeding skills are reported to have higher energy and nutrient intakes (Carruth et al., 2004). Foods that are a choking risk that can lodge in the trachea, such as grapes, nuts, hard raw vegetables and fruits, and popcorn, should be avoided. Introduction to a cup usually occurs after 6 months, and by 12 months most infants are drinking from a "sippy cup."

### Chewing Ability

Advances in gross motor skills parallel advances in dentition as the first primary teeth erupt at 7 to 8 months and continue throughout the first 2 years, with approximately 15 teeth by 19 to 24 months of age. Carruth and coworkers (2004) found the ability to consume foods that required chewing increases with age. Nutrient intakes of energy, fat, protein, vitamin $B_6$, vitamin $B_{12}$, folate, zinc, thiamin, niacin, and magnesium were greater for infants younger than 1 year of age who were able to eat foods that required chewing. Individual differences in the age of eruption of teeth can influence the ability to chew certain foods, especially meat and fibrous vegetables.

Feeding difficulties, particularly difficulty with chewing tough or fibrous foods, in Japanese children are thought to be caused by inappropriate transition from a milk-based diet to a diet of family foods. Sakashita, Inoue, and Kamegai (2004) found that at 2 years of age many preschool children swallowed without chewing or were unable to chew and swallow certain foods and that many kindergarten children did not chew properly, retained food in the side of their mouth, or frequently spit food out. A transitional diet containing very soft and puréed foods for an extended period has been suspected of preventing children from developing proper masticatory system and chewing and swallowing ability. Leafy vegetables were usually offered early as a weaning food but were not well accepted because the fiber makes them difficult to chew. Meats were often introduced later than recommended, possibly because of parental concerns related to food allergies (Sakashita, Inoue, & Tatsuki, 2003). In Japan, foods were specially cooked and fed to children from a spoon, inhibiting the proper development of the masticatory system and mature chewing and swallowing behavior.

### Determinants of Food Acceptance

Sakashita and colleagues (2004) found that acceptance of new foods was greatest in children who were offered food prepared from the family table and was lowest in children fed jarred baby food. Offering infants foods prepared from the family table promotes feeding progress by giving the infant an opportunity to experience a variety of food textures from an early age. Infants first offered lumpy solid foods between ages 6 and 9 months had fewer feeding difficulties and improved acceptance than infants not introduced to these foods until after age 10 months. Observing other family members eating at the family table and having the opportunity to try new foods are also important components of transitioning an infant to family foods. Sakashita and colleagues (2004) found that first-born children experience more feeding difficulties than did second- or third-born children. This may be a result of limited opportunities to observe other family members eating and to learn feeding behavior from older siblings.

The number of accepted foods increases rapidly from 6 months to 1 year and continues to increase throughout the first 2 years. Foods requiring significant chewing before swallowing, such as leafy vegetables and sliced meat, may be poorly accepted. Processed sliced deli meats are often more readily accepted when offered. Because chewing ability affects ability to swallow and therefore food acceptance, breastfed infants who have more opportunity to develop the masticatory system have a higher rate of food acceptance than bottle-fed infants do. Exposure to food flavors through mother's milk also prepares the infant for a variety of flavors.

Breastfeeding seems to facilitate increased acceptance of different foods as a result of the greater variation in breast milk flavors compared with infant formula.

Other causes of food refusal include dislike of the taste or smell and an unfamiliar appearance. Often, a child's food preference reflects those of other family members. Early food experiences can be imprinted on the memory, and when children refuse to eat vegetables at an early age, these food preferences may remain throughout the childhood and adolescent years with significant health consequences. Child-feeding practices contribute to the development of food intake controls and energy balance and can affect childhood obesity. Obese individuals tend to prefer fatty foods to fruits and vegetables and dislike tough or fibrous texture. Exposure to fruits and vegetables in infancy and early childhood should be encouraged to reduce risk factors for obesity and obesity-related diseases.

### Caregiver Behaviors

Although early childhood malnutrition can be attributable to poverty and lack of resources, family and caregiver characteristics, such as education and household management or coping skills of the mother, can determine normal growth and development. Lack of knowledge regarding appropriate foods and feeding practices can contribute to malnutrition to a greater degree than lack of food. Not only is providing the appropriate combination of complementary foods to meet the child's nutritional needs important, feeding practices such as frequency of feeds and feeding style need to be considered. Caregiving behaviors that have been identified as promoting normal growth and development are (1) active or interactive feeding, (2) selecting foods appropriate to the child's motor skills and taste preferences, (3) feeding in response to the child's hunger cues, (4) feeding in a nondistracting safe environment, and (5) talking and playing with the child in the context of the meal. This type of responsive parenting has been described as sensitive and supportive caregiving associated with good growth and development. Feeding interactions should include the caregiver observing the infant's intake and nonverbal cues and responding accordingly (Pelto, 2000). If children refuse many foods, parents should be encouraged to be creative and experiment with different food combinations, tastes, and textures. Parents should be taught to encourage children to eat, but never to force because this can lead to aversion to food and behavioral problems.

### Effect of Feeding Mode in Infancy

In early infancy, parents choose whether the child will be breastfed or bottle-fed and whether human milk or formula will be consumed. They may also control the timing of the feedings and the volume consumed, although this is less likely when the infant is breastfed. When a mother breastfeeds and her infant's sucking slows or stops, the mother assumes the child is satisfied and is finished eating. The amount of milk consumed is primarily under the infant's control. Breastfed infants are able to adjust the amount of milk consumed to maintain a constant energy intake. Formula-feeding mothers may rely on visual cues of formula remaining in the bottle and encourage the infant to continue feeding after he or she has exhibited signs of satiety.

Taveras and associates (2004) found that the longer a mother breastfed, the less likely she was to restrict her child's intake at 1 year. Compared with mothers who formula fed, mothers who exclusively breastfed for 6 months were less likely to restrict their child's intake. Breastfeeding for at least 12 months was associated with lower levels of controlling feedings and resulted in improved intake by toddlers (Orlet Fisher, Birch, Smiciklas-Wright, & Picciano, 2000). Breastfeeding may protect against obesity by allowing the infant to naturally regulate energy intake based on hunger cues and by preventing parents from overriding these cues by controlling the feeding. Mothers who breastfeed may be more responsive to their infants' signals regarding the timing and volume of feedings. Not only does the mode of feeding influence weight gain patterns in the first year of life but also the type of milk. Infants who are fed human milk by bottle gain more weight per month compared to infants fed at the breast, indicating that mode of milk delivery may influence later development of obesity. Compared to infants receiving human milk by bottle, infants receiving nonhuman milk by bottle had higher rates of weight gain (Li, Magadia, Fein, & Grummer-Strawn, 2012).

### Feeding Relationship

As the child transitions to a variety of family foods, the need to be independent and autonomous becomes evident in the feeding relationship

as the child assumes more control of his or her eating. The feeding relationship reflects the overall parent–child relationship, and feeding struggles may be indicative of other difficulties involving parent–child interactions. Feeding is a major area of frequent daily exchanges between the parent and the child, reflecting the characteristics of both the parent and the child that can either support or hinder the child's development. Feeding involves more than providing the correct mix of calories and vitamins to ensure adequate nutrition. The feeding relationship itself is crucial for the child's growth and development (Slaughter & Bryant, 2004). Feeding is a blend of nutrition, parenting, and human development and provides an opportunity for parents to be present and to provide love, support, and attention that can affect the child's physical, social, and emotional health.

As infants progress from a milk-based diet to sharing family foods, they develop unique likes and dislikes regarding the foods they are offered and communicate these preferences to their parents. How the parents respond to this assertiveness can affect the child's developing sense of self and autonomy. The ability to refuse food and have this be accepted by the parents is paramount to future interactions between the child and the parents and provides a base for all future social interactions. It is important for the child's development to be able to say "no" and still be unconditionally loved and supported. If the parent withholds love from the child or forces or pressures the child to eat, the child feels helpless and abandoned. Furthermore, the child learns that he or she does not have the ability to say "no" and be respected, which can have far-reaching effects. By allowing a child to refuse to eat a certain food or to not eat at all because he or she is not hungry, parents are giving the child permission to express his or her needs without fear of repercussions.

High levels of maternal control over when and what children eat are associated with increased adiposity and an increased desire to consume restricted foods. Maternal restrictive feeding practices have been found to increase the child's preference for the restricted food and to promote overeating when the restricted foods are available and are counterproductive in preventing obesity (Birch, Orlet Fisher, & Krahnstoever Davison, 2003). In place of restricting desirable foods, parents should be taught skills that help children learn how to consume appropriate portion sizes, to like healthy foods, and to recognize hunger and satiety cues to determine when and how much to eat.

### Portion Size

Children demonstrate an innate ability for self-regulation of energy intake. They can compensate for changes in energy density by adjusting the quantity of food they consume. Parents and caregivers potentially interfere with this natural hunger-driven mechanism by coercing children to eat when they are not hungry or by directing them to "finish their plate" or to "take one more bite" when they have demonstrated signs of satiety. Overrestriction of intake to prevent overeating in infants and toddlers can have negative consequences by preventing the natural development of feeding self-regulation. **Table 2.4** indicates food types and corresponding development infants usually demonstrate; some variances should be expected.

The presence of self-regulation of dietary intake in infants and toddlers was confirmed by analysis of the relationship among portion size, number of eating occasions, number of unique foods, and energy density (Fox, Devaney, Reidy, Razafindrakoto, & Ziegler, 2006). Children who ate less often during the day consumed larger portions, and children who ate more often ate smaller portions. For infants, energy density was negatively associated with portion size. As the energy density increased, portion size decreased, and as energy density decreased, portion size increased. The number of different foods consumed by 6- to 11-month-olds was also positively associated with portion size, indicating that infants with a more varied diet consume larger portions.

Children younger than 2 years of age typically eat seven times a day, although the number of meals and snacks reported ranges from 3 to 15. It is appropriate for infants and toddlers to consume many small meals and snacks because of their small stomachs and high energy demands. Snacks often provide about 25% of a toddler's energy intake (Skinner, Ziegler, Pac & Devaney, 2004). The breakfast, lunch, dinner, and snacks pattern emerges at ages 7 to 8 months and is well established by 9 to 11 months.

**TABLE 2.4** Infant Feeding by Age and Development

| Age | Development | What to Feed |
|---|---|---|
| Birth to 6 months | • Baby can suck and swallow.<br>• Baby should be held for feeding. | • Breast milk is best.<br>• Use formula if not breastfeeding.<br>• No water or juice. |
| 6–8 months | • Baby can sit with support and control head movement.<br>• Spoon-feeding begins.<br>• No honey entire first year. | • Breastfed infants: begin pureed meats first and then eggs, pureed fruits and vegetables, and infant cereal.<br>• Formula-fed infants: begin infant cereals and then pureed fruits, vegetables, and meats and eggs.<br>• Wait 3–5 days between new foods.<br>• Watch for signs of food allergies such as rash, vomiting, or diarrhea. |
| 7–9 months | • Baby can chew, grasp, and hold items.<br>• Finger feeding begins.<br>• Introduce a cup with water, juice, breast milk, or formula. | • Try well-cooked carrots, sliced bananas, unsweetened dry cereals, graham crackers, soft cheeses, pancake bits, and well-cooked pasta. |
| 9–12 months | • Baby can eat with a spoon and will feed self more often.<br>• Expect baby to eat with hands and make a mess. | • Offer new tastes and textures such as plain yogurt, cottage cheese, tofu, and refried beans.<br>• Offer soft foods from the family meal.<br>• Limit juice to 4 oz/day.<br>• Offer fewer pureed foods and more foods from the family meal.<br>• Always try to eat together as a family.<br>• Parents should set a good example by eating fruits and vegetables.<br>• Avoid dangerous foods that are a choking hazard: raw vegetables, nuts, seeds, whole grapes or cherry tomatoes, hot dogs, popcorn, and spoonfuls of peanut butter. |
| 1 year and beyond | • Encourage self-feeding.<br>• Continue breastfeeding.<br>• Wean from bottle.<br>• Begin offering whole cow's milk in cup. No low-fat or skim milk until 2 years of age. | • Infant should eat three meals and two to three snacks each day.<br>• Feeding should be a happy time for the entire family.<br>• Let infant decide when enough is enough.<br>• Never force infant to eat or drink.<br>• No sweetened drinks or soda.<br>• Avoid sweets. Offer fruit for dessert. |

## Special Supplemental Nutrition Program for Women, Infants, and Children

Pregnancy, infancy, and early childhood are critical periods of rapid growth and development. Nutritional insult during this time can have far-reaching consequences on cognitive and emotional health and can adversely affect health outcomes. The U.S. Department of Agriculture's Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) is the largest governmental nutrition program and is designed to assist low-income, nutritionally at-risk, pregnant, breastfeeding and non-breastfeeding postpartum women and infants and children up to 5 years of age. Millions of families have benefited from the WIC program, and it has successfully improved the nutrient intakes of its participants, particularly by reducing the prevalence of iron-deficiency anemia and improving physical, emotional, and cognitive development. Federal regulations specify that state agencies must encourage and support breastfeeding through specific activities such as nutritional education and peer-counseling programs. Peer counseling has been shown to increase WIC participation, increase breastfeeding initiation and duration, and reduce supplementation (Bronner, Barber, & Miele, 2001; Bronner, Barber, Vogelhut, & Kovar Resnick, 2001).

Nearly half of all mothers of infants born in the United States each year are enrolled in WIC (Jensen & Labbok, 2011). Participants are eligible to receive vouchers for supplemental food packages to meet their nutritional needs. All mothers have the option to receive infant formula, and as a result approximately 54% of all infant formula sold in the United States is for WIC participants. Enhanced food packages for breastfeeding mothers were created in 2009 based on recommendations from the Institute of Medicine to encourage more women to breastfeed. Yet, the retail and perceived market value of the formula packages exceed the value of the breastfeeding package and may limit the impact of the food packages.

Although women participating in the WIC program have lower rates of breastfeeding initiation, duration, and exclusivity than do women of similar socioeconomic backgrounds who are eligible for but do not participate in WIC (68% vs. 78% initiation, 34% vs. 48% at 6 months, and 18% vs. 31% at 1 year), breastfeeding rates have increased for WIC participants (see the 2013 Breastfeeding Report Card at www.cdc.gov/breastfeeding/pdf/2013BreastfeedingReportCard.pdf) From 2000 to 2007, rates for any breastfeeding for WIC participants increased from 62% to 68%, although 6-month, 1-year, and exclusive breastfeeding rates did not increase. WIC programs throughout the United States are an important resource for providing much needed lactation education and support for breastfeeding families.

### EFFECT OF EARLY DIET ON HEALTH OUTCOMES

It is well known that a relationship exists between many chronic diseases and nutrition. It has been postulated that the diet during infancy and early childhood can impact the progression of chronic diseases that develop later in life, such as cancer, obesity, diabetes, hypertension, allergy, and osteoporosis. Premature weaning, or not breastfeeding, is associated with health risks. Both short-term and long-term health outcomes have been described. The degree to which these health outcomes are realized depends on the duration, frequency and exclusivity of breastfeeding. The Agency for Healthcare Research and Quality (AHRQ) screened over 9,000 abstracts from studies conducted in developed countries across the world. They reviewed 43 primary studies on infant health outcomes and 29 systematic reviews or meta-analyses that covered 400 individual studies. They concluded that there was substantial evidence that there is significant risk to health outcomes when human milk is not provided.

#### Obesity

Increasing trends in childhood obesity, with its associated comorbidities and the likelihood of persistence of obesity into adulthood, compelled researchers to investigate preventive strategies. Approximately 10–20% of infants and toddlers in the United States are overweight, and nearly 10% of infants and toddlers from birth to 2 years are obese (Dattilo et al., 2012). Treatment of childhood obesity is costly and rarely effective. Interventions to adjust caloric intake or increase energy expenditure after infancy have little impact on children's weight or adiposity. Childhood obesity is associated not only with adult obesity but also with adverse health outcomes in adulthood independent of weight status. The AAP Section on Breastfeeding (2012) suggests that any campaign to reduce obesity should begin with breastfeeding support. One of the critical periods of attainment of excess weight is in infancy. Indeed, the more rapid and earlier an infant gains excess weight, the more likely he or she is to have undesirable weight later in life. Preventing early excess weight gain through parental feeding practices begins at birth.

A study by Stettler and associates (2005) found that in formula-fed infants, weight gain in the first week of life may be a critical determinant for the development of obesity in later life. Formula feeding is associated with a more rapid increase in weight gain in early infancy and an increased risk for obesity in childhood and adolescence.

An earlier multicenter cohort study by Stettler, Zemel, Kumanyika, and Stallings (2002) demonstrated that a pattern of rapid weight gain during the first 4 months of life was associated with an increased risk of overweight status at 7 years, independent of birth weight and weight at 1 year. For each 100 g of weight gain increase per month, the risk of overweight status at 7 years was increased by 30%. There was a clear association between the rate of early weight gain and childhood overweight status. The greatest proportional weight gain in postnatal life occurs during the time when birth weight is doubled by 4 to 6 months, and this may correspond with a critical period for energy balance regulation mechanisms.

Harder, Bergmann, Kallischnigg, and Plagemann (2005) performed a comprehensive meta-analysis of 17 studies on duration of breastfeeding and risk of overweight in later life. They found that the duration of breastfeeding is inversely and linearly associated with the risk of overweight. The risk of obesity is reduced by 4% for each month of breastfeeding. The dose–response effect continued to decrease the risk of obesity with longer breastfeeding duration. Compared to less than 1 month of breastfeeding, breastfeeding for 9 months showed a 30% risk reduction.

Martorell, Stein, and Schroeder (2001) reviewed and critiqued the literature to determine whether nutrition in early life predisposes individuals to be overweight later in life. They looked at three plausible hypotheses: (1) overnutrition increases the risk of later excess weight; (2) undernutrition, at the other extreme, also is a risk for excess weight; and (3) optimal nutrition during infancy represented by breastfeeding is protective of future obesity. They found the link between undernutrition in infancy and later obesity contradictory and inconsistent. Intrauterine overnutrition, high birth weight, and gestational diabetes were found to be associated with later obesity. Breastfeeding was found to have an enduring influence on the development of subsequent obesity.

Owen, Martin, Whincup, Davey Smith, and Cook (2005) published a quantitative review of the effects of infant feeding on the risk of obesity later in life. Initial breastfeeding protected against obesity later in life, and the association was stronger with prolonged breastfeeding. The consistency of the association they found with increasing age suggested a protective effect of early breastfeeding that was independent of dietary and physical activity patterns later in life. Confounding by maternal factors such as social class and obesity, both of which are associated with childhood obesity and a tendency to formula feed, was a limitation of the observational studies.

Hediger, Overpeck, Kuczmarski, and Ruan (2001) found a reduced risk of obesity for ever breastfed 3- to 5-year-olds compared with

those never breastfed, but they found a much stronger association with maternal obesity. Scott, Ng, and Cobiac (2012) found in a study of 2,066 Australian children aged 9 to 16 years that compared to those never breastfed, children who were breastfed more than 6 months were less likely to be overweight (adjusted odds ratio 0.64) or obese (adjusted odds ratio 0.51) after adjusting for maternal characteristics and children's age, gender, mean energy intake, level of activity, screen time, and sleep duration. Von Kries and colleagues (1999) studied 9,357 German children at the time they entered school at ages 5 and 6 years. They found a remarkably consistent, protective, and dose-dependent effect of breastfeeding on excess weight and obesity. This cross-sectional study found that obesity was reduced by 35% when children were breastfed for 3 to 5 months. This protective effect was not attributable to social class or lifestyle factors and remained significant after adjusting for potential confounding factors. Gillman and colleagues (2001) found that adolescents who were mostly or only fed breast milk in the first 6 months of life were at a 22% lower risk of being overweight than were adolescents who were only formula fed. They found an estimated 8% reduction in the risk of adolescent obesity for every 3 months of breastfeeding. Compared to formula-fed infants, breastfed infants are less likely to receive complementary foods before 4 months and are less frequently offered high-fat or high-sugar-containing foods at 1 year. Early introduction of complementary foods is positively associated with rate of weight gain in infants, toddlers, and children (Dattilo et al., 2012). Among infants who were never breastfed or who stopped breastfeeding before 4 months, the introduction of complementary foods before 4 months was associated with a sixfold increase in the odds of obesity at 3 years (Huh, Rifas-Shiman, Taveras, Oken, & Gillman, 2011).

Bergmann and coworkers (2003) found that maternal obesity, bottle-feeding, maternal smoking during pregnancy, and low socioeconomic status were risk factors for becoming overweight and having high adiposity at age 6 years in a longitudinal study of German children from birth. At age 3 months, body mass index and triceps skin-fold thickness were already significantly higher in the children who were formula fed. Children who were formula fed continued to have a higher prevalence of excess weight and obesity, and the findings remained stable after adjusting for maternal weight, maternal smoking, and socioeconomic status. Gale and associates (2012) conducted a systematic review and meta-analysis to compare infant body composition in breastfed and formula-fed infants and found altered body composition in formula-fed infants. Fat-free mass was higher at 3 to 4 months, at 8 to 9 months, and at 12 months, and fat mass was lower at 3 to 4 months and at 6 months compared to breastfed infants. At 12 months, the trend was reversed and fat mass was higher in formula-fed infants. This switch from higher adiposity in breastfed infants at 3–4 months to higher adiposity in formula-fed infants at 12 months may reflect the effects of early metabolic programming, appetite regulation, and abnormal adipose tissue development in formula-fed infants.

Questions regarding the optimal duration of exclusive breastfeeding or whether combining breastfeeding with formula supplementation may weaken the preventive influence of breastfeeding need to be addressed. Gillman and colleagues (2001) found that infants who received more breast milk than formula in the first 6 months of life had a lower risk of obesity in older childhood and adolescence than did children who received mostly or only formula. In a retrospective cohort study, Bogen, Hanusa, and Whitaker (2004) found that in a population of low-income families breastfeeding was associated with a reduced risk of obesity at age 4 years only among white children whose mothers did not smoke in pregnancy and only when breastfeeding continued for at least 16 weeks without formula or at least 26 weeks with formula.

Several explanations are offered for the protective effect of breastfeeding against obesity. Breast milk production is stimulated by the infant's sucking, and it is unlikely that rapid weight gain in an exclusively breastfed infant is a result of overfeeding. A breastfed infant establishes a point of satiety based on internal physiologic cues rather than on external social cues. Children can naturally regulate their energy intake, but parents' behavior can override the child's appetite signals. It is possible that during bottle feeding parents exhibit more control of the feeding and prevent self-regulation by the child. Parents who do not recognize the child's hunger and satiety cues may contribute to the risk of later obesity. Overfeeding in infancy may increase adipose number and fat content at a critical time period and prevent development of lifelong patterns of healthy appetite regulation that would protect against the risk of obesity.

Metabolic consequences of ingesting human milk may help regulate appetite and food consumption. Leptin, a hormone that regulates food intake and energy metabolism, is present in human milk. In a study by Savino, Costamagna, Prino, Oggero, and Silvestro (2002), serum leptin levels were higher in breastfed infants than in formula-fed infants. High levels of appetite-regulating hormones and growth factors (leptin, insulin, and glucose) and inflammatory factors (interleukin 6 and tumor necrosis factor alpha) found in human milk may be bioactive and influence the accrual of fat and lean body mass in breastfed infants (Fields & Demerath, 2012). The effects of human milk components on appetite regulation may be diminished when the milk is expressed and fed in a bottle. Compared to feeding directly at the breast, expressed human milk in a bottle is associated with increased intake, poorer self-regulation, and greater weight gain. The more bottles the infant receives, the greater the weight gain (Li et al., 2012).

Breastfeeding may help to program the infant against later energy imbalance (Gillman et al., 2001). Owen and associates (2005) suggested that breastfeeding affects intake of calories and protein, insulin secretion, and modulation of fat deposition and adipocyte development. A higher protein to nitrogen content of infant formula might induce a metabolic response of increased insulin production in formula-fed infants, leading to excessive weight gain. Protective mechanisms of breastfeeding are difficult to identify because many of the same factors associated with obesity, such as race, ethnicity, maternal education, social status, and maternal obesity, are also associated with the initiation and duration of breastfeeding or the decision to formula feed. The effects of breastfeeding on the later development of obesity can be sustained and persist into adulthood either through learned behavior or perhaps through a more complex programming mechanism.

### Allergies

Most food allergies are acquired in the first year or two of life. Sensitization often occurs with the first exposure to an antigen. Data suggest that there has been a rise in the prevalence of food allergies during the past 10–20 years (Burks et al., 2011).

The prevalence of food allergy in children 0–2 years of age is 6.3%. Milk allergies are the most commonly reported, followed by peanut and egg. Less common allergies are reported for shellfish, fish, tree nuts, strawberries, wheat, and soy (Gupta et al., 2011). Symptoms manifest as urticaria, angioedema, anaphylaxis, atopic dermatitis, respiratory symptoms, or gastrointestinal disorders. Children with food allergies are more likely to have related conditions such as asthma, atopic dermatitis, and respiratory allergies compared to children without food allergies. Severe reactions occur in 39% of allergic children and are more common in children with tree nut, peanut, shellfish, fish, and soy allergies. The odds of a severe reaction increases with age, and there is a twofold increase in risk of severe reaction in adolescents compared to children ages 0–2 years.

Food allergies can be classified as (1) IgE mediated, with symptoms such as angioedema, urticaria, wheezing, rhinitis, vomiting, eczema, and anaphylaxis reactions; (2) mixed gastrointestinal syndromes involving both IgE- and T-cell-mediated components, such as

eosinophilic esophagitis; or (3) non-IgE-mediated allergies, such as protein-induced enterocolitis. Public health strategies for primary prevention of food allergies are necessary, but results of studies to determine the etiology of food allergies are conflicting. Breastfeeding is presumed to be protective, but study results vary and the size of the effect is controversial. Ingesting small quantities of allergens via breast milk in the presence of anti-inflammatory cytokines may actually offer protection against subsequent development of food allergy later in life.

Recent recommendations to avoid common allergens in infancy to prevent the development of food allergies has come under scrutiny by researchers and public health policy makers. Early, rather than late, exposure to food allergens may promote tolerance during this "critical early window" of development. A Cochrane review concluded that exclusive breastfeeding for 6 months compared to 3 to 4 months with partial breastfeeding to 6 months reduced morbidity from gastrointestinal disease but did not further reduce the risk of allergic disease (Kramer & Kakuma, 2012). Although the WHO recommends 6 months of exclusive breastfeeding for optimal health outcomes, there is an emerging body of evidence that introduction of complementary foods from 4–6 months may reduce the risk of development of food allergies. Delaying introduction of common food allergens may hinder the development of normal discrimination, reduce oral tolerance, and result in inappropriate allergic responses (Bammann, 2012; Kneepkens & Brand, 2010; Prescott et al., 2008). The KOALA (Kind, Ouders en gezondheid: Aandacht voor Leefstijl en Aanleg) Birth Cohort Study of 2,558 infants in the Netherlands found that a delay in the introduction of cow's milk products was associated with a higher risk for eczema and that delayed introduction of other foods was associated with a greater risk of atopy at 2 years (Snijders, Thijs, van Ree, & van den Brandt, 2008).

Postponement of strongly allergenic foods, such as peanuts and eggs, into the second and third of year of life not only does not prevent allergies but is suspected to actually increase the prevalence of food allergies. DuToit and coworkers (2008) found that Israeli schoolchildren typically consume 7 g peanut protein per day from 8–14 months and have a prevalence of 0.17% peanut allergy compared to children in the United Kingdom, who consume no peanut protein at this age and have a prevalence of 1.85% peanut allergy. Koplin and colleagues (2010) found that among 12-month-old infants with confirmed egg allergies, those introduced to egg at 10–12 months had an increased risk of OR 1.6 and those introduced after 12 months had an even greater risk of OR 3.5 compared to those introduced to egg between 4 and 6 months. Breastfeeding until 6 months of age with the introduction of complementary foods between 4 and 6 months has been suggested as a strategy for allergy prevention (Burks et al., 2011; Greer, Sicherer, Burks, American Academy of Pediatrics Committee on Nutrition, & American Academy of Pediatrics Section on Allergy and Immunology, 2008). Restricting maternal diet during pregnancy or lactation is not recommended to prevent allergies (Burks et al., 2011; Greer et al., 2008).

Early introduction of foreign proteins, including cow's milk, wheat, soy, rice, eggs, fish, and chicken, could induce a T-cell-mediated immune reaction of the intestinal mucosa associated with inflammation, villous atrophy, diarrhea, and failure to thrive (Schmitz, 2000). The earlier these foods are introduced to the infant, the greater the risk of developing enteropathy. These enteropathies are linked to the immaturity of the gut's immune system, leading to sensitization rather than to tolerance when exposed to foreign proteins. It is unknown precisely when the gut has matured sufficiently to accept foreign protein, although it is unlikely to occur in the first few months of life. Infants with dietary-induced proctocolitis, a non-IgE-mediated allergy, appear healthy but have visible specks or streaks of blood in their stool. Blood loss is minimal, and anemia is rare. This type of allergy is not usually associated with vomiting, diarrhea, or growth failure and often is caused by sensitivity to cow's milk or soy protein, often through the maternal diet while breastfeeding (Sicherer, 2003). Breastfed infants who develop allergic proctocolitis should not be limited in their exposure to other major food allergens. Infants and their breastfeeding mothers should only avoid the foods that have been identified through a maternal elimination diet as triggering symptoms (Academy of Breastfeeding Medicine, 2011).

Atopic dermatitis is a chronic skin condition often seen in young children and is often the first sign of allergic sensitization in infants. The pathophysiology remains unclear, but it is increased in families with a history of atopic disorders, suggesting a genetic component. In infancy, atopic dermatitis is closely related to both IgE- and non-IgE-mediated food hypersensitivities that occur in formula-fed and breastfed infants. Intact food allergens, particularly from cow's milk, eggs, and peanuts, may be secreted in small quantities by the mammary gland epithelium, causing a reaction with the mucosal immune system in the infant's intestinal lumen (Heine, Hill, & Hosking, 2004).

Use of soy or hypoallergenic infant formula as primary prevention of milk allergy is controversial. The actual prevalence of milk protein allergy in infancy is only 2–3%. Because of the increased costs of using a hypoallergenic formula, their use should be limited to infants with well-defined clinical symptoms. Infants with cow's milk allergies should not be fed milk from goats, sheep, or other animals because of the likelihood of allergic reaction to other mammalian milk. Soy milk is often used as a substitute for cow's milk infant formula and may be well tolerated. Soy formula feeding is not recommended for primary prevention of allergies in high-risk infants (Greer et al., 2008). Infants with IgE-mediated cow's milk allergies may have better tolerance to soy than do infants with non-IgE-mediated symptoms. Eight percent to 14% of infants with IgE-mediated cow's milk allergies have adverse reactions to soy, although anaphylaxis is rare. A higher prevalence of concomitant reactions (25–60%) is seen when soy is fed to infants with non-IgE-mediated cow's milk allergies; therefore, soy is not recommended as a substitute for infants with proctocolitis and enterocolitis reactions. For these children, an extensively hydrolyzed protein formula or a free amino-acid-based infant formula should be used. Benefits should be seen within 2 to 4 weeks, and the formula should be continued until the infant is at least 1 year of age. There is no evidence that there are increased benefits to partially or extensively hydrolyzed infant formula compared to exclusive breastfeeding in the prevention of atopic disease (Greer et al., 2008).

A family history of allergy, defined as both parents or one parent and one sibling with **allergic disease**, is the strongest predictor of allergic disease in children. In high-risk infants, up to 6% of exclusively breastfed infants developed food-specific IgE allergies with symptoms occurring with the first reported direct food exposure. However, in the general population food allergy in exclusively breastfed infants ranges from 0.04% to 0.5% (Zeiger, 2003).

**allergic disease**
Sensitization to allergens manifested as urticaria, angioedema, anaphylaxis, atopic dermatitis, respiratory symptoms, or gastrointestinal disorder.

Formula feeding and early exposure to potential food allergens are risk factors for atopic disease. Studies suggest that combined maternal avoidance of food allergens while breastfeeding and infant avoidance of allergens for at least the first 6 months may reduce eczema and food allergy in early childhood (Zeiger, 2003). Data supporting a protective effect on respiratory allergy and asthma in later childhood are less compelling. Exclusive, rather than partial, breastfeeding for at least 4 months appears to have a significant impact on the occurrence of atopic dermatitis, allergic rhinitis, and respiratory symptoms (Kill, Wickman, Lilja, Nordvall, & Pershagen, 2002). High-risk infants should be breastfed throughout the first year of life or longer or, alternately, fed with a hypoallergenic formula. Breast milk contains immunomodulating properties to regulate the immune system and also contains milk-specific IgA to bind cow's milk allergens and prevent allergies.

Food antigens have been detected in the milk of mothers after consumption of allergenic foods such as cow's milk, eggs, wheat, and peanuts, and the concentrations are sufficient to trigger reactions in allergic children (Zeiger, 2003). Concentrations of antigens in human milk depend on the amount consumed by the mother and appear in human milk 1 to 6 hours after ingestion. Peanut protein may be quickly cleared from the milk within 3 hours after ingestion. Secretion of antigens into human milk is variable. In a study by Vadas, Wai, Burks, and Perelman (2001), only 48% of lactating women secreted peanut protein in their milk after ingestion of peanuts. Similarly, approximately two-thirds of women secrete cow's milk and egg protein into their milk after ingestion.

Characteristics of women who secrete food antigens into their milk have not been accounted for, making it difficult to determine preventive strategies. One study from Finland found that a maternal diet high in saturated fat and low in vitamin C while breastfeeding was associated with an increased risk of atopic sensitization in the infant (Joppu, Kalliomaki, & Isolauri, 2000). Allergic disease has also been linked to a maternal diet with a high ratio of omega-6 to omega-3 fatty acids, typical of Western diets containing processed and fried foods (Koletzko, 2000). Fish oil supplementation to provide omega-3 fatty acids during pregnancy and to infants older than 6 months has been shown to reduce the severity of atopic disease (Upham & Holt, 2005).

Gut microflora may play a role in immunomodulation in infants, reducing the risk of early atopic disease. Probiotics have successfully been used to reduce atopic eczema in high-risk children by having mothers take *Lactobacillus* GG (GG refers to a healthy strain) 2 to 4 weeks before delivery and 6 months postnatally while breastfeeding (Kalliomaki et al., 2001). However, a Cochrane review found insufficient evidence to recommend probiotics for prevention of allergic disease or food hypersensitivity because study results were inconsistent (Osborn & Sinn, 2007). The hygiene hypothesis continues to be a plausible explanation for the increased prevalence of allergic disease in children. Reduced exposure to microbial stimuli in early life may result in insufficient stimulation of regulatory T cells and an inappropriate allergic response when exposed to food allergens.

There is no way to predict when a child will outgrow a food allergy, but 75% to 90% of milk-allergic children can tolerate cow's milk by 4 years of age. Some infants lose their milk allergy in as little as a few months, whereas others may remain symptomatic for as long as 8 to 10 years (Wood, 2003). Many also become tolerant to egg, soy, and wheat, although fish, tree nut, and peanut allergies may persist throughout the lifetime (Nowak-Wegrzyn, 2003). Other foods that may cause allergic reactions in infants and young children include berries, tomatoes, citrus, and apples. Children with non-IgE-mediated cow's milk allergy often outgrow their allergies by 5 years of age without the development of additional allergic complications. Children with IgE-mediated allergies often have persistent allergic symptoms at 8 years of age. They also more frequently have asthma, rhinoconjunctivitis, atopic eczema, and sensitization to other allergens and are at increased risk for sensitization to inhalant allergens (Saarinen, Pelkonen, Makela, & Savilahti, 2005).

### Diabetes and Celiac Disease

The type of feedings in infancy may influence the development of type 1 diabetes. A systematic review of the literature showed that a short duration and/or lack of breastfeeding along with early introduction of cow's milk and formula increases the risk of developing type 1 diabetes (Patelarou et al., 2012). A case-control study of siblings with and without type 1 diabetes showed that the siblings with type 1 diabetes had a shorter duration of breastfeeding (3.3 vs. 4.6 months) and were introduced earlier to cow's milk (Alves, Figueiroa, Meneses, & Alves, 2012).

Two studies by Norris and coworkers (Norris, Barriga, Hoffenber et al., 2005; Norris, Barriga, Klingensmith et al., 2005) looked at the association between the development of type 1 diabetes and celiac disease and early introduction of gluten-containing foods. The Diabetes Autoimmunity Study in the Young is a prospective study of triggers for diabetes and celiac disease in genetically predisposed children with a parent or sibling with type 1 diabetes or celiac disease. The timing of introduction of gluten-containing cereals was found to be associated with the risk of developing diabetes or celiac disease in children at increased risk for the disease. Children initially exposed to wheat, barley, or rye between birth and 3 months or later than 7 months were at increased risk of developing diabetes and celiac disease than were children first exposed to cereal between 4 and 6 months.

In Sweden, where the prevalence of celiac disease is 1–2% of Swedish children, introducing gluten-containing foods at 4 to 6 months is recommended during the time of exclusive breastfeeding. Enacted in 1996, this policy was a change from previous recommendations to introduce gluten after 6 months. New evidence showed that the risk of childhood celiac disease could be reduced with concurrent breastfeeding during the time that gluten is introduced into the infant's diet.

Recommendations to avoid introduction of gluten before 4 months and after 7 months and that gluten be introduced into the diet while the infant is being breastfed are based on multiple studies on the development of celiac disease (Henriksson, Boström, & Wiklund, 2012; Shamir, 2012; Szajewska et al., 2012). A study in Belgrade of infants with celiac disease found that longer breastfeeding and breastfeeding at the time of gluten introduction significantly delayed the onset of celiac disease (Radlovic, Mladenovic, Lekovic, Stojsic, & Radlovic, 2010). The study did not find that early introduction before 4 months resulted in earlier onset of the disease compared to those introduced at 4 to 6 months, but that introduction after 6 months significantly delayed onset of the disease from 12–15 months to 22 months. Because the group of infants receiving gluten after 6 months also had longer duration of breastfeeding, it is unclear whether the timing of gluten introduction was solely responsible for the delayed onset of the disease.

The mechanisms for breastfeeding inducing tolerance to gluten include the presence of gliaden in human milk; the reduction in acute gastroenteritis in breastfed infants; differences in gut flora; and reduced intestinal permeability in breastfed infants. Despite public health programs to inform families of the recommendations, a survey in 2004 showed that only 45% were compliant with the recommendation to introduce gluten earlier than 6 months while breastfeeding. As many as 45% continued to avoid gluten until after 6 months and another 10% introduced gluten without breastfeeding (van Odijk, Hulthen, Ahlstedt, & Borres, 2004).

## Case Study 1

### Vitamin D Deficiency in Early Infancy

Emily Burritt, MS, RD, CNSC

David, an 8-week-old Hispanic male, was admitted to the pediatric intensive care unit with seizures and respiratory distress. His birth weight was 2.98 kg (6 lbs 9 oz), and his admission weight was 4.6 kg. It appeared his weight was tracking the 5th–10th percentile for age using the World Health Organization growth chart. Mom did not remember what David's length was at birth, and there was not a length measured on admission. David was breastfed for 20–30 minutes (10–15 minutes each breast) every 2–3 hours prior to admission. He was not taking any other vitamin supplements or medications. Mom reported she had a good appetite and food intake during pregnancy. She took the standard prenatal vitamin (400 IU vitamin $D_3$) as prescribed during pregnancy but had stopped after the baby was born.

Initial laboratory results revealed a normal sodium level, a calcium level of 7.5 mg/dL (low), and a phosphorus level of 5.5 mg/dL (normal). Vitamin D deficiency was suspected and 25-hydroxyvitamin D level was obtained, which was consistent with deficiency at 13 ng/mL. The registered dietitian (RD) recommended starting vitamin $D_3$ 1,000–2,000 IU/day orally. However, liquid vitamin $D_2$ was started, 8,000 IU/day orally.

A repeat 25-hydroxyvitamin D level 2 weeks later was 158 ng/mL (toxicity), and the vitamin $D_2$ supplement was held. Another 2 weeks passed, and the repeat 25-hydroxyvitamin D level had normalized. A maintenance dose of vitamin $D_3$ 400 IU/day orally was prescribed.

The RD assessed the following factors:

1. Infant's growth pattern
2. Infant's diet history
3. Maternal diet history
4. Vitamin D levels and dosing of supplement

#### Questions

1. What is a major cause of vitamin D deficiency? What foods are good sources of vitamin D?
2. Was David's intake of vitamin D adequate prior to supplementation?
3. What other factors placed David at risk for vitamin D deficiency?
4. What is the difference between vitamin $D_2$ (ergocalciferol) and vitamin $D_3$ (cholecalciferol)?
5. What caused David's vitamin D level to become toxic?

## Case Study 2

### Normal Infant Nutrition

Rachelle Lessen, MS, RD, IBCLC

Caleb was born by standard vaginal delivery to a healthy 30-year-old mother. Caleb's mother decided while she was pregnant to exclusively breastfeed him for the first 6 months of his life because she was familiar with the advantages associated with exclusive breastfeeding, such as reduction of illness and allergies, enhanced intelligence, convenience of feedings, and cost savings. Caleb weighed 7 pounds at birth (25% percentile) and gained weight appropriately for the first 6 months of life. At his 6-month checkup he weighed 18 pounds and was at the 50% to 75% percentile for weight.

Caleb's mother chose to introduce puréed foods she prepared herself when Caleb was 6 months old. At this time, he exhibited an interest in what his parents were eating and had developed good head and neck control. She prepared sweet potatoes, carrots, squash, and peas and puréed them in a food processor until they were a smooth consistency. She froze them in single serving portions in an ice cube tray. She introduced one food at a time and waited 3 to 5 days between foods while observing Caleb for signs of food allergy. Gradually, she added more foods to his diet, including chicken, turkey, beef, cereal, pears, peaches, and bananas. When Caleb was 7 months old he was offered a sippy cup with water at meals.

Caleb was developing fine motor control and was able to grasp foods and attempt self-feeding. His mother offered him small pieces of toast, Cheerios, cut-up fresh melon, soft-cooked carrots, French toast, and pieces of cheese and turkey. He also began to eat cottage cheese and yogurt and a greater variety of foods from the family meal. Caleb continued to breastfeed, but the number of feedings per day began to decrease as he increased his intake of complementary foods. Caleb was breastfed without any supplemental formula until he was 13 months old, when he was offered whole cow's milk by cup. By 1 year of age he had gradually transitioned from an all-milk diet in the first 6 months to a mixed diet of breast milk and puréed foods and finally to a diet of family foods including a variety of fruits and vegetables, grains, meats, and dairy.

#### Question

1. What did Caleb's mother do correctly?

## Case Study 3

### Infant Nutrition

Rachelle Lessen, MS, RD, IBCLC

Emory is a 6-week-old former full-term infant. Her mother is 30 years old, healthy, does not smoke, and this is her first baby. Emory has been exclusively breastfeeding since birth. She latched well from the beginning, and mom denies any difficulties or problems with sore nipples. Her output includes 10–11 wet diapers and 2 stools per day. Her mother reports that she breastfeeds more than 12 times per day and that her feedings are very long, typically more than 1 hour. Mom's goal is to breastfeed her for more than 1 year.

Emory's birth weight was 3.487 kg (75th percentile on the WHO growth chart). Her discharge weight from the hospital was 2.98 kg (10% below birth weight). At 1 week, she weighed 3.345 kg, and at 2 weeks she weighed 3.289 kg. Now at 6 weeks she weighs 3.52 kg (<2nd percentile on the WHO growth chart). She has gained 33 g since birth. Her head circumference and length are within normal.

#### Questions

1. What is the primary nutrition concern?
2. What is the most likely cause of the problem?
3. How would you improve her nutritional status while taking into consideration the mother's breastfeeding goals?

## Case Study 4

### Failure to Thrive

Ancy Thomas, MS, RD, CSP, LDN

RS is 8 months and 3 weeks old. She is a full-term female with a history of gastroesophageal reflux, now presenting with suboptimal weight gain and poor feeding for 2–3 months. RS has been falling off her growth curve for weight, but length and head circumference growth velocities have been maintained. She had age-appropriate intake of a standard formula (Good Start Gentle) until 2–3 months ago. The patient's mother switched formula to a soy-based formula (Good Start Soy) because RS had emesis after each feeding; however, the emesis was ultimately attributed to a viral gastroenteritis rather than formula intolerance. At a visit for weight check, her primary pediatrician sent RS to the emergency department for concern about failure to thrive (FTT).

Anthropometrics/Growth Plotted on the WHO Growth Chart:

- Admission weight: 6.14 kg (< 2nd percentile)
- Length: 68 cm (25thpercentile). Standard length/age: 70 cm
- Ideal body weight (IBW): 7.8 kg
- Head circumference: 41.7 cm (5–10th percentile)
- Weight-for-length: Less than 2nd percentile
- Weight history: At 7 months was 6.2 kg (2–5th percentile)
- Diet history: Mom reports mixing 4 scoops of Gerber Good Start Soy powder in 4 oz of water with 2 tablespoons of rice cereal. RS drinks 1–2 oz six times daily.
- Pertinent medications: Cefuroxime, multivitamin 1 mL daily
- Significant labs: Unremarkable. Albumin normal
- Estimated nutritional needs: ≥100 kcal/kg/day; protein: 1.2 g/kg/day; fluids: 100 mL/kg/day

This patient's growth parameters reveal that she is at 79% of her IBW, indicative of moderate wasting per Waterlow Criteria and suggestive of acute malnutrition. She is 97% of the standard height-for-age, which is normal. RS has lost 60 g total from 7 months of age to present, as compared to the goal of gaining 10–13 g per day.

Based on the history, RS is admitted with nonorganic FTT because no other disease or disorder is identified as the cause of her FTT. Diet history reveals that her mother was incorrectly mixing the formula and adding excessive amounts of rice cereal, making it a 40 kcal/oz formula (standard concentration of infant formula is 20 kcal/oz). The patient's mother reported that rice cereal was added for reflux, as suggested by the patient's primary provider. However, no clear guidance was given on how much rice cereal was to be added. This resulted in a thick mixture of inappropriate caloric concentration. RS was taking in only 12 ounces of this mixed formula daily, which provided 480 calories (78 kcal/kg/day), 12 g of protein per day (2 g/kg/day), and 59 mL/kg/day of fluid (59% of her maintenance daily fluid requirement).

After performing an initial nutrition assessment, the dietitian suggested Gerber Good Start Soy at 24 kcal/oz with 1 tsp rice cereal per 1 oz of formula with a goal of minimum intake of 770 mL/day, which provided the following: 125 mL/kg/day, 693 calories per day (113 kcal/kg/day), and 17 g of protein per day (2.8 g/kg/day). The RDA for a 7- to 12-month-old child is 98 kcal/kg/day and 1.2 g of protein/kg/day. The nutritional plan was to make adjustments, as needed, to the nutrition prescription based on change in growth parameters.

#### Questions

1. Other than the information provided in this case study, what other questions would you ask Mom?
2. What other services do you believe should be consulted while the patient is admitted in the hospital?
3. What instructions would you give to the mom or the caregiver?

## Case Study 5

### Failure to Thrive and Breastfeeding

Phuong Huynh, MS, RD, CSP

A 14-day-old term male infant presented to the pediatrician's office and was found to have significant weight loss. His weight was down by 7% and 15% during his first visit at day of life (DOL) 5 and during the second visit at DOL 14, respectively. He appeared lethargic, leading to further work-up that showed elevated serum sodium and dehydration. He was sent to the emergency department to receive intravenous fluid for rehydration and to be admitted for failure to thrive.

Birth history was uneventful; birth weight was 3,500 g. He was discharged home at 48 hours of life. He is the first child to his parents. He is exclusively breastfed. He feeds on demand about every 3–4 hours during the day and every 5–6 hours at night. He stays on the breast 5–10 minutes on each side. His mother felt milk came in on DOL 3, but breasts do not always feel full. Mom feels breastfeeding is going well, but she feels baby seems very sleepy. He stools 2–4 times daily and has 3–5 wet diapers, but they are not always heavy. Stool is seedy, yellowish green.

#### Questions

1. Is maternal milk supply adequate?
2. Is milk transfer effective?
3. Should breastfeeding be supplemented?

## Issues to Debate

1. Discuss obstacles to breastfeeding that women encounter and possible public health strategies to overcome these challenges.
2. What are the effects of early feeding on the development of obesity and what can be done to reduce the increasing rates of childhood obesity?
3. Infant formula manufacturers add DHA and AA to their products. This has greatly raised the cost to consumers (including the U.S. government, which is the largest purchaser of formula because of the WIC program), yet studies fail to show long-term benefit of these additions. Discuss the ethical implications of this practice.
4. What are some of the cultural aspects that affect the transitioning from an all-milk infant diet to a diet of family foods?

## References


Academy of Breastfeeding Medicine. (2011). Protocol #24: Allergic proctocolitis in the exclusively breastfed infant. *Breastfeeding Medicine*, *6*, 435–440.

Academy of Breastfeeding Medicine Protocol Committee. (2009). ABM clinical protocol #3: Hospital guidelines for the use of supplementary feedings in the healthy term breastfed neonate, revised 2009. *Breastfeeding Medicine*, *4*(3), 176–181. Retrieved from www.bfmed.org/Media/Files/Protocols/Protocol%203%20English%20Supplementation.pdf

Alipui, N. (2012). Making room for breastfeeding on the worldwide agenda. *Breastfeeding Medicine*, *7*, 329–331.

Altucher, K., Rasmussen, K., Barden, E., & Habicht, J. (2005). Predictors of improvement in hemoglobin concentration among toddlers enrolled in the Massachusetts WIC program. *Journal of the American Dietetic Association, 105*, 709–715.

Alves, J. G., Figueiroa, J. N., Meneses, J., & Alves, G. V. (2012). Breastfeeding protects against type 1 diabetes mellitus: A case-sibling study. *Breastfeeding Medicine*, *7*(1), 25–28.

American Academy of Pediatrics. (2004). *Pediatric nutrition handbook* (5th ed.). Elk Grove Village, IL: Author.

American Academy of Pediatrics Committee on Nutrition. (2000). Hypoallergenic infant formulas. *Pediatrics, 106*, 346–349.

American Academy of Pediatrics Committee on Nutrition. (2003). Prevention of pediatric overweight and obesity. *Pediatrics, 112*, 424–430.

American Academy of Pediatrics Section on Breastfeeding. (2005). Breastfeeding and the use of human milk. *Pediatrics, 115*, 496–506.

American Academy of Pediatrics Section on Breastfeeding. (2012). Breastfeeding and the use of human milk. *Pediatrics, 129*, e827–e841.

American Dietetic Association. (2003). Position of the American Dietetic Association and Dietitians of Canada: Vegetarian diets. *Journal of the American Dietetic Association, 103*, 748–765.

Auestad, N., Halter, R., Hall, R., Blatter, M., Bogle, M., Burks, W., … Bornstein, M. H. (2001). Growth and development in term infants fed long-chain polyunsaturated fatty acids: A double-masked, randomized, parallel, prospective, multivariate study. *Pediatrics, 108*, 372–381.

Baker, R. D. (2002). Infant formula safety. *Pediatrics, 110*, 833–835.

Bammann, M. (2012). Feeding in the first year of life: Emerging benefits of introducing complementary solids from 4 months. *Australian Family Physician*, *41*, 226–229.

Bartick, M., & Reinhold, A. (2010). The burden of suboptimal breastfeeding in the United States: A pediatric cost analysis. *Pediatrics*, *125*(5), e1048–1056.

Basile, L. A., Taylor, S. N., Wagner, C. L., Horst, R. L., & Hollis, B. (2006). The effect of high-dose vitamin D

supplementation on serum vitamin D levels and milk calcium concentration in lactation women and their infants. *Breastfeeding Medicine, 1*(1), 27–35.

Basile, L. A., Taylor, S. N., Wagner, C. L., Quinones, L., & Hollis, B. W. (2007). Neonatal vitamin D status at birth at latitude 32 degrees 72': Evidence of deficiency. *Journal of Perinatology*, *27*(9), 568–571.

Bergmann, K. E., Bergmann, R. L., von Kries, R., Böhm, O., Richter, R., Dudenhausen, J. W., & Wahn, U. (2003). Early determinants of childhood overweight and adiposity in a birth cohort study: Role of breast-feeding. *International Journal of Obesity*, *27*,162–172.

Birch, L., Orlet Fisher, J., & Krahnstoever Davison, K. (2003). Learning to overeat: Maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *American Journal of Clinical Nutrition*, *78*, 215–220.

Bogen, D. L., Hanusa, B. H., & Whitaker, R. C. (2004). The effect of breast-feeding with and without formula use on the risk of obesity at 4 years of age. *Obesity Research*, *12*,1527–1535.

Briefel, R. (2010). New findings from the feeding infants and toddlers study: Data to inform action. *Journal of the American Dental Association*, *110*, S5–7.

Briefel, R., Reidy, K., Karwe, V., & Devaney, B. (2004). Feeding Infants and Toddlers Study: Improvements needed in meeting infant feeding recommendations. *Journal of the American Dietetic Association*, *104*, S31–S37.

Briefel, R., Reidy, K., Karwe, V., Jankowski, L., & Hendricks, K. (2004). Toddlers' transition to table foods: Impact on nutrient intakes and food patterns. *Journal of the American Dietetic Association, 104*, S38–S44.

Bronner, Y., Barber, T., & Miele, L. (2001). Breastfeeding peer counseling: Rationale for the National WIC survey. *Journal of Human Lactation*, *17*, 135–141.

Bronner, Y., Barber, T., Vogelhut, J., & Kovar Resnick, A. (2001). Breastfeeding peer counseling: Results from the National WIC Survey. Journal of Human Lactation, *17*, 119–125.

Brown, K. H., Engle-Stone, R., Krebs, N., & Peerson, J. M. (2009). Recent advances in knowledge of zinc nutrition and human health. *Food and Nutrition Bulletin*, 30(1 Suppl.), S144–169.

Burks, A. W., Jones, S. M., Boyce, J. A., Sicherer, S. H., Wood, R. A., Assa'ad, A., & Sampson, H. A. (2011). NIAID-sponsored 2010 guidelines for managing food allergy: Applications in the pediatric population. *Pediatrics*, *128*(5), 955–965. doi:10.1542/peds.2011-0539

Butte, N. F. (2005). Energy requirements of infants. *Public Health Nutrition*,*8*(7A), 953–967.

Butte, N., Cobb, K., Dwyer, J., Graney, L., Heird, W., & Rickard, K. (2004). The Start Healthy Feeding Guidelines for infants and toddlers. *Journal of the American Dietetic Association*, *104*, 442–454.

Butte, N., Fox, M. K., Briefel, R. R., Siega-Riz, A. M., Dwyers, J. T., Deming, D. M., & Reidy, K. C. (2010). Nutrient intakes of US infants, toddlers, and preschoolers meet or exceed dietary reference intakes. *Journal of the American Dental Association*, *110*, S27–37.

Butte, N., Wong, W., Hopkinson, J., Heinz, C., Mehta, N., & Smith, E. O. (2000). Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. *American Journal of Clinical Nutrition*, *72*, 1558–1569.

Butte, N., Wong, W., Hopkinson, J., Smith, E. O., & Ellis, K. (2000). Infant feeding mode affects early growth and body composition. *Pediatrics, 106*, 1355–1366.

Canadian Paediatric Society. (2003). Dietary manipulations for infantile colic. *Paediatrics & Child Health, 8*, 449–452.

Carruth, B. R., Skinner, J., Houck, K., & Moran, J. D. (2000). Addition of supplementary foods and infant growth. *Journal of the American College of Nutrition, 19*, 405–412.

Carruth, B. R., Ziegler, P., Gordon, A., & Hendricks, K. (2004). Developmental milestones and self-feeding behaviors in infants and toddlers. *Journal of the American Dietetic Association, 104*, S51–S56.

Centers for Disease Control and Prevention. (2012). Breastfeeding Report Card 2012, United States: Outcome Indicators. Retrieved from www.cdc.gov/breastfeeding/data/reportcard2.htm

Centers for Disease Control and Prevention. (2002). *Enterobacter sakazakii* infections associated with the use of powdered infant formula—Tennessee, 2001. *Morbidity and Mortality Weekly Report*, *51*, 297–300.

Chen, A., & Rogan, W. J. (2004). Breastfeeding and the risk of postneonatal death in the United States. *Pediatrics*, *113*(5), e435–e439.

Clifford, T., Campbell, K., Speechley, K., & Gorodzinsky, F. (2002). Infant colic: Empirical evidence of the absence of an association with source of early infant nutrition. *Archives of Pediatric and Adolescent Medicine, 156*, 1123–1128.

Dattilo, A. M., Birch, L., Krebs, N., Lake, A., Taveras, E. M., & Saavedra, J. M. (2012). Need for early intervention in the prevention of pediatric overweight: A review and upcoming directions. *Journal of Obesity*, *2012*. doi:10.1155/2012/123023

Dawodu, A., Adarwal, M., Hossain, M., Kochiyil, J., & Zayed, R. (2003). Hypovitaminosis D and vitamin D deficiency in exclusively breastfeeding infants and their mothers in summer: A justification for vitamin D supplementation of breast-feeding infants. *Journal of Pediatrics, 142*, 169–173.

de Onis, G., Garza, C., Onyango, A. W., & Borghi, E. (2007). Comparison of the WHO Child Growth Standards and the CDC 2000 Growth Charts. *Journal of Nutrition, 137*,144–148.

Devaney, B., Kalb, L., Briefel, R., Zavitsky-Novak, T., Clusen, N., & Ziegler, P. (2004a). Feeding Infants and Toddlers Study: Overview of the study design. *Journal of the American Dietetic Association, 104*, S8–S13.

Devaney, B., Ziegler, P., Pac, S., Karwe, V., & Barr, S. (2004b). Nutrient intakes of infants and toddlers. *Journal of the American Dietetic Association, 104*, S14–S21.

Dewey, K. G. (2000a). Complementary feeding and breastfeeding. *Pediatrics,* 106, 1301–1302.

Dewey, K. G. (2000b). Protein and amino acids. *Pediatrics, 106*, 1292.

Dewey, K. G. (2001). Nutrition, growth, and complementary feeding of the breastfed infant. *Pediatric Clinics of North America, 48*, 87–104.

Dewey, K. G., Beaton, G., Fjeld, C., Lonnerdal, B., & Reeds, P. (1996). Protein requirements of infants and children. *European Journal of Clinical Nutrition, 50*, S119–S150.

Dewey, K. G., Cohen, R. J., Rivera, L. L., Canahuati, J., & Brown, K. H. (1996). Do exclusively breast-fed infants require extra protein? *Pediatric Research, 39*, 303–307.

Dewey, K. G., Domellof, M., Cohen, R., Rivera, L. L., Hernell, O., & Lonnerdal, B. (2002). Iron supplementation affects growth and morbidity of breast-fed infants: Results of a randomized trial in Sweden and Honduras. *Journal of Nutrition, 132*, 3249–3255.

Dewey, K. G., Peerson, J. M., & Brown, K. H. (1998). Effects of age of introduction of complementary foods on iron status of breastfed infants in Honduras. *American Journal of Clinical Nutrition, 67*, 878–884.

Dietz, W. H. (2001). Breastfeeding may help prevent childhood overweight. *Journal of the American Medical Association, 285*, 2506–2507.

Domellof, M., Lonnerdal, B., Abrams, S., & Hernall, O. (2002). Iron absorption in breast-fed infants: Effects of age, iron status, iron supplements, and complementary foods. *American Journal of Clinical Nutrition, 76*, 198–204.

Druet, C., Stettler, N., Sharp, S., Simmons, R. K., Cooper, C., Smith, G. D., … Ong, K. K. (2012). Prediction of childhood obesity by infancy weight gain: an individual-level meta-analysis. *Paediatric Perinatal Epidemiology,* 26, 1, 19–26.

DuPont, C. (2003). Protein requirements during the first year of life. *American Journal of Clinical Nutrition, 77*(Suppl.), 1544S–1549S.

DuToit, G., Katz, Y., Sasieni, P., Mesher, D., Maleki, S. J., Fisher, H. R., … Lack, G. (2008). Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. *Journal of Allergy and Clinical Immunology*, *122*, 984–991.

Dwyer, J., Suitor, C., & Hendricks, K. (2004). FITS: New insights and lessons learned. *Journal of the American Dietetic Association, 104*, S5–S7.

Eichenberger Gilmore, J., Hong, L., Broffitt, B., & Levy, S. (2005). Longitudinal patterns of vitamin and mineral supplement use in young white children. *Journal of the American Dietetic Association, 105*, 763–772.

Engelmann, M. D. M., Sandstrom, B., & Michaelsen, K. (1996). Meat intake and iron status in late infancy: An intervention study. *Journal of Pediatric Gastroenterology and Nutrition, 26*, 26–33.

Ermis, B., Demirel, F., Demircan, N., & Gurel, A. (2002). Effects of three different iron supplementations in term healthy infants after 5 months of life. *Journal of Tropical Pediatrics, 48*, 280–284.

European Food Safety Authority. (2004, November 18). EFSA Panel advises on how to avoid microbiological risks in infant formulae—at home and in hospital. Retrieved from www.efsa.europa.eu/en/press/news/biohaz041118.htm

FAO/WHO. (1989). Protein Quality Evaluation: Report of the Joint FAO/WHO Expert Consultation. Food and Nutrition Paper 51.

Fields, D. A., & Demerath, E. W. (2012). Relationship of insulin, glucose, leptin, IL-6 and TNF-α in human breast milk with infant growth and body composition. *Pediatric Obesity*, *7*(4), 304–312. doi:10.1111/j.2047-6310.2012.00059.x

Fomon, S. (2001). Feeding normal infants: Rationale for recommendations. *Journal of the American Dietetic Association, 101*, 1002–1005.

Fox, M. K., Devaney, B., Reidy, K., Razafindrakoto, C., & Ziegler, P. (2006). Relationship between portion size and energy intake among infants and toddlers: Evidence of self-regulation. *Journal of the American Dietetic Association, 106*, S77–S83.

Fox, M. K., Pac, S., Devaney, B., & Jankowski, L. (2004). Feeding Infants and Toddlers Study: What foods are infants and toddlers eating? *Journal of the American Dietetic Association, 104*, S22–S30.

Fox, M. K., Reidy, K., Karwe, V., & Ziegler, P. (2006). Average portions of foods commonly eaten by infants and toddlers in the United States. *Journal of the American Dietetic Association, 106*, S66–S76.

Fox, M. K., Reidy, K., Novak, T., & Ziegler, P. (2006). Sources of energy and nutrients in the diets of infants and toddlers. *Journal of the American Dietetic Association, 106,* S28–S42.

Gale, C., Logan, K. M., Santhakumaran, S., Parkinson, J. R., Hyde, M. J., & Modi, N. (2012). Effect of breastfeeding compared with formula feeding on infant body composition: A systematic review and meta-analysis. *American Journal of Clinical Nutrition*, *95*(3), 656–669.

Gartner, L., Greer, F., and the Section on Breastfeeding and Committee on Nutrition. (2003). Prevention of rickets and vitamin D deficiency: New guidelines for vitamin D intake. *Pediatrics, 111*, 908–910.

Gibson, R., & Holtz, C. (2000). The adequacy of micronutrients in complementary foods. *Pediatrics, 106*, 1298–1300.

Gillman, M. W., Rifas-Shiman, S. L., Camargo, C. A., Berkey, C. S., Frazier, A. L., Rockett, H. R. H., … Colditz, G. A. (2001). Risk of overweight among adolescents who were breastfed as infants. *Journal of the American Medical Association, 285*, 2461–2467.

Greer, F., Shannon, M., the Committee of Nutrition, & the Committee on Environmental Health. (2005). Infant methemoglobinemia: The role of dietary nitrate in food and water. *Pediatrics, 116*, 784–786.

Greer, F. R., Sicherer, S. H., Burks, A. W., American Academy of Pediatrics Committee on Nutrition, & American Academy of Pediatrics Section on Allergy and Immunology. (2008). Effects of early nutritional interventions on the development of atopic disease in infants and children: The role of maternal dietary restriction, breastfeeding, timing of introduction of complementary foods, and hydrolyzed formulas. *Pediatrics*, *121*(1), 183–191.

Griffiths, L. J., Smeeth, L., Sherburne-Hawkins, S., Cole, T. J., & Dezateux, C. (2009). Effects of infant feeding practice on weight gain from birth to 3 years. *Archives of Disease in Childhood, 94*, 577–582.

Grummer-Strawn, L., Reinold, C., & Krebs, N. F. (2010). Use of World Health Organization and CDC Growth Charts for Children Aged 0–59 Months in the United States. Retrieved from www.cdc.gov/mmwr/preview/mmwrhtml/rr5909a1.htm

Gupta, R. S., Springston, E. E., Warrier, M. R., Smith, B., Kumar, R., Pongracic, J., & Holl, J. L. (2011). The prevalence, severity, and distribution of childhood food allergy in the United States. *Pediatrics*, *128*, e9–e17.

Hadders-Algra, M. (2010). Effect of long-chain polyunsaturated fatty acid supplementation on neurodevelopmental outcome in full-term infants. *Nutrients, 2*, 790–804.

Hanson, L.A. (2004). *Immunobiology of human milk: How breastfeeding protects babies*. Amarillo, TX: Pharmasoft.

Harder, T., Bergmann, G., Kallischnigg, G., & Plagemann, A. (2005). Duration of breastfeeding and risk of overweight: A meta-analysis. *Journal of Epidemiology*, *162*, 397–403.

Heath, A. M., Tuttle, C. R., Simons, M. S. L., Cleghorn, C., & Parnell, W. (2002). Longitudinal study of diet and iron deficiency anaemia in infants during the first two years of life. *Asia Pacific Journal of Clinical Nutrition, 11*, 251–257.

Hediger, M., Overpeck, M., Kuczmarski, R., & Ruan, W. J. (2001). Association between infant breastfeeding and overweight in young children. *Journal of the American Medical Association, 285*, 2453–2460.

Heine, R., Hill, D., & Hosking, C. (2004). Primary prevention of atopic dermatitis in breastfed infants: What is the evidence? *Journal of Pediatrics, 44*, 564–567.

Henderson, A. (2005). Vitamin D and the breastfed infant. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 34*, 367–372.

Henriksson, C., Boström, A. M., & Wiklund, I. E. (2012). What effect does breastfeeding have on coeliac disease? A systematic review update. *Evidence Based Medicine*. Advance online publication. Retrieved from www.ncbi.nlm.nih.gov/pubmed/22864373

Hill, D., Roy, N., Heine, R., Hosking, C., Francis, D., Brown, J., … Carlin, J. B. (2005). Effect of a low-allergen maternal diet on colic among breastfed infants: A randomized, controlled trial. *Pediatrics, 116*, e709–e715.

Huh, S. Y., Rifas-Shiman, S. L., Taveras, E. M., Oken, E., & Gillman, M. W. (2011). Timing of solid food introduction and risk of obesity in preschool-aged children. *Pediatrics*, *127*(3), e544–551.

Human Milk Banking Association of North America. (n.d.). HMBANA position paper on donor milk banking. Retrieved from www.hmbana.org/downloads/position-paper-donor-milk.pdf

Hutton, E. K., & Hassan, E. S. (2007). Late vs. early clamping of the umbilical cord in full term neonates: Systemic review and meta-analysis. *Journal of the American Medical Association, 297*, 1241–1252.

Institute of Medicine. (2001). *Dietary Reference Intake for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Washington, DC: National Academy Press.

Institute of Medicine. (2002). *Dietary Reference Intake for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Part I*. Prepublication copy. Washington, DC: National Academy Press.

Institute of Medicine. (2005). Report brief. WIC food packages: Time for a change. Retrieved from www.iom.edu/Reports/2005/WIC-Food-Packages-Time-for-a-Change.aspx

Ip, S., Chung, M., Raman, G., Chew, P., Magula, N., DeVine, D., … Lau, J. (2007, April). *Breastfeeding and maternal and infant health outcomes in developed countries*. Evidence report/technology assessment No. 153 (Prepared by Tufts-New England Medical Center Evidence-Based Practice Center, under Contract No. 290-02-0022). AHRQ Publication No. 07-E007. Rockville, MD: Agency for Healthcare Research and Quality.

Jensen, C., Voigt, R., Prager, T., Zou, Y., Fraley, J. K., Rozelle, J., … Heird, W. C. (2005). Effects of maternal docosahexaenoic acid intake on visual function and neurodevelopment in breastfed term infants. *American Journal of Clinical Nutrition, 8*,125–132.

Jensen, E., & Labbok, M. (2011). Unintended consequences of the WIC formula rebate program on infant feeding outcomes: Will the new food packages be enough? *Breastfeeding Medicine*, *6*, 145–149.

Johnson, W., Wright, J., & Cameron, N. (2012). The risk of obesity by assessing infant growth against the UK-WHO charts compared to the UK90 reference: findings from the Born in Bradford birth cohort study. *BMC Pediatrics, 12*, 104.

Joppu, U., Kalliomaki, M., & Isolauri, E. (2000). Maternal diet rich in saturated fat during breastfeeding is associated with atopic sensitization of the infant. *European Journal of Clinical Nutrition, 54*, 702–705.

Kalliomaki, M., Salminen, S., Arvilommi, H., Kero, P., Koskinene, P., & Isolauri, E. (2001). Probiotics in primary prevention of atopic disease; a randomised placebo-controlled trial. *Lancet, 357*, 1076–1079.

Kattelmann, K. K., Ho, M., & Specker, B. L. (2001). Effect of timing of introduction of complementary foods on iron and zinc status of formula fed infants at 12, 24, and 36 months of age. *Journal of the American Dietetic Association, 101*, 443–447.

Kazal, L. (2002). Prevention of iron deficiency in infants and toddlers. *American Family Physician, 66*, 1217–1224.

Khedr, E. M., Farghaly, W. M., Amry Sel, D., & Osman, A. A. (2004). Neural maturation of breastfed and formula-fed infants. *Acta Paediatrics*, *93*, 734–738.

Kill, I., Wickman, M., Lilja, G., Nordvall, S. L., & Pershagen, G. (2002). Breast feeding and allergic diseases in infants—a prospective birth cohort study. *Archives of Diseases in Children, 87*, 478–481.

Kleinman, R. (2000a). American Academy of Pediatrics recommendations for complementary feeding. *Pediatrics, 106*, 1274.

Kleinman, R. (2000b). Complementary feeding and later health. *Pediatrics, 106*, 1287–1288.

Kleinman, R. (2000c). Complementary feeding and neuromuscular development. *Pediatrics, 106*, 1279.

Kneepkens, C. M. K, & Brand, P. L. P. (2010). Breastfeeding and the prevention of allergies. *European Journal of Pediatrics*. doi:10.1007/s00431-010-1141-7

Koletzko, B. (2000). Complementary foods and the development of food allergy. *Pediatrics, 106*, 1285.

Koo, W. (2003). Efficacy and safety of docosahexaenoic acid and arachidonic acid addition to infant formulas: Can one buy better vision and intelligence? *Journal of the American College of Nutrition, 22*, 101–107.

Koplin, J. J., Osborne, N. J., Wake, M., Martin, P. E., Gurrin, L. C., Robinson, M. N., … Allen, K. J. (2010). Can early introduction of egg prevent egg allergy in infants? A population-based study. *Journal of Allergy and Clinical Immunology*, *126*(4), 807–813.

Kramer, M., Guo, T., Platt, R., Shapiro, S., Collet, J. P., Chlamers, B., … PROBIT Study Group. (2002). Breastfeeding and infant growth: Biology or bias? *Pediatrics, 110*, 343–347.

Kramer, M. S., & Kakuma, R. (2012). Optimal duration of exclusive breastfeeding. *Cochrane Database of Systematic Reviews*, (8), CD003517.

Krebs, N. (2000). Dietary zinc and iron sources, physical growth and cognitive development of breastfed infants. *Journal of Nutrition, 130*, 358S–360S.

Krebs, N. F., Reidinger, C. J., Robertson, A. D., & Hambridge, K. M. (1994). Growth and intakes of energy and zinc in infants fed human milk. *Journal of Pediatrics, 124*, 32–39.

Krebs, N. F., Westcott, J. E., Butler, N., Robinson, C., Bell, M., & Hambidge, K. M. (2006). Meat as a first complementary food for breastfed infants: Feasibility and impact on zinc intake and status. *Journal of Pediatric Gastroenterology and Nutrition*, *42*(2), 207–214.

Kreiter, S., Schwartz, R., Kirkman, H., Charlton, P., Calikoglu, A., & Davenport, M. (2000). Nutritional rickets in African-American breastfed infants. *Journal of Pediatric, 137*, 153–157.

Lack, G., Fox, D., Northstone, K., & Golding, J. (2003). Factors associated with the development of peanut allergy in childhood. *New England Journal of Medicine, 348*, 977–985.

Lawrence, R. (1999). *Breastfeeding: A guide for the medical profession* (5th ed.). St. Louis, MO: Mosby.

Li, R., Magadia, J., Fein, S. B., & Grummer-Strawn, L. M. (2012). Risk of bottle-feeding for rapid weight gain during the first year of life. *Archives of Pediatrics and Adolescent Medicine*, *166*, 431–436.

Lucas, A., & St. James-Robert, I. (1998). Crying, fussing and colic behavior in breast and bottle-fed infants. *Early Human Development, 53*, 9–18.

Lust, K., Brown, J., & Thomas, W. (1996). Maternal intake of cruciferous vegetables and other foods and colic symptoms in exclusively breastfed infants. *Journal of the American Dietetic Association, 96*, 47–48.

Mangels, R., & Driggers, J. (2012). The youngest vegetarians: Vegetarian infants and toddlers. *Infant, Child, and Adolescent Nutrition*, *4*, 8–20.

Martorell, R., Stein, A. D., & Schroeder, D. G. (2001). Early nutrition and later adiposity. *Journal of Nutrition, 131*, 874S–880S.

Meeks Gardner, J. M., Powell, C. A., Baker-Henningham, H., Walker, S. P., Cole, T. J., & Grantham-McGregor, S. M. (2005). Zinc supplementation and psychosocial stimulation: Effects on the development of undernourished Jamaican children. *American Journal of Clinical Nutrition, 82*, 399–405.

Meier, P. (2001). Breastfeeding in the special care nursery. *Pediatric Clinics of North America*, *48*, 425–442.

Michaelsen, K. F. (2000). Cow's milk in complementary feeding. *Pediatrics, 106*, 1302–1303.

Milner, J., Stein, D., McCarter, R., & Moon, R. (2004). Early infant multivitamin supplementation is associated with increased risk for food allergy and asthma. *Pediatrics, 114*, 27–32.

Morale, S., Hoffman, D., Castañeda, Y., Wheaton, D., Burns, R., & Birch, E. (2005). Duration of long-chain polyunsaturated fatty acids availability in the diet and visual acuity. *Early Human Development, 81*, 197–203.

Motarjemi, Y. (2000). Research priorities on safety of complementary feeding. *Pediatrics, 106*, 1304–1305.

Muraro, A., Dreborg, S., Halken, S., Høst, A., Niggemann, B., Aalberse, R., … Zeiger, R. S. (2004). Dietary prevention of allergic diseases in infants and small children. Part III. Critical review of published peer-reviewed observational and interventional studies and final recommendations. *Pediatric Allergy and Immunology, 15*, 291–307.

National Center for Health Statistics. (2000). Vital and health statistics of the Centers for Disease Control and Prevention. *Advance Data,* 314.

National WIC Association. (2004). *Breastfeeding promotion and support in the WIC Program*. Washington, DC: Author.

Nielsen, S. B. (2011). Adequacy of milk intake during exclusive breastfeeding: A longitudinal study. *Pediatrics, 128,* e907–914.

Norris, J., Barriga, K., Hoffenber, E., Taki, I., Miao, D., Haas, J., … Rewers, M. (2005). Risk of celiac disease autoimmunity and timing of gluten introduction in the diet of infants at increased risk of disease. *Journal of the American Medical Association, 293*, 2343–2351.

Norris, J., Barriga, K., Klingensmith, G., Hoffman, M., Eisenbarth, G., Erlich, H., & Rewers, M. (2005). Timing of initial cereal exposure in infancy and risk of islet autoimmunity. *Journal of the American Medical Association, 290*, 1713–1720.

Nowak-Wegrzyn, A. (2003). Future approaches to food allergy. *Pediatrics*, 111, 1672–1680.

Orlet Fisher, J., Birch, L., Smiciklas-Wright, H., & Picciano, M. F. (2000). Breast-feeding through the first year predicts maternal control in feeding and subsequent toddler energy intakes. *Journal of the American Dietetic Association, 100*, 641–646.

Osborn, D. A., & Sinn, J. K. (2007). Probiotics in infants for prevention of allergic disease and food hypersensitivity. *Cochrane Database of Systematic Reviews*, (4), CD006475.

Otten, J. J., Hellwig, J. P., & Meyers, L. D. (Eds.). (2006). *Dietary Reference Intakes: The essential guide to nutrient requirements*. Washington, DC: National Academies Press.

Owen, C. G., Martin, R. M., Whincup, P. H., Davey Smith, G., & Cook, D. G. (2005). Effect of infant feeding on the risk of obesity across the life course: A quantitative review of the published evidence. *Pediatrics, 115*,1367–1377.

Patelarou, E., Girvalaki, C., Brokalaki, H., Patelarou, A., Androulaki, Z., & Vardavas, C. (2012). Current evidence on the associations of breastfeeding, infant formula, and cow's milk introduction with type 1 diabetes mellitus: A systematic review. *Nutrition Reviews*, *70*, 509–519. doi:10.1111/j.1753-4887.2012.00513.x

Pelto, G. (2000). Improving complementary feeding practices and responsive parenting as a primary component of interventions to prevent malnutrition in infancy and early childhood. *Pediatrics, 106*, 1300–1301.

Ponza, M., Devaney, B., Ziegler, P., Reidy, K., & Squatrito, C. (2004). Nutrient intakes and food choices of infants and toddlers participating in WIC. *Journal of the American Dietetic Association, 104*, S71–S79.

Prescott, S. L., Smith, P., Tang, M., Palmer, D. J., Sinn, J., Huntley, S. J., … Makrides, M. (2008). The importance of early complementary feeding in the development of oral tolerance: Concerns and controversies. *Pediatric Allergy and Immunology*, *19*, 375–380.

Radlovic, N. P., Mladenovic, M. M., Lekovic, Z. M., Stojsic, Z. M., & Radlovic, V. N. (2010). Influence of early feeding practices on celiac disease in infants. *Croatian Medical Journal*, *51*(5), 417–422.

Rajakumar, K., & Thomas, S. (2005). Reemerging nutritional rickets: A historical perspective. *Archives of Pediatric and Adolescent Medicine, 159*, 335–341.

Rossander-Hulten, L., Brune, M., Sandstrom, B., Lonnerdal, B., & Hallberg, L. (1991). Comparative inhibition of iron absorption by manganese and zinc in humans. *American Journal of Clinical Nutrition, 54*, 152–156.

Ryan, A. S., Wenjun, Z., & Acosta, A. (2002). Breastfeeding continues to increase into the new millennium. *Pediatrics, 110*, 1103–1109.

Saadi, H. F., Dawodu, A., Afandi, B., Zayed, R, Benedict, S., Nagelkerke, N., Hollis, B. W. (2009). Effect of combined maternal and infant vitamin D supplementation on vitamin D status of exclusively breastfed infants. *Maternal and Child Nutrition,5*, 25–32.

Saarinen, K. M., Pelkonen, A. S., Makela, M. G., & Savilahti, E. (2005). Clinical course and prognosis of cow's milk allergy are dependent on milk-specific IgE status. *Journal of Allergy and Clinical Immunology, 116*, 869–875.

Sakashita, R., Inoue, N., & Kamegai, T. (2004). From milk to solids: A reference standard for the transitional eating process in infants and preschool children in Japan. *European Journal of Clinical Nutrition, 58*, 643–653.

Sakashita, R., Inoue, N., & Tatsuki, T. (2003). Selection of reference foods for a scale of standards for use in assessing the transitional process from milk to solid foods in infants and pre-school children. *European Journal of Clinical Nutrition, 57*, 803–809.

Savino, F., Costamagna, M., Prino, A., Oggero, R., & Silvestro, L. (2002). Leptin levels in breast-fed and formula-fed infants. *Acta Paediatrics, 91*, 897–902.

Schach, B., & Haight, M. (2002). Colic and food allergy in the breastfed infant: Is it possible for an exclusively breastfed infant to suffer from food allergy? *Journal of Human Lactation, 18*, 50–52.

Schmitz, J. (2000). Complementary feeding and enteropathies. *Pediatrics, 106*, 1286.

Scott, J. A., Ng, S. Y., & Cobiac, L. (2012). The relationship between breastfeeding and weight status in a national sample of Australian children and adolescents. *BMC Public Health*, *7*, 107. doi:10.1186/1471-2458-12-107

Shamir, R. (2012). Can feeding practices during infancy change the risk of celiac disease? *Israel Medical Association Journal*, *14*, 50–52.

Sicherer, S. (2003). Clinical aspects of gastrointestinal food allergy in childhood. *Pediatrics, 111*, 1609–1616.

Siega-Riz, A. M., Deming, D. M., Reidy, K. C., Fox, M. K., Condon, E., & Briefel, R. R. (2010). Food consumption patterns of infants and toddlers: Where are we now? *Journal of the American Dietetic Association*, *110*, S38–51.

Silano, M., Agostoni, C., & Guandalini, S. (2010). Effect of the timing of gluten introduction on the development of celiac disease. *World Journal of Gastroenterology*, *16*, 1939–1941.

Skinner, J., Ziegler, P., Pac, S., & Devaney, B. (2004). Meal and snack patterns of infants and toddlers. *Journal of the American Dietetic Association, 104*, S65–S70.

Skinner, J., Ziegler, P., & Ponza, M. (2004). Transitions in infants' and toddlers' beverage patterns. *Journal of the American Dietetic Association, 104*, S45–S50.

Slaughter, C., & Bryant, A. H. (2004). Hungry for love: The feeding relationship in the psychological development of young children. *Permanente Journal, 8*, 23–29.

Snijders, B. E., Thijs, C., van Ree, R., & van den Brandt, P. A. (2008). Age at first introduction of cow milk products and other food products in relation to infant atopic manifestations in the first 2 years of life: The KOALA Birth Cohort Study. *Pediatrics*, *122*(1), e115–122.

Spatz, D. L. (2004). Ten steps for promoting and protecting breastfeeding for vulnerable infants. *Journal of Perinatal and Neonatal Nursing*, *18*, 385–396.

Stang, J. (2006). Improving the eating patterns of infants and toddlers. *Journal of the American Dietetic Association, 106*, S7–S9.

Stettler, N., Stallings, V. A., Troxel, A., Zhou, J., Schinnar, R., Nelson, S. E., … Strom, B. L. (2005). Weight gain in the first week of life and overweight in adulthood. *Circulation, 111*,1897–1903.

Stettler, N., Zemel, B. S., Kumanyika, S., & Stallings, V. A. (2002). Infant weight gain and childhood overweight status in a multicenter, cohort study. *Pediatrics, 109*, 194–199.

Szajewska, H., Chmielewska, A., Pieścik-Lech, M., Ivarsson, A., Kolacek, S., Koletzko, S., … PREVENTCD Study Group. (2012). Systematic review: Early infant feeding and the prevention of coeliac disease. *Alimentary Pharmacology and Therapeutics*, *36*(7), 607–18. doi:10.1111/apt.12023

Taveras, E., Scanlon, K., Birch, L., Rifas-Shiman, S., Rich-Edwards, J., & Gillman, M. (2004). Association of breastfeeding with maternal control of infant feeding at age 1 year. *Pediatrics, 114*, e577–e583.

Upham, J., & Holt, P. (2005). Environment and development of atopy. *Current Opinion in Allergy and Clinical Immunology, 5*, 167–172.

U.S. Food and Drug Administration. (2002). FDA warns about possible *Enterobacter sakazakii* infections in hospitalized newborns fed powdered infant formulas. FDA Talk Paper. Retrieved from www.lommen.com/pdf/FDA-Warns-About-Possible-Enterobacter-Sakazakii-In.aspx

Vadas, P., Wai, Y., Burks, W., & Perelman, B. (2001). Detection of peanut allergens in breast milk of lactating women. *Journal of the American Medical Association, 285*, 1746–1748.

van Odijk, J., Hulthen, L., Ahlstedt, S., & Borres, M. P. (2004). Introduction of food during the infant's first year: A study with emphasis on introduction of gluten and of egg, fish, and peanut in allergy-risk families. *Acta Paediatrics, 93*, 464–470.

Van Rheenen, P., & Brabin, B. J. (2004). Late umbilical cord-clamping as an intervention for reducing iron deficiency anaemia in term infants in developing and industrialized countries: A systematic review. *Annals of Tropical Paediatrics, 24*, 3–16.

Vennemann, M. M., Bajanowski, T., Brinkmann, B., Jorch, G., Yucesan, K., Sauerland, C., & Mitchell, E. A. (2009). Does breastfeeding reduce the risk of sudden infant death syndrome? *Pediatrics*, *123*(3), e406–e410.

Villalpando, S. (2000). Feeding mode, infections, and anthropometric status in early childhood. *Pediatrics, 106*, 1282–1283.

von Kries, R., Koletzko, B., Sauerwald, T., Mutius, E. von, Barnert, K., Grunert, V., & von Ross, H. (1999). Breast feeding and obesity: Cross sectional study. *British Medical Journal, 319*,147–150.

Wagner, C. L., Greer, F. R., and the Section on Breastfeeding and Committee on Nutrition. (2008). Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. *Pediatrics, 122*, 1142–1152.

Wagner, C. L., Hulsey, T. C., Fanning, D., Ebeling, M., & Hollis, B. W. (2006). High-dose vitamin D3 supplementation in a cohort of breastfeeding mothers and their infants; a 6-month follow-up pilot study. *Breastfeeding Medicine, 1*, 59–70.

Weaver, L. (2000). Gastrointestinal digestive and absorptive function. *Pediatrics*, *106*, 1280–1281.

Weisberg, P., Scablon, K., Li, R., & Cogswell, M. (2004). Nutritional rickets among children in the United States: Review of cases reported between 1986 and 2003. *American Journal of Clinical Nutrition, 80*(Suppl.), 1697S–16705S.

Wharton, B. (2000). Patterns of complementary feeding (weaning) in countries of the European Union: Topics for research. *Pediatrics, 106*, 1273–1274.

Wood, R. A. (2003). The natural history of food allergy. *Pediatrics, 111*, 1631–1637.

World Health Organization. (2003). *Global strategy for infant and young child feeding* [pamphlet]. Geneva, Switzerland: Author.

World Health Organization. (2009). *Acceptable medical reasons for use of breast-milk substitutes*. Geneva, Switzerland: Author. Retrieved from http://whqlibdoc.who.int/hq/2009/WHO_FCH_CAH_09.01_eng.pdf

World Health Organization in collaboration with Food and Agriculture Organization of the United Nations. (2007). *Safe preparation, storage and handling of powdered infant formula: Guidelines*. Geneva, Switzerland: WHO. Retrieved from www.who.int/foodsafety/publications/micro/pif_guidelines.pdf

World Health Organization Working Group on the Growth Reference Protocol & WHO Task Force on Methods for the Natural Regulation of Fertility. (2002). Growth of healthy infants and the timing, type and frequency of complementary foods. *American Journal of Clinical Nutrition, 76*, 620–627.

Zeiger, R. (2003). Food allergen avoidance in the prevention of food allergy in infants and children. *Pediatrics, 11*, 1662–1671.

Ziegler, E. E., Hollis, B. W., Melson, S. E., & Jeter, J. M. (2006). Vitamin D deficiency in breastfed infants in Iowa. *Pediatrics*, *118*, 603–610.

Zutavern, A., von Mutius, E., Harris, J., Mills, P., Moffatt, S., White, C., & Cullinan, P. (2004). The introduction of solids in relation to asthma and eczema. *Archives of Diseases in Children, 89*, 303–308.

# Special Section on the Social and Cultural Aspects of Breastfeeding

*Yeemay Su Miller, MS, RD, Virginia L. Marchant-Schnee, BS, and Rachelle Lessen, MS, RD, IBCLC*



## CHAPTER OUTLINE



### Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Describe who breastfeeds, and why some women do not breastfeed.
2. Compare breastfeeding legislation around the United States.

## A Brief History of Breastfeeding

Deciding whether to breastfeed and for how long is not as simple as deciding which car to buy. Many complex social and cultural factors play interrelated roles in a woman's decision to breastfeed. Until the mid-eighteenth century, aristocratic families, and later the urban middle class, employed wet nurses to feed their infants as the social norm until the age of weaning, usually at 2 years of age. The ability to hire a wet nurse was regarded as a status symbol, and the mothers who hired wet nurses could then carry out upper-class social obligations and civic duties. One could draw parallels

between these women and today's mothers who choose to bottle-feed, free from being the only one who can perform the frequent and sometimes taxing task of feeding their infants. In an ironic twist of modern times, breastfeeding rates are higher among more educated and higher-income older mothers, whereas less-educated lower-income mothers are more likely to bottle-feed their infants (Centers for Disease Control and Prevention [CDC], 2005).

## Current Trends Affecting Breastfeeding

Throughout history men have dictated the significance of the female breast, from sacred and life giving to a sensual erotic object. Not until the later half of the twentieth century did women begin to repossess their breasts, with increasing numbers of women seeking breast augmentation surgeries, which place second only to liposuction as the most common cosmetic surgery. Sociologist Barbara Behrmann notes in her book, *The Breastfeeding Café* (2005), "Women in the U.S. nurse [their babies] in a culture in which our breasts are used to sell everything from cars to beer; in which deep cleavage dominates the checkout aisle … and in which the number of women who artificially enhance their breasts has increased 533% from 1992 to 2002 (p. 190)."

Surveys conducted by the American Society of Plastic Surgeons in 2001 reveal that 206,354 women underwent breast augmentation (i.e., breast implants) in the United States. The actual risk of impeding future breastfeeding varies, depending on the type of procedure, the skill and techniques of the surgeon, and the physiology of the individual woman's breasts. Some women who receive breast implants may have the ability to do some breastfeeding as long as the surgery is done skillfully with proper technique (Hefter, Lindholm, & Elvenes, 2003).

Reduction mammoplasty (breast reduction surgery) procedures have tripled from 47,874 performed in 1997 to 126,614 in 2002 (Plastic Surgery Information Service, n.d.). Unfortunately, women who undergo this particular surgery are likely unable to breastfeed exclusively or successfully for very long (Souto, Giugliani, Giugliani, & Schneider, 2003). The research of Souto and coworkers with a cohort of Brazilian women who had undergone reduction mammoplasty revealed that most of them expressed a strong wish and intention to breastfeed and were told overoptimistically by their surgeons that lactation would be preserved. However, the women who had undergone reduction mammoplasty were not as successful with any or exclusive breastfeeding when compared with control subjects. These women expressed that they would have undergone the surgery even if told lactation may not be preserved (Souto et al., 2003).

## Who Breastfeeds?

Breastfeeding rates vary greatly by state, ethnicity, maternal education, maternal age, marital status, and family income (CDC, 2011, 2013). The Centers for Disease Control and Prevention (CDC) report card on breastfeeding collects data state by state to examine breastfeeding practices and monitor progress. It is used to identify opportunities for health professionals, legislators, employers, business owners, families, and communities to protect, promote, and support breastfeeding. It is indeed encouraging to see that since the release of the first CDC Breastfeeding Report Card in 2007 there has been a steady increase in exclusive breastfeeding rates from 30% to 35% at 3 months and from 11% to 15% at 6 months. In addition to measuring breastfeeding rates as outcome indicators, the CDC also reports on process indictors that affect breastfeeding. These include state-by-state birth facility support, professional and peer counselor support, state health department programs, and support in childcare settings. These indicators allow program developers and public health leaders to monitor progress toward achieving their breastfeeding goals.

In 2012, 77% of all infants born in the United States ever breastfed, 47% were still breastfeeding at 6 months and 26% continued to breastfeed until 1 year. At 1 week of age, 54% were exclusively breastfeeding, indicating that many breastfed infants are also receiving infant formula. (See the Breastfeeding section on the CDC website at www.cdc.gov/breastfeeding/data/NIS_data/index.htm.) In fact, 25% of breastfed infants are supplemented with infant formula within the first 2 days of life. The greatest number of breastfeeding women resides in the western states, while rates are lowest in the South. Highest rates of breastfeeding are seen among married women older than 30 years of age with higher levels of education. There are also substantial racial and ethnic differences in

breastfeeding rates with additional differences noted state by state. Non-Hispanic blacks had a lower prevalence of breastfeeding initiation than did non-Hispanic whites. Hispanics generally have lower rates of breastfeeding than whites in western states, but higher rates in eastern states. Black women have the lowest rates of breastfeeding (see "Racial and Ethnic Differences in Breastfeeding Initiation and Duration," 2010).

A report by the CDC showed that children were more likely to have ever been breastfed if they were ineligible for or did not receive Women, Infants, and Children (WIC) assistance (CDC, 2004). Another study showed that women who participated in the WIC program were significantly less likely to breastfeed to 6 months of age and that this factor was stronger than other demographic characteristics (Ryan & Zhou, 2006). Research also showed that overweight and obese mothers are less likely to breastfeed and for shorter duration than mothers of normal weight and body mass index (Kugyelka, Rasmussen, & Frongillo, 2004; Lovelady, 2005; Rasmussen & Kjolhede, 2004).

## Barriers to Breastfeeding

In 2011, the U.S. Surgeon General published the *Call to Action to Support Breastfeeding* that describes the steps needed from a public health perspective to increase support for breastfeeding families in the community and employment setting (U.S. Department of Health and Human Services [U.S. DHHS], 2011). This document acknowledges the importance of breastfeeding for the health of mothers and babies and recognizes that without the commitment of clinicians, employers, communities, researchers, and government leaders, mothers will be unable to achieve their personal goals. Specifically, the document addresses barriers to breastfeeding in the United States and the actions needed to overcome these barriers.

According to the American Academy of Pediatrics, breastfeeding and the use of human milk are public health issues and not merely lifestyle choices (American Academy of Pediatrics [AAP] Section on Breastfeeding, 2012). The academy urges pediatricians to become strong advocates for breastfeeding by supporting families in their practices and to avoid nonmedically indicated formula supplementation. They call on pediatricians to serve as breastfeeding supporters and educators and to advocate for change in hospital practices. Of particular concern, it appears that pediatricians' attitudes and commitment to breastfeeding have deteriorated and many pediatricians reportedly believe that the difficulties or inconvenience of breastfeeding outweigh any benefits (Feldman-Winter, Schanler, O'Connor, & Lawrence, 2008). Education and training of physicians can improve knowledge, attitudes, practice patterns, confidence, and breastfeeding rates.

Within the U.S. medical system, all healthcare providers, including obstetricians, pediatricians, and nurses, need more education on the physiology of lactation and the mechanics of breastfeeding to best support their breastfeeding patients. Yet the breadth of knowledge on these topics taught by most medical schools is likely limited. Providers inadvertently sabotage a mother's efforts to breastfeed exclusively by recommending supplementation with formula when they fail to recognize that breastfed infants gain weight differently from formula-fed peers or recommend complete weaning when a mother reports a lactation problem rather than a referring her to a qualified lactation consultant. National surveys reveal that healthcare professionals who are educated on how best to support lactation in their patients play an important role in influencing the mother's decision to breastfeed and for longer duration (Lu, Lange, Slusser, Hamilton, & Halfon, 2001).

When the method a mother chooses to feed her baby can be viewed as a lifestyle decision, it can be determined by the mother's and other family members' attitudes and beliefs about breastfeeding as well as healthcare professionals' views, employment, stress levels, and amount of social support (Donath, Amir, & ALSPAC Study Team, 2003). A mother's perception of the father's attitude or preferences on how the baby should be fed is one top determinant of the mother's decision to initiate bottle-feeding over breastfeeding (Arora, McJunkin, Wehrer, & Kuhn, 2000; Freed, Fraley, & Schanler, 1992). Studies have also reported that the maternal grandmother's attitude also influences the type of feeding method used by the mother and positively correlates with longer duration of breastfeeding if she supports her daughter's decision to breastfeed (Donath et al., 2003; Swanson & Power, 2005). The perceived influence of other people's views (subjective norms), including the views of the women's partners, other family members, and healthcare providers, is an important predictor of

infant feeding behavior (Swanson & Power, 2005). Therefore, promoting breastfeeding as a positive norm and as the ideal method to feed an infant within a mother's broad social context increases initiation and continuation of breastfeeding.

## Routine Maternity Care Practices

Research has identified specific hospital nursery and maternity ward practices that interfere with breastfeeding, especially in the critical first week. Separating the mother and newborn without medical necessity can prevent the establishment of breastfeeding during the first hour of life when newborns are most alert. Introducing pacifiers and bottles to newborns too early often causes "nipple confusion" because the method of sucking artificial replacements is completely different from the type of suck needed to extract milk from a breast. As a result, an infant may suck improperly or inadequately at the breast.

Supplementation with formula or "sugar water" and pacifier use can also depress the infant's instinct to breastfeed frequently, which helps establish the mother's milk supply. The most recent research correlates early pacifier use with a significant decline in exclusive breastfeeding. In addition, "across all types of breastfeeding (exclusive, full, and overall), the most significant predictor of duration was the receipt of supplemental feedings while in the hospital (p. 516)" (Howard et al., 2003).

The World Health Organization (WHO) outlines a number of maternity care practices for the birthplace, whether it is hospital, clinic, or birth center, that facilitate and support breastfeeding; if these practices are fully implemented, the hospital earns the designation of "baby-friendly." A recently published study of baby-friendly hospitals and birth centers in the United States showed that women who gave birth in a baby-friendly setting initiated breastfeeding and exclusively breastfed their infants in the early postpartum period at significantly higher rates than state, regional, and national rates, and these rates were consistently elevated in a variety of settings (Merewood, Mehta, Chamberlain, Philipp, & Bauchner, 2005). The number of baby-friendly facilities in the United States is increasing. In 2012, there were 150 hospitals and birth centers in 34 states with the designation, with an increase from 2.9% in 2009 to 5.8% in 2012 of all U.S. birth facilities providing the recommended care for lactating mothers and babies. In addition to the UNICEF and WHO Baby-Friendly Hospital Initiative, the U.S. Department of Health and Human Services and the CDC advocate that every facility providing maternity services and care for newborn infants should follow these 10 steps to successful breastfeeding:

1. Have a written breastfeeding policy that is routinely communicated to all healthcare staff
2. Train all healthcare staff in skills necessary to implement this policy
3. Inform all pregnant women about the benefits and management of breastfeeding
4. Help mothers initiate breastfeeding within a half-hour of birth
5. Show mothers how to breastfeed and how to maintain lactation even if they should be separated from their infants
6. Give newborn infants no food or drink other than breast milk, unless *medically* indicated, and under no circumstances provide breast milk substitutes, feeding bottles, or pacifiers free of charge or at low cost
7. Practice rooming-in (allow mothers and infants to remain together) 24 hours a day
8. Encourage breastfeeding on demand
9. Give no artificial teats or pacifiers to breastfeeding infants
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic

## Physiologic and Psychological Factors

One of the most common reasons reported by women who introduce formula to their babies and thereby begin to decrease breastfeeding is "lack of confidence in the sufficiency of their breast milk" (Donath et al., 2003; Swanson & Power, 2005), which is a self-efficacy issue. Confidence in one's body to nourish an infant adequately from the breast is not necessarily a physiologic issue of adequate production or supply of milk. Unlike bottle-feeding, in which one can actually see how many ounces an infant is drinking, other measures, such as the number of wet diapers per day and proper weight gain over time, determine the adequacy of a breastfed infant's intake. Increasing a mother's self-efficacy and confidence with breastfeeding

appears to be important in helping women breastfeed successfully and for longer duration (Kools, Thijs, & de Vries, 2005; Mitra, Khoury, Hinton, & Carothers, 2004; Noel-Weiss, Bassett, & Cragg, 2006). Therefore, helping women combat their lack of self-confidence, providing reassurance, and reinforcing that the more she breastfeeds the more milk she will produce assist mothers in their breastfeeding goals.

Breastfeeding can be painful initially for many women even when done correctly (i.e., correct positioning and hold of the infant, proper latch-on). Flat or inverted nipples, scar tissue, bacterial or yeast infection, changes in hormone levels, plugged ducts, an infant with a vigorous suck, or other underlying causes may not be immediately diagnosed, leading a mother to believe that breastfeeding will always cause pain and to wean her infant prematurely. However, often these problems can be addressed by knowledgeable healthcare professionals or may even resolve themselves within a few weeks. A study of breastfeeding discontinuation reported that 14% of women who initiated breastfeeding discontinued between the first and third weeks because of breast pain or soreness, whereas only 4% discontinued between the fourth and sixth weeks, and none after the seventh week (Taveras et al., 2003).

In addition, women with more symptoms of maternal depression had greater odds of discontinuing breastfeeding by 12 weeks. A woman's predelivery perception of breastfeeding and the strength of her intention to breastfeed also determine whether or not she will initiate breastfeeding and continue to do so "through the vulnerable post delivery period when women may experience the most discomfort" (Ahluwalia, Morrow, & Hsia, 2005). Healthcare providers interested in promoting breastfeeding should provide women with the tools and support to overcome potential difficulties, including pain and soreness.

## Social Support and Acculturation

In some industrialized societies, but not all, breastfeeding has been intertwined with sexuality, making it difficult for some people to separate the two distinctly different functions of the breast. Some ignore the biological role of the breast as a mammary gland to produce milk ideally suited for the nutritional needs of a human child. Other individuals misconstrue breastfeeding in public as a form of indecent exposure. As author Gabrielle Palmer (1988) states in *The Politics of Breastfeeding*, "The very reason it [breastfeeding] is frowned upon in public is that breasts are perceived exclusively as objects of sexual attention" (p. 119).

To increase the proportion of women who breastfeed and continue exclusively for at least 6 months, the U.S. Department of Health and Human Services in conjunction with the Ad Council launched the first-ever nationwide breastfeeding awareness campaign in 2004. Rather than just promoting the benefits of breastfeeding, the campaign featured television, radio, and print ads that emphasized the risks of not breastfeeding and its impact on infant and child health. The 2005 postsurvey results revealed that awareness of messages about breastfeeding rose from 28% to 38% and significantly more women surveyed had breastfed a child in the 2004–2005 study (73%) than in the 2004 study (63%). In regard to breastfeeding in public, 42% of respondents surveyed in 2005 reported being somewhat comfortable or very comfortable with breastfeeding their infant in public, a 3% increase from the 2004 study, whereas 48% of women surveyed in 2005 reported being somewhat uncomfortable or very uncomfortable with breastfeeding their infant in a public place, which is a 1% increase from 2004 survey results. Clearly, much more needs to be done to enable women to feel more comfortable with breastfeeding in public and to make it a social norm.

Not only is American society often unsupportive of breastfeeding women, it can be outright hostile to women who breastfeed in public. Women from various regions of the country and different walks of life have reported incidents of discrimination and harassment while breastfeeding their children in public places such as restaurants, public pools, shopping malls, and supermarkets. Many times these stories appear in the local media, accompanied by editorials or letters to the editor, and some even make it into the national news. These media controversies and the public debate over public breastfeeding lead many women to view breastfeeding negatively when deciding on how to feed their infants.

The same factors that influence whether a mother initiates breastfeeding, as previously mentioned, also determine the *duration* of breastfeeding. Based on the AAP recommendation that a mother breastfeed her infant for at least 12 months, breastfeeding beyond this age has been reported in U.S. literature as "extended

breastfeeding." However, the average age of weaning throughout the world, historically and currently, falls between ages 2 and 4 years (Stuart-Macadam & Dettwyler, 1995).

In addition, an analysis of data from NHANES indicates that the more highly acculturated a Hispanic woman becomes in the United States, the less likely she will breastfeed. Conversely, the less acculturated a Hispanic woman is in the United States, the more likely she will breastfeed in keeping with the rate of her country of origin (Gibson et al., 2005).

## Marketing of Breast Milk Substitutes

Expectant mothers are heavily influenced by the plethora of advertisements in parenting magazines, marketing materials in their doctors' offices, coupons for free or steeply discounted breast milk substitutes (formula) that arrive in the mail, and the free samples distributed by the hospital where they give birth. Research suggests that this advertising during pregnancy seriously undermines a future breastfeeding relationship (Howard, Howard, Lawrence, Andresen, & DeBlieck, 2000). Formula companies also target their marketing to women who remain undecided or intend to just "give breastfeeding a try." This deluge of marketing material affects the feeding decisions of mothers to such an extent that the manufacture and sale of breast milk substitutes has grown into an $8 billion industry.

Hospital distribution of discharge packs or "gift bags" provided by major pharmaceutical companies has been shown to undermine breastfeeding, especially exclusive breastfeeding (Donnelly, Snowden, Renfew, & Woolridge, 2004). These bags contain cans or bottles of formula, with marketing materials including coupons for more formula and sometimes packaged with "breastfeeding success" brochures or books on infant care. In 2007, a nationwide study found that only 9% of U.S. hospitals did not distribute discharge packs. As a result, a national campaign called Ban the Bags was launched to encourage changes in hospital practice related to industry-sponsored formula sample-pack distribution (Merewood et al., 2010). In 2010, a follow-up study found a significant increase in the number of hospitals that eliminated the practice of formula pack distribution. By looking at the states with the highest and lowest rates of formula pack distribution, they found huge disparities in practice. Among the best record states, Rhode Island went from 43% to 86% of hospitals eliminating distribution packs, and in Massachusetts, the home of the Ban the Bags movement, rates of eliminating distribution packs increased from 27% in 2007 to 64% in 2010. Equally impressive was Texas where the Texas Ten Steps Program encourages hospitals to support breastfeeding including eliminating distribution of formula packs. In 2010, 15% of Texas hospital discontinued formula pack distribution compared to only 1% of hospitals in 2007 (Sadacharan, Grossman, Sanchez, & Merewood, 2011).

As awareness of the negative effects of hospital distribution of industry-sponsored formula discharge packs increases, formula companies are seeking alternative pathways to market and distribute their products, including distribution through pediatrician and clinic offices. To address this concern, the AAP issued a compelling statement that distribution of free samples and literature is a form of marketing and creates a conflict of interest for healthcare professionals. They urge pediatricians not to provide formula company gift bags, coupons, and industry-authored handouts to the parents of newborns and infants in office and clinic settings (AAP Section on Breastfeeding, 2012). (See Divesting from Formula Marketing in Pediatric Care at www2.aap.org/breastfeeding/files/pdf/DivestingfromFormulaMarketinginPediatricCare.pdf.)

To curtail the inappropriate marketing of formula that interferes with lactation, the WHO set forth an International Code of Marketing of Breast Milk Substitutes in 1981 (World Health Organization [WHO], 1981), which the United States also adopted in 1994. The main goal of the WHO Code is "to contribute to the provision of safe and adequate nutrition for infants, by the protection and promotion of breastfeeding and by the proper use of breast milk substitutes, when these are necessary, on the basis of adequate information and through appropriate marketing and distribution" (WHO, 1981). The Code specifically prohibits the following activities:

- Advertising of breast milk substitutes
- Distributing free samples of breast milk substitutes to mothers
- Promoting breast milk substitutes through healthcare facilities
- Using company-appointed "nurses" to "advise" mothers on bottle-feeding

- Giving gifts or personal samples to health workers
- Placing words or pictures idealizing artificial feeding, including pictures of infants, on the labels of the products
- Promoting unsuitable products for infants, such as sweetened condensed milk

In addition, the Code states that

- Information to health workers should be scientific and factual
- All information on artificial feeding, including the labels, should explain the benefits of breastfeeding and the costs and hazards associated with artificial feeding
- All products should be of high quality and take into account the climatic and storage conditions of the country where they are used

## Returning to Work

The most recent available statistics from the U.S. Department of Labor (2008) show that 56% of female employees are women with infants and children younger than the age of 1 year, and they are the fastest growing segment of today's labor force. At least 50% of women who are employed when they become pregnant return to the labor force by the time their child reaches 3 months of age. Research indicates that the timing of the mother's resumption of employment is a key factor that influences the duration of exclusive breastfeeding, and workplace policies and practices, particularly maternity/parental leave provisions, have considerable potential to positively influence breastfeeding practices (Galtry, 2003). Because there is a positive association between length of maternity leave and duration of breastfeeding, some contend that a country's breastfeeding rates are influenced by and reflected in its maternity leave programs (U.S. Department of Labor, 2008).

In Norway and Sweden, which have the highest breastfeeding rates in the world, women are entitled to 12 months and 18 months, respectively, of job-protected maternity and child care leave and compensation at 80% to 100% of normal earnings (Organization for Economic Co-operation and Development, 2001). A 2005 report of the International Labor Organization found that for most industrialized countries, 75% to 100% of pay is guaranteed for up to 16 weeks of maternity leave. In stark contrast, the United States mandates only up to a 12-week maternity leave with no entitled pay and has relatively low breastfeeding rates in comparison with other industrialized countries. In addition, a 2000 report to the U.S. Congress on family and medical leave policies found that 77% of those surveyed who were eligible for Family Medical Leave did not take it, stating they could not afford to do so (Waldfogel, 2001).

The earlier a mother returns to work, the more likely her duration of breastfeeding will decrease (Piper & Parks, 1996; Visness & Kennedy, 1997; Vogel, Hutchison, & Mitchell, 1999), and the odds of a woman being able to continue breastfeeding after returning to work increase when her work hours are part-time as opposed to full-time. "Improved maternity leave provisions and more flexible working conditions may help women to remain at home with their infants longer and/or to combine successfully breastfeeding with employment outside the home" (Scott, Binns, Oddy, & Graham, 2006). Employers with more than 50 employees are now required to provide reasonable break time for an employee to express breast milk for her nursing child for 1 year after the child's birth. The employee will be provided a place other than a bathroom that is shielded from view and free from intrusion from coworkers and the public. This break time was mandated when the Affordable Care Act was signed into law on March 23, 2010, and is seen as a major advance in the protection and support for breastfeeding.

The *Business Case for Breastfeeding* is a comprehensive program designed by the U.S. Department of Health and Human Services to educate employers about the value of supporting breastfeeding employees in the workplace (U.S. DHHS, 2012). The program emphasizes the benefits to the entire company with significant payoff through retention of experienced employees, reduction in sick time taken by both moms and dads for children's illnesses, and lower healthcare and insurance costs. Companies that are successful in retaining employees after childbirth have found that having a dedicated space that provides privacy for women to express their milk at work and providing workplace lactation support really make a difference. Employers are encouraged to be creative in designing programs to support breastfeeding mothers in the workplace, such as dedicated rooms in airports or malls for employees, or designated

spaces in public buildings such as a WIC office or city government building for women with careers that require them to be on the road such as police officers, bus drivers, or mail carriers.

Although providing lactation rooms for pumping allows mothers to continue providing breast milk for their infants, this is not the only option for working mothers. Direct breastfeeding also can be combined with employment. This has been found to be the most effective strategy for successfully combining breastfeeding and work (Fein, Mandal, & Roe, 2008). Direct breastfeeding, rather than using an employee lactation room for pumping, can be achieved with on-site child care, telecommuting, taking the infant to work, allowing the mother to leave work to breastfeed the infant, or having the infant brought to the workplace.

## Legislation: Protecting a Woman's Right to Breastfeed

Women encounter multiple obstacles and barriers preventing them from achieving breastfeeding success. A woman's right to breastfeed is best protected by the government. A federal law states that a mother may breastfeed her child on any federal property or in any federal building where she and her child are otherwise allowed to be. In addition, enacting state legislation has been an effective way to protect, promote, and support breastfeeding. Florida and New York enacted the first state laws concerning breastfeeding in 1993 and 1994, and since then many state legislatures have added breastfeeding to their general statutes as a matter of public health policy. This legislation ranges from the protection of a mother's right to breastfeed in public, to requirements that an employer should accommodate a nursing mother's need to express her milk, to exemptions from jury duty for nursing mothers, to family law including child custody issues, to support services and education. Forty-five states, the District of Columbia, and the Virgin Islands have laws that specifically allow women to breastfeed in any public or private location, including Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, Florida, Georgia, Hawaii, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Vermont, Washington, Wisconsin, and Wyoming.

Twenty-eight states, the District of Columbia, and the Virgin Islands exempt breastfeeding from public indecency laws, including Alaska, Arizona, Arkansas, Florida, Illinois, Kentucky, Louisiana, Massachusetts, Michigan, Minnesota, Mississippi, Montana, Nevada, New Hampshire, New York, North Carolina, North Dakota, Oklahoma, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Utah, Virginia, Washington, Wisconsin, and Wyoming.

Twenty-four states, the District of Columbia, and Puerto Rico have laws related to breastfeeding in the workplace, including Arkansas, California, Colorado, Connecticut, Georgia, Hawaii, Illinois, Indiana, Maine, Minnesota, Mississippi, Montana, New Mexico, New York, North Dakota, Oklahoma, Oregon, Rhode Island, Tennessee, Texas, Vermont, Virginia, Washington, and Wyoming.

Twelve states and Puerto Rico exempt breastfeeding mothers from jury duty, including California, Idaho, Illinois, Iowa, Kansas, Kentucky, Mississippi, Montana, Nebraska, Oklahoma, Oregon, and Virginia.

Five states and Puerto Rico have implemented or encouraged the development of a breastfeeding awareness education campaign, including California, Illinois, Minnesota, Missouri, and Vermont.

States that have unique laws related to breastfeeding rights include Virginia, which allows women to breastfeed on any land or property owned by the state, and Puerto Rico, which requires shopping malls, airports, public service government centers, and other select locations to have accessible areas designed for breastfeeding and diaper changing that are not bathrooms.

At least two states have laws related to child-care facilities and breastfeeding. Louisiana prohibits any child-care facility from discriminating against breastfed babies. Mississippi requires licensed child-care facilities to provide breastfeeding mothers with a sanitary place that is not a toilet stall to breastfeed their children or express milk, to provide a refrigerator to store expressed milk, to train staff in the safe and proper storage and handling of human milk, and to display breastfeeding promotion information to the clients of the facility.

California requires the Department of Public Health to develop a training course of

hospital policies and recommendations that promote exclusive breastfeeding and specify staff for whom this model training is appropriate. The recommendation is targeted at hospitals with patients who ranked in the lowest 25% of the state for exclusive breastfeeding rates.

Maryland exempts the sale of tangible personal property that is manufactured for the purpose of initiating, supporting, or sustaining breastfeeding from the sales and use tax.

California, New York, and Texas have laws related to the procurement, processing, distribution, or use of human milk. And New York created a Breastfeeding Mothers Bill of Rights, which is required to be posted in maternal healthcare facilities. New York also created a law that allows a child younger than 1 year of age to accompany the mother to a correctional facility if the mother is breastfeeding at the time she is committed (National Conference of State Legislatures, 2011).

## References


Ahluwalia, I. B., Morrow, B., & Hsia, J. (2005). Why do women stop breastfeeding? Findings from the pregnancy risk assessment and monitoring system. *Pediatrics*, *116*, 1408–1412.

American Academy of Pediatrics Section on Breastfeeding. (2012). Policy statement: Breastfeeding and the use of human milk. *Pediatrics*, *129*, e827–e841.

Arora, S., McJunkin, C., Wehrer, J., & Kuhn, P. (2000). Major factors influencing breastfeeding rates: Mother's perception of father's attitude and milk supply. *Pediatrics*, *106*, E67.

Baby Milk Action. (n.d.). Home page. Retrieved from www.babymilkaction.org

Behrmann, B. (2005). *The breastfeeding café*. Ann Arbor: University of Michigan Press.

Bulk-Bunschoten, A. M., van Bodegom, S., Reerink, J. D., Pasker-de Jong, P. C., & de Groot, C. J. (2001). Reluctance to continue breastfeeding in the Netherlands. *Acta Paediatrics*, *90*, 1047–1053.

Celi, A. C., Rich-Edwards, J. W., Richardson, M. K., Kleinman, K. P., & Gillman, M. W. (2005). Immigration, race/ethnicity, and social and economic factors as predictors of breastfeeding initiation. *Archives of Pediatrics & Adolescent Medicine*, *159*, 255–260.

Centers for Disease Control and Prevention. (2005). Breastfeeding: Data and statistics. Breastfeeding practices—results from the 2004 National Immunization Study. Retrieved from www.cdc.gov/breastfeeding/data/NISdata/data2004.htm

Centers for Disease Control and Prevention. (2011). Breastfeeding report card—United States, 2011. Retrieved from www.cdc.gov/breastfeeding/pdf/2011BreastfeedingReportCard.pdf

Centers for Disease Control and Prevention. (2013). Breastfeeding. Retrieved from www.cdc.gov/breastfeeding/data/NIS_data/index.htm

Donath, S. M., Amir, L. H., & ALSPAC Study Team. (2003). Relationship between prenatal infant feeding intention and initiation and duration of breastfeeding: A cohort study. *Acta Paediatrics*, *92*, 352–356.

Donnelly, A., Snowden, H. M., Renfew, M. J., & Woolridge, M. W. (2000). Commercial hospital discharge packs for breastfeeding women. *Cochrane Database of Systematic Reviews*, (2), CD002075.

Ekulona, E. (1996). Family health: Taking responsibility/making the difference. *Healthy Start Father's Journal*, 1–18.

Fein, S. B., Mandal, B., & Roe, B. E. (2008). Success of strategies for combining employment and breastfeeding. *Pediatrics*, *122*, S56–S62.

Feldman-Winter, L. B., Schanler, R. J., O'Connor, K. G., & Lawrence, R. A. (2008). Pediatricians and the promotion and support of breastfeeding. *Archives of Pediatric and Adolescent Medicine*, *162*, 1142–1149.

Freed, G. I., Fraley, J. K., & Schanler, R. J. (1992). Attitudes of expectant fathers regarding breastfeeding. *Pediatrics*, *90*, 224–227.

Galtry, J. (2003). The impact on breastfeeding of labour market policy and practice in Ireland, Sweden, and the USA. *Social Science and Medicine*, *57*, 167–177.

Gibson, M. V, Diaz, V. A., Mainous, A. G., & Greesey, M. E. (2005). Prevalence of Breastfeeding and Acculturation in Hispanics: Results from the NHANES 1999–2000 Study. *Birth, 32*, 93–98.

Gibson-Davis, C. M., & Brooks-Gunn, J. (2006). Couples' immigration status and ethnicity as determinants of breastfeeding. *American Journal of Public Health*, *96*, 641–646.

Giles, F. (2003). *Fresh milk: The secret lives of breasts*. New York, NY: Simon and Schuster.

Giovannini, M., Riva, E., Banderali, G., Scaglioni, S., Veehof, S. H., Sala, M., … Agostoni, C. (2004). Feeding practices of infants through the first year of life in Italy. *Acta Paediatrica*, *93*, 492–497.

Haynes, S. G. (2005). *National Breastfeeding Awareness Campaign Results* [Slide Presentation]. Washington, DC: U.S. Department of Health and Human Services Office on Women's Health.

Heath, A. L., Tuttle, C. R., Simons, M. S., Cleghorn, C. L., & Parnell, W. R. (2002). A longitudinal study of breastfeeding and weaning practices during the first year of life in Dunedin, New Zealand. *Journal of the American Dietetic Association*, *102*, 937–943.

Hefter, W., Lindholm, P., & Elvenes, O. P. (2003). Lactation and breast-feeding ability following lateral pedicle mammaplasty. *British Journal of Plastic Surgery*, *56*, 746–751.

Howard, C. R., Howard, F. M., Lanphear, B., Eberly, S., deBlieck, E. A., Oakes, D., & Lawrence, R. A. (2003). Randomized clinical trial of pacifier use and bottle-feeding or cupfeeding and their effect on breastfeeding. *Pediatrics*, *111*, 511–518.

Howard, C. R., Howard, F., Lawrence, R., Andresen, E., & DeBlieck, E. (2000). Office prenatal formula advertising and its effects on breastfeeding patterns. *Obstetrics and Gynecology*, *95*, 296–303.

Hurst, N. (2003). Breastfeeding after breast augmentation. *Journal of Human Lactation*, *19*, 7–8.

International Labour Organization. (2005, June 15). International Labour Organization adopts new convention and

recommendation on maternity protection. Retrieved from www.ilo.org/global/about-the-ilo/media-centre/press-releases/WCMS_007900

Johnson, J. O., & Downs, B. (2005). Maternity leave and employment patterns of first time mothers: 1961–2000. *Current Population Reports*. Retrieved from www.census.gov/prod/2005pubs/p70-103.pdf

Jordan, L. (1999, July 29). *Recent Family and Medical Leave proposals and European leave laws*. OLS Research Report 99-0760. Connecticut General Assembly, Office of Legislative Research. Retrieved from www.cga.ct.gov/ps99/rpt/olr/99-r-0760.doc

Kools, E. J., Thijs, C., & de Vries, H. (2005). The behavioral determinants of breast-feeding in the Netherlands: Predictors for the initiation of breast-feeding. *Health Education Behaviors*, *32*, 809–824.

Kronborg, H., & Vaeth, M. (2004). The influence of psychosocial factors on the duration of breastfeeding. *Scandinavian Journal of Public Health*, *32*, 210–206.

Kugyelka, J. G., Rasmussen, K. M., & Frongillo, E. A. (2004). Maternal obesity is negatively associated with breastfeeding success among Hispanic but not Black women. *Journal of Nutrition*, *134*, 1746–1753.

Le Leche League International. (n.d.). Breastfeeding and the law: A current summary of breastfeeding legislation in the US. Retrieved from www.llli.org/law/lawus.html

Lewin, A. M. (2005). Breast-feeding moms take action: "Lactivists" and lawmakers push to allow public nursing. *ABC News*, December 20.

Lovelady, C. A. (2005). Is maternal obesity a cause of poor lactation performance? *Nutrition Reviews*, *63*, 352–355.

Lu, M. C., Lange, L., Slusser, W., Hamilton, J., & Halfon, N. (2001). Provider encouragement of breast-feeding: Evidence from a national survey. *Obstetrics and Gynecology*, *97*, 290 –295.

McIntyre, E., Hiller, J. E., & Turnbull, D. (2001). Attitudes towards infant feeding among adults in a low socioeconomic community: What social support is there for breastfeeding? *Breastfeeding Reviews*, *9*, 13–24.

Merewood, A., Grossman, K., Cook, J., Sadacharan, R., Singleton, M., Peters, K., & Navidi, T. (2010). US hospitals violate WHO policy on the distribution of formula sample packs: Results of a national survey. *Journal of Human Lactation*, *26*, 363–367.

Merewood, A., Mehta, S. D., Chamberlain, L. B., Philipp, B. L., & Bauchner, H. (2005). Breastfeeding rates in US baby-friendly hospitals: Results of a national survey. *Pediatrics*, *116*, 628–634.

Meyerink, R. O., & Marquis, G. S. (2002). Breastfeeding initiation and duration among low-income women in Alabama: The importance of personal and familial experiences in making infant-feeding choices. *Journal of Human Lactation*, *8*, 38–45.

Mitra, A. K., Khoury, A. J., Hinton, A. W., & Carothers, C. (2004). Predictors of breastfeeding intention among low-income women. *Maternal and Child Health Journal*, *8*, 65–70.

National Conference of State Legislatures. (2011, May). Breastfeeding laws. Retrieved from www.ncsl.org/issues-research/health/breastfeeding-state-laws.aspx

Noel-Weiss, J., Bassett, V., & Cragg, B. (2006). Developing a prenatal breastfeeding workshop to support maternal breastfeeding self-efficacy. *Journal of Obstetric, Gynecologic, and Neonatal Nursing*, *35*, 349–357.

Nommsen-Rivers, L. (2003). Cosmetic breast surgery—is breastfeeding at risk? *Journal of Human Lactation*, *19*, 7–8.

Oregon Department of Human Services. (2013). Working to create a community that supports breastfeeding. Retrieved from http://public.health.oregon.gov/HealthyPeopleFamilies/Babies/Breastfeeding/Pages/index.aspx

Organization for Economic Co-operation and Development. (2001). Balancing work and family life: Helping parents into paid employment. In *Employment Outlook* (pp. 129–166). Paris, France: Author.

Palmer, G. (1988). *The politics of breastfeeding*. London, England: Pandora Press.

Piper, S., & Parks, P. L. (1996). Predicting the duration of lactation: Evidence from a national survey. *Birth*, *23*, 7–12.

Plastic Surgery Information Service. Breast augmentation. (n.d.). Retrieved from www.plasticsurgery.org/cosmetic-procedures/breast-augmentation.html

Racial and ethnic differences in breastfeeding initiation and duration, by state—National Immunization Survey, United States, 2004–2008. (2010). *Morbidity and Mortality Weekly Report*, *59*, 327–334. Retrieved from www.cdc.gov/mmwr/preview/mmwrhtml/mm5911a2.htm#fig

Rasmussen, K. M., & Kjolhede, C. L. (2004). Prepregnant overweight and obesity diminish the prolactin response to suckling in the first week postpartum. *Pediatrics*, *113*, e465–e471.

Rea, M. F., & Morrow, A. L. (2004). Protecting, promoting, and supporting breastfeeding among women in the labor force. *Advances in Experimental Medical Biology*, *554*, 121–132.

Ryan, A. S., & Zhou, W. (2006). Lower breastfeeding rates persist among the special supplemental nutrition program for women, infants, and children participants, 1978–2003. *Pediatrics*, *117*, 1136–1146.

Ryan, A. S., Zhou, W., & Acosta, A. (2002). Breastfeeding continues to increase into the new millennium. *Pediatrics*, *110*, 1103–1109.

Sadacharan, R., Grossman, X., Sanchez, E., & Merewood, A. (2011). Trends in US hospital distribution of industry-sponsored infant formula sample packs. *Pediatrics*, *128*, 702–705.

Scott, J. A., Binns, C. W., & Aroni, R. A. (1995). Infant feeding practices in Perth and Melbourne: Report 1 (p. 71). Glen Iris, Australia: Australia Breastfeeding Association.

Scott, J. A., Binns, C. W., Oddy, W. H., & Graham, K. I. (2006). Predictors of breastfeeding duration: Evidence from a cohort study. *Pediatrics*, *117*, e646–e655.

Shealy, K. R., Li, R., Benton-Davis, S., & Grummer-Strawn, L. M. (2005). *The CDC guide to breastfeeding interventions*. Washington, DC: U.S. Department of Health and Human Services and Centers for Disease Control and Prevention.

Souto, G. C., Giugliani, E. R., Giugliani, C., & Schneider, M. A. (2003). The impact of breast reduction surgery on breastfeeding performance. *Journal of Human Lactation*, *19*, 43–49, quiz 66–69, 120.

Stuart-Macadam, P., & Dettwyler, K. A. (1995). *Breastfeeding: Biocultural perspectives*. Hawthorne, NY: Aldine De Gruyter.

Swanson, V., & Power, K. G. (2005). Initiation and continuation of breastfeeding: Theory of planned behaviour. *Journal of Advanced Nursing*, *50*, 272–282.

Sweden's Statistics in Health and Diseases. (1998). Retrieved from www.sos.se/FULLTEXT/0042-007/0042-007.pdf

Taveras, E. M., Capra, A. M., Braveman, P. A., Jensvold, N. G., Escobar, G. J., & Lieu, T. A. (2003). Clinician support and psychosocial risk factors associated with breastfeeding discontinuation. *Pediatrics*, *112*, 108–115.

U.S. Department of Labor. (2008). Family Medical Leave Act. Retrieved from www.dol.gov/whd/fmla/index.htm. Accessed 09/24/2013. U.S. Department of Health and Human Services. (2000). *HHS blueprint for action on breastfeeding*. Washington, DC: U.S. Department of Health and Human Services, Office on Women's Health.

U.S. Department of Health and Human Services. (2011). *The surgeon general's call to action to support breastfeeding*. Washington, DC: U.S. Department of Health and Human Services, Office of the Surgeon General.

U.S. Department of Health and Human Services. (2012). *The business case for breastfeeding: Steps for creating a breastfeeding friendly work site*. Retrieved from www.womenshealth.gov/breastfeeding/government-in-action/business-case-for-breastfeeding/easy-steps-to-supporting-breastfeeding-employees.pdf

U.S. Department of Health and Human Services & Centers for Disease Control and Prevention. (n.d.). *Healthy People 2020: Maternal, infant, and child health*. Retrieved from http://healthypeople.gov/2020/default.aspx

U.S. Food and Drug Administration. (2000, September). Breast implants: An information update—2000. Retrieved from www.vidyya.com/pdfs/0915bip.pdf

Vance, M. R. (2005.) Breastfeeding legislation in the U.S. *Leaven, 41*, 3, 51–54.

van der Wal, M. F., de Jonge, G. A., & Pauw-Plomp, H. (2001). Increased percentages of breastfed infants in Amsterdam. *Ned Tijdschr Geneeskd*, *18*, 1597–1601. In Dutch.

Visness, C., & Kennedy, K. (1997). Maternal employment and breast-feeding: Findings from the 1988 National Maternal and Infant Health Survey. *American Journal of Public Health*, *87*, 945–950

Vogel, A., Hutchison, B., & Mitchell, E. (1999). Factors associated with the duration of breastfeeding. *Acta Paediatrica*, *88*, 1320–1326.

Waldfogel, J. (2001). Family and medical leave: Evidence from the 2000 surveys. *Monthly Labor Review*, *124*, 17–23. Retrieved from www.bls.gov/opub/mlr/2001/09/art2full.pdf

Walker, M. (2002). Breastfeeding after breast augmentation. *Journal of Human Lactation*, *19*, 70–71.

World Health Organization. (1981). *International code of marketing of breast-milk substitutes*. Geneva, Switzerland: Author.

Yalom, M. (1997). *A history of the breast*. New York, NY: Ballantine.

# Special Section on Postpartum Depression and Maternal Nutrition

*Rachelle Lessen, MS, RD, IBCLC*



## Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Discuss the importance on healthy nutrition on fetal development.

During fetal development, the growth of the brain depends on the accumulation of large amounts of omega-3 fatty acids. Docosahexaenoic acid (DHA) and arachidonic acid (AA) are incorporated into the neural cell membranes during the period of rapid fetal brain development in the third trimester of gestation. A portion of DHA can be endogenously synthesized by the fetus or newborn from its precursor alpha-linolenic acid; however, it is likely that most is provided directly from the mother through the placenta or in breast milk. Although the needs of the growing fetus and infant have been extensively studied, less is known about the needs of the mother, who is the primary source of DHA for her fetus or breastfed child. Makrides and Gibson (2000) reported that there does not appear to be a detectable reduction of omega-3 fatty acids during pregnancy, but there is a clear decrease of approximately 30% in the postpartum period. This decline occurs gradually from birth to 6 weeks and persists until 12 weeks or beyond. The decrease is largely independent of whether or not the mother is breastfeeding, suggesting that lactation is not contributing to this postpartum depletion. This decline is preventable and reversible with DHA supplementation.

Literature reports that an Adequate Intake (AI) of omega-3 fatty acids is associated with major depression and other affective disorders, and there are reports of decreased levels of omega-3 fatty acids in depressed patients. DHA supplementation has been reported to be effective in treatment of bipolar disorder and schizophrenia. DHA is thought to modulate synaptic function directly through its effect

on membrane structure. Because of the evidence supporting a relationship between DHA and brain function and the knowledge that DHA levels decrease in late pregnancy and lactation, researchers have proposed a relationship between DHA and postpartum depression. Some evidence exists that eicosapentaenoic acid (EPA) may have a more positive influence on treating depression than DHA does (Martins, 2009).

Seafood is a major source of DHA, and studies have shown that women who regularly consume fish have higher levels of DHA in their breast milk than do women who rarely or never eat fish. Also reported is an association between increased seafood consumption and decreased depression. Hibbeln (2002) proposed that the DHA content of mother's milk and seafood consumption would predict the prevalence rates of postpartum depression. Hibbeln hypothesized that because seafood consumption protects women from omega-3 fatty acid depletion during pregnancy, rates of postpartum depression would be lower in countries with greater rates of seafood consumption. Because the DHA content of breast milk serves as a marker of maternal DHA status postpartum, Hibbeln also hypothesized that higher concentrations of DHA in breast milk would predict lower rates of postpartum depression. The findings did indeed support the conclusion that lower DHA content in mothers' milk and lower seafood consumption were both associated with higher rates of postpartum depression. Because data on confounding variables were not available for all countries in the study, it could not be proved that higher levels of DHA caused a lower prevalence of rates of postpartum depression.

The Mothers, Omega-3 and Mental Health Study will enroll pregnant women at risk for depression in a randomized controlled trial to assess the effects of two different fish oil supplements, one high in EPA and one high in DHA, compared to placebo on the relative effectiveness of preventing depressive symptoms during pregnancy and up to 6 weeks postpartum (Mozurkewich et al., 2011).

Llorente and colleagues (2003) found that DHA supplementation of 200 mg/day for 4 months after delivery prevented a decrease in plasma DHA that is often seen in postpartum women. In a randomly assigned, double-masked, interventional study, they found that the mothers who received DHA supplementation increased their DHA levels by 8% compared with the placebo group, which had a 31% decrease in DHA. After 4 months of supplementation, the supplemented group had a 50% higher DHA level than did the unsupplemented group. However, these changes were not found to be associated with rates of depression. Repeated measurements of depression at 3 weeks, 2 months, and 4 months showed no difference between the groups at any time. Rates of depression were equally low in both groups. The authors conceded that perhaps a higher dose of DHA, a combination of DHA and AA, or initiating supplementation during pregnancy may have more beneficial effects on postpartum depression.

## References


Hibbeln, J. R. (2002). Seafood consumption, the DHA content of mothers' milk and prevalence rates of postpartum depression: A cross-national, ecological analysis. *Journal of Affective Disorders, 69*, 15–29.

Llorente, A., Jensen, C. L., Voigt, R. G., Fraley, J. K., Berretta, M. C., & Heird, W. C. (2003). Effect of maternal docosahexaenoic acid supplementation on postpartum depression and information processing. *American Journal of Obstetrics and Gynecology, 188*, 1348–1353.

Makrides, M., & Gibson, R. (2000). Long-chain polyunsaturated fatty acid requirements during pregnancy and lactation. *American Journal of Clinical Nutrition, 71*, 307S–311S.

Martins, J. G. (2009). EPA but not DHA appears to be responsible for the efficacy of omega-3 long chain polyunsaturated fatty acid supplementation in depression: Evidence from a meta-analysis of randomized controlled trials. *Journal of the American College of Nutrition*, *28*, 525–542.

Mozurkewich, E., Chilimigras, J., Klemens, C., Keeton, K., Allbaugh, L., Hamilton, S., … Vahratian A. (2011). The Mothers, Omega-3 and Mental Health study. *BMC Pregnancy and Childbirth, 22*, 46.

CHAPTER 3

# Normal Nutrition for Toddler Through School-Aged Children and the Role of Parents in Promoting Healthy Nutrition in Early Childhood

*Jennifer Sabo, RD, LDN, CNSD, and*
*Barbara Robinson, MPH, RD, CNSC, LDN*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Understand the different nutritional factors that may affect growth and development in a toddler.
2. Understand the use of vitamins and minerals in a toddler and how to provide them naturally.
3. Identify the differences in portion sizes that are appropriate for toddlers versus adults.
4. Understand the importance of "mealtime" with toddlers.
5. Identify reasons for excess weight gain in toddlers and how this can be prevented.
6. Use the information obtained in the chapter to assess three problems common to toddlers and determine the solution.
7. Identify the antecedents to eating behaviors that occur in the early childhood.
8. Discuss components of the mealtime feeding environment (structure, exposure, modeling, and repetition).

After infancy, nutrition continues to be vital to the growth and development of children. As the child becomes less dependent on breast milk and/or formula, a more comprehensive form of nutrition becomes essential to provide an appropriate variety of foods. This ensures that the growing child is obtaining the necessary macro- and micronutrients for growth. This goal is made difficult by the emerging independence of the child. Strategies designed to incorporate, understand, and compensate for particular behaviors centered on mealtime and eating may be just as vital as providing healthy choices.

# *Normal Nutrition for Toddler Through School-Aged Children*

*Jennifer Sabo, RD, LDN, CNSD*

## Growth Expectations

Growth is measured and plotted on standard Centers for Disease Control and Prevention (CDC) growth charts based on age and sex. Head circumference and weight for height is measured and plotted until 36 months of age. After age 2 years, body mass index (BMI) is used to assess appropriate weight for height (National Institutes of Health, n.d.). The use of BMI after the age of 2 years is appropriate because of a correlation between height and adiposity before age 12 years (Freedman et al., 2004).

Growth rates may vary considerably for each individual child. This is thought to be associated with a variety of factors, including parents' growth history and patterns. It is essential for clinicians to note that approximately 25% of normal infants and toddlers in the first 2 years of life drop to a lower growth percentile and subsequently remain on this new growth track (Krugman & Dubowitz, 2003). **Table 3.1** and **Table 3.2** demonstrate average weight and height growth, respectively, for the 1st through 10th years of life. It is helpful for healthcare professionals to be aware of these growth trends to assess each child's growth.

## Energy and Nutrient Needs

As growth rates differ for each child, so do their energy needs. The energy requirements of toddlers and children vary greatly based on differences in both growth rate and level of activity (Zlotkin, 1996). As demonstrated in Tables 3.1 and 3.2, the

**TABLE 3.1 Weight Growth**

| Age (yr) | Average Daily Growth (g/day) |
|---|---|
| 1–3 | 4–10 |
| 4–6 | 5–8 |
| 7–10 | 5–12 |

Data from Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: Methods and development. National Center for Health Statistics. *Vital Health Stat* 11(246). 2002.

**TABLE 3.2 Height Growth**

| Age (yr) | Average Daily Growth (cm/month) |
|---|---|
| 1–3 | 0.7–1.1 |
| 4–6 | 0.5–0.8 |
| 7–10 | 0.4–0.6 |

Data from Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: Methods and development. National Center for Health Statistics. *Vital Health Stat* 11(246). 2002.

growth rate for ages 1 to 3 years and 7 to 10 years are more rapid, thus necessitating greater energy needs. The chronologic age and the current stage of **development** of the child also relate to and influence energy needs. Current energy need recommendations from the Food and Agriculture Organization, World Health Organization, and United Nations University Expert Consultation (1985) for growing children ages 1 to 10 years are based on the actual observed intakes of healthy children and can aid clinicians in establishing norms.

**development** A process in which something passes by degrees to a different more advanced stage.

Current weight and height, as well as trends in growth velocity, can also be used to assess a particular child's needs. It is essential to note that throughout childhood there appears to be no difference in energy requirements for boys and girls. A study by Butte and associates (2000) used the total energy expenditure and energy deposition data of a group of healthy children to demonstrate that current recommendations for energy intake in the first 2 years of life may exceed modern energy expenditures and perhaps need to be revised.

**TABLE 3.3 Protein Requirements**

| Age (yr) | Dietary Reference Intake for Protein (g/day) |
|---|---|
| 1–3 | 13 |
| 4–8 | 19 |
| 9–13 | 34 |

Data from Institute of Medicine. (2005). *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)*. Washington, DC: The National Academies Press.

**Learning Point** As a general rule, energy requirements are designed to promote an optimal rate of growth and adequate body composition (Butte et al., 2000). However, the overall energy requirements can be adjusted based on need for weight loss or gain, weight maintenance, or catch-up growth.

### Protein

Protein is the primary component in many body tissues. Proteins build, maintain, and restore tissues in the body, such as muscles and organs. As a child grows and develops, protein is a crucial nutrient needed to provide optimal growth. Current recommendations state that protein intake should comprise approximately 10% to 20% of the child's daily intake. This recommendation is designed to ensure that enough energy is provided to the body from all nutrients so protein is spared for growth and development of tissues. **Table 3.3** provides the current Dietary Reference Intake for protein for growing toddlers and children.

### Fat

Until age 3 years, dietary fat plays a role in brain development. Fat comprises approximately 60% of the central and peripheral nervous systems that essentially control, regulate, and integrate every body system; thus, it is essential that growing toddlers obtain adequate fat from their diet. Furthermore, the fat content of the diet is known to be the crucial element in providing satiety. Therefore, low-fat meals or snacks for children can lead to hunger and subsequent overeating between meals. After infancy most children are able to meet their daily calorie and nutrient requirements for growth with a diet consisting of 30% of total calories from fat (American Academy of

**Learning Point** Protein deficiencies are uncommon in the United States because most children exceed the daily recommended intake for protein. Children at risk for protein deficiency include those from low-income homes, those with multiple diet restrictions because of food allergies, and those who are provided a strict vegetarian diet that excludes most or all animal products (referred to as a vegan diet).

Pediatrics [AAP] Committee on Nutrition, 1992; Butte, 2000). If excess weight gain is a concern, often only minor changes in dietary choices are needed to help keep fat intake at or below 30% of daily calories to promote optimal growth (Fisher, Van Horn, & McGill, 1997).

It should be noted that all fats are not equal in their benefits to one's health. Most fat intake should be derived from poly- and monounsaturated fat, such as fish, most nuts, and vegetable oils (American Heart Association [AHA], 2013). These fats help to keep total cholesterol low and "good" cholesterol, or **high-density lipoproteins**, high. Foods high in saturated fat, such as butter, cheese, and beef, should be offered in moderation because these can contribute to high total cholesterol and high **low-density lipoproteins**. Though hypercholesterolemia has not traditionally been a concern for this age group, recent trends in obesity coincide with a rise in cholesterol levels, therefore mandating that clinicians become more cognizant of fat intake.

**high-density lipoproteins** Blood constituents involved in the transport of cholesterol and associated with a decreased risk of heart attack.

**low-density lipoproteins** Lipoproteins that transport cholesterol in the blood, composed of a moderate amount of protein and a large amount of cholesterol. High levels are associated with increased risk of heart disease.

## Vitamins and Minerals

### Calcium

Calcium is the principal mineral required by the body for the process of bone mineralization. Toddlers and young children have an increased need for calcium to promote the rapid bone growth and skeletal development that takes place during these early years of life. Despite the essential nature of this mineral, studies demonstrate only about 50% of children aged 1 to 5 years meet the Dietary Reference Intake for calcium (Dennison, 1996). **Table 3.4** provides the current Dietary Reference Intake for calcium in growing children (IOM Food and Nutrition Board, 2011).

**TABLE 3.4 Calcium Requirements**

| Age (yr) | Dietary Reference Intake for Calcium (mg/day) |
|---|---|
| 1–3 | 500 |
| 4–8 | 800 |
| 9–13 | 1,100 |

Data from Institute of Medicine. (2011). *Dietary Reference Intakes for Calcium and Vitamin D.* Washington, DC: The National Academies Press.

Storey, Forchee, and Anderson (2004) demonstrated that soft drink and juice consumption begins to increase at 4 to 8 years of age, often replacing milk at meals and snacks. This study found that intake of milk and milk products decreased more significantly with age for girls as compared with boys. These authors suggest discouraging and/or restricting juice and soft drink intake to avoid the displacement of milk with these largely unhealthy beverages (Storey et al., 2004).

If it is determined that the recommended daily calcium intake is not being met, by using diet histories and food frequencies, health professionals should consider recommending a daily calcium supplement. Many studies have shown that males demonstrate a superior result in building peak bone mass with calcium supplementation (Carter et al., 2001). It is essential to recognize that milk, milk products, and some milk substitutes contain additional necessary nutrients for health, including protein and vitamin D, which continues to make these a healthy method of meeting calcium needs. Thus, healthcare professionals should continue to find means of increasing or maintaining intake of milk and milk products (Storey et al., 2004). Because vitamin D is needed for the best utilization of calcium, many calcium supplements are now including vitamin D to aid in the efficiency of the product. As demonstrated by Johnston and associates (1992), daily calcium supplementation lasting longer than 12 months may result in great gains in bone mass as compared with control groups.

**CRITICAL Thinking**

Though controversial, flavored milk can be a good source of calcium and may increase compliance in young children. It may be helpful to choose lower fat milk and add flavoring with sugar-free syrups as a way to keep fat and added sugar intake low for toddlers older than age 2 years and still receive the benefits of calcium.

Recent studies indicate that calcium intake of about 800 milligrams (mg) per day is associated with adequate bone mineralization in preadolescent children (AAP, 1999). The importance of providing adequate calcium to growing children is becoming more apparent. Storey and coworkers (2004) found that adequate calcium intake in preadolescent and adolescent girls is necessary to help decrease the incidence of osteoporosis in adulthood. Therefore, good prevention efforts for osteoporosis should focus on "young people in their growing years" (Carter et al., 2001).

Dairy products are typically the main source of calcium in the diet, including milk, yogurt, cheese, and ice cream. However, more recently and perhaps reflecting trends of inadequate calcium intake, many foods are being fortified with calcium, such as waffles, juice, and cereals. Soy, almond, and rice milk are also usually fortified with calcium and vitamin D. Calcium is often represented as a percent daily value on food labels, using 1,000 mg/day as 100% daily value (AAP, 1999). These fortified foods should be assessed for overall nutritional quality and not consumed solely for calcium supplementation. **Table 3.5** provides ways to increase calcium in a child's diet.

### Vitamin D

Vitamin D is available to humans through the photochemical action of sunlight or ultraviolet light on 7-dehydrocholesterol in skin and through dietary sources such as fish oils, fatty fish, and foods fortified with vitamin D, including cow's milk and infant or supplemental formulas (Holick, Shils, Olsen, Shike, & Ross, 1999). The amount of vitamin D synthesized through sunlight exposure is affected by time spent outside, the amount of skin exposed, air pollution, cloud cover, time of day, latitude, time of year, and skin pigmentation (Tomashek et al., 2001). The use of sunscreen also limits the synthesis of vitamin D.

Specker, Valanis, Hertzberg, Edwards, and Tsang (1985) estimated that white infants require approximately 30 minutes of sunlight per week to obtain adequate vitamin D if wearing only a diaper but require 2 hours per week if fully clothed without a hat. The melanin in our skin decreases the amount of vitamin D synthesized from sunlight. Therefore, children with darker skin need to spend more time outside than do those with lighter skin to obtain sufficient vitamin D (Weisberg, Scanlon, Li, & Cogswell, 2004).

Adequate vitamin D intake is important for optimal calcium absorption. Dietary products such as yogurts and orange juice are now fortified with vitamin D. As toddlers are advanced off of infant formula and onto table foods, parents should be educated to offer a diet adequate in both calcium and vitamin D (Weisberg et al., 2004). The AAP (2008) now recommends 400 IU per day of vitamin D for all children, beginning at birth and through to adulthood. National data

**TABLE 3.5** **Easy Ways to Increase Calcium in the Diet**

- Drink milk or a fortified milk substitute. Two 8-oz glasses of milk each day can provide more than 600 mg of calcium. Lactose-reduced milk is a good source of calcium if regular milk is not tolerated.
- Use heated milk in place of hot water to make foods, such as oatmeal or hot chocolate.
- Add cheese to sandwiches, casseroles, meat loaf, salads, and snacks.
- Add broccoli and cheese to a baked potato.
- Choose desserts made with milk such as pudding, custard, frozen yogurt, and ice cream.
- Substitute milk and yogurt in recipes instead of cream or sour cream.
- Add dried powdered milk or evaporated milk when preparing soups, mashed potatoes, sauces, and hot cereals.
- Drink a glass of calcium-fortified juice, such as orange juice.
- Add almonds to muffin and bread recipes.
- Remember to read labels for percentage of calcium. If it has greater than 30% of your calcium requirement for the day, it would be a high-calcium choice. Many foods are fortified with calcium, such as orange juice, rice, cereals, waffles, and pasta.
- Choosing one high-calcium food at each meal will help you to meet your goal every day!

Low-fat products contain the same amount of calcium as whole milk products.

as to the prevalence of hypovitaminosis D among children are not yet available (Weisberg et al., 2004). Therefore, more extensive study is needed to determine the extent of vitamin D deficiency in the United States. Low levels of vitamin D in childhood are now being linked to a variety of diseases, including type 1 diabetes, multiple sclerosis, and a variety of cancers (AAP, 2008).

### Iron

Daily requirements for iron intake are based on age and iron stores. During periods of rapid growth, the body's need for iron increases, as shown in **Table 3.6** (IOM Food and Nutrition Board, 2011).

Iron can be classified as being derived from heme or nonheme sources. Heme sources include animal meats and products, such as beef and chicken, whereas nonheme sources include fortified grains, fruits, and vegetables (IOM Food and Nutrition Board, 2011). Though iron can be obtained from either source, absorption of this essential nutrient is found to be higher from heme sources than from nonheme. Of note, consuming foods that include vitamin C while eating an iron-containing food can promote better absorption of iron in the body. Conversely, taking antacids during the meal may inhibit iron absorption. **Table 3.7** provides ways to increase iron in the diet.

**anemia** A condition is which your blood has a lower-than-normal red blood cell (RBC) count.

Iron-deficiency **anemia** is the most common nutritional deficiency in the world and remains relatively common among at-risk groups in the United States (Schneider et al., 2005). Risk factors for iron deficiency include low household income, lack of consistent medical care, poor diet quality, and parents with minimal education. Identification of deficiency is crucial because side effects for infants and children with iron deficiency may include impaired neurodevelopment, leading to decreased attention span and lower scores on standardized tests (Grantham-McGregor & Ani, 2001).

Deficiency often occurs as a secondary development to insufficient dietary iron intake. It also should be noted that increased calcium intake may lead to anemia. This is because calcium and iron compete for absorption at the same receptor sites within the body. Therefore, drinking large amounts of high-calcium milk is a common cause of iron deficiency in children. Refusal of meats is also a common cause of low iron intake in developing children. The increased availability of iron-fortified foods and formulas has helped to decrease the incidence of iron deficiency.

**TABLE 3.6** Iron Requirements

| Age (yr) | Dietary Reference Intake for Iron (mg/day) |
|---|---|
| 1–3 | 7 |
| 4–8 | 10 |
| 9–13 | 8 |

Data from Institute of Medicine. (2001). *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.* Washington, DC: The National Academies Press.

**TABLE 3.7** Increasing Iron in the Diet

- Include a protein source at meals, such as fish, beef, or poultry.
- Look for iron-enriched or fortified grains, such as cereals, breads, and pasta.
- Offer oatmeal or cream of wheat at breakfast.
- Add foods high in vitamin C to the meal, including citrus fruit, melon, and dark green leafy vegetables, for improved iron absorption.
- Add puréed meats to pasta sauce or casseroles for toddlers with difficulty advancing to chewing meats.
- Encourage children with advanced chewing skills to eat the skin of the baked potato for added dietary iron.

An objective of Healthy People 2010—and also of the 2020 goals—was to reduce iron deficiency in 1- to 2-year-olds by 5% and in 3- to 4-year-olds by 1% (U.S. Department of Health and Human Services, 2000). The goal to reduce anemia and iron deficiency in high-risk populations, such as toddlers, has been addressed through programs such as the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) of the U.S. Department of Agriculture by enrolling families at the highest risk for anemia and iron deficiency and providing them with vouchers for iron-fortified formulas and cereals. WIC also screens children for low hemoglobin with the goal of identifying at-risk children, educating families, and reducing the incidence of anemia (Schneider et al., 2005).

### Vitamin Supplements

After infancy supplemental vitamin use decreases, but it is still relatively common. Typically, a general

multivitamin is not routinely recommended for toddlers and young children unless the diet history demonstrates the child's intake to be inadequate.

Supplementation is recommended for children without the means to eat a balanced diet and for those who may lose necessary vitamins through chronic medical conditions or treatments, including dialysis, cystic fibrosis, gastrointestinal conditions, and food allergies. Megadoses of vitamins and minerals should be discouraged unless prescribed by a doctor for a specific medical condition.

Many everyday foods are being fortified with multiple vitamins and minerals; therefore, vitamin and mineral deficiencies are rare in the United States (Dietz & Stern, 1999). Consuming the recommended daily servings of the many food groups typically provides adequate nutrients. A diet evaluation by a healthcare professional is recommended for those who are questioning their child's need for multivitamin intake. Many pediatricians believe minimal risk is involved if a child is given the daily standard multiple vitamin. However, although vitamin and mineral toxicities are rare, possible excess intakes of these nutrients resulting from supplements and high intake of fortified foods should be considered (Briefel, Hanson, Fox, Novak, & Ziegler, 2006).

**Learning Point** Many vitamins often look and taste like candy; therefore, special care should be taken to keep them out of reach of children and under no circumstances should they be referred to as a treat by parents or caregivers.

If a child has an inadequate diet, simply providing a daily vitamin only addresses the problem in the short term. Working on improving the diet is the best way to ensure that a child is receiving optimal nutrition. Healthcare professionals should stress the importance of whole foods rather than individual nutrients (Fox, Reidy, Novak, & Ziegler, 2006) to ensure that the diet is complete with all nutrients, including fiber.

### Fluoride

Fluoride has been demonstrated to promote tooth formation and also to inhibit the progression of dental caries (IOM Food and Nutrition Board, 2000/2005). However, children who begin to use fluoride toothpaste before age 2 years are at higher risk for enamel **fluorosis**. This is because of a poorly controlled swallowing reflex that leads to increased ingestion of fluoride. To avoid excess ingestion, child-strength toothpaste (which contains a decreased quantity of fluoride) may be used in children aged 2 to 6 years. After age 6 years, regular fluoridated toothpaste is approved in children (Centers for Disease Control and Prevention [CDC], 2001).

**fluorosis** An abnormal condition caused by excessive intake of fluorides, characterized in children by discoloration and pitting (mottling) of the teeth.

Deficiencies in fluoride are not common but may occur in areas with nonfluoridated water or in children who receive nonfluoridated bottled water. Fluoride supplementation may be prescribed by a healthcare professional for infants older than age 6 months who are at risk for dental caries and primarily drink water with minimal fluoride content (CDC, 2001).

## Water

Water helps the body to maintain **homeostasis**, allows for the transport of nutrients into cells, and also functions in the removal of the waste products of metabolism (IOM Food and Nutrition Board, 2001/2005). Therefore, water is a very important, though often overlooked, component of the daily diet. All beverages contain water, as do high-moisture foods, such as fruit, soups, and ice cream. Water can be offered to the growing child for hydration, but care should be taken not to replace adequate milk or formula intake to prevent displacing vital nutrients in the diet.

**homeostasis** The tendency of the body to seek and maintain a condition of balance or internal stability.

## Whole Milk

The use of whole milk should typically stop after age 2 years, unless otherwise directed by a healthcare professional. To promote adherence, families should take care to drink skim or 1% milk together.

## Foods at 1 Year

After 1 year of age, toddlers begin to eat more like their caregivers. An important aspect to feeding toddlers is the knowledge that portion sizes should be kept appropriate for their age. Toddlers often eat six small meals each day versus three larger meals. Dietz and Stern (1999) suggested that

toddler serving sizes be one-fourth to one-half that of adults. Some examples follow:

- *Grains:* Bread, ¼ to ½ slice; cereal, rice, or pasta, cooked, 4 tablespoons; dry cereal, ¼ cup
- *Cooked vegetables:* 1 tablespoon per year of age
- *Fruit:* Cooked or canned, ½ cup; fresh, one-half piece; 100% juice, ¼ to ½ cup
- *Dairy:* Milk, ½ cup; cheese, ½ ounce; yogurt, ⅓ cup
- *Protein:* Chicken, turkey, beef, or fish, 1 oz; ground meat, 2 tablespoons

## Mealtime with Toddlers

As toddlers continue to advance in their feeding skills, it is crucial to provide a healthy feeding and eating environment. Structure at meals can have a significant influence on a child's eating patterns (Patrick & Nicklas, 2005). Suggestions to provide such a structured environment include the complete absence of television and other distractions of a similar ilk. As often as possible the family should sit at the dining room or kitchen table together and enjoy the meal as a unit. In addition to providing structure and promoting family bonding, eating together as a family has been shown to be associated with increased intakes of fruits and vegetables at a meal (Patrick & Nicklas, 2005).

As with most behaviors, the child's stage of development can influence food choices. Toddlers may prefer finger foods that encourage them to demonstrate their new independence. **Table 3.8** provides examples of finger foods that are typically safe to offer. To avoid conflict at meals, parents should be educated that the child may also decide mealtime is over before a large amount of food is consumed because of their comparatively small stomach size. The American Hospital Association (AHA) recommends that infants and young children should not be forced to finish a meal because calorie intake is likely to vary at each mealtime (AHA, n.d.). In fact, Fisher and Birch (1999) found that children forced to eat everything on their plates became less sensitive to their body's signs of satiety.

## Introducing New Foods

As toddlers and young children develop new feeding skills, there is potential for greater dietary variety. To the frustration of many caregivers, children often initially refuse a new food simply because it is unfamiliar. However, multiple exposures to a new food have been found to ultimately achieve food acceptance. It has been found that up to 10 exposures to the new food promote clear changes in acceptance of the food (Sullivan & Birch, 1994). However, Carruth and coworkers (1998) found that most caregivers tried a new food an average of 2.5 times before deciding the child disliked that particular food, falling well below the recommended number of experiences needed to truly determine an actual dislike for the food. This demonstrates that parents and caregivers can greatly benefit from consistent education regarding presentation of new foods.

It is important to introduce one food at a time to avoid confusion or overwhelming the child. Consistency with each new food, offering at least once per day, helps the child to develop a familiarity. Offering vegetables more frequently throughout the day has been shown to help increase a child's daily intake (Dennison, Rockwell, & Baker, 1998). Similar-appearing foods may be more readily received once one type of food has previously been accepted. Conversely, limited exposure to a variety of foods could lead to rejection of other products that have a similar visual appearance to the initially rejected food (Carruth et al., 1998). Many parents state their child does not like vegetables, but vegetables are rarely available in the home for the child to become familiar with. Therefore, not only exposure but also the *opportunity* to taste a food increases food acceptance (Birch & Fisher, 1998).

Caregivers should be educated on providing a healthy variety of new foods to their child. Foods without overall nutritional value should not be introduced merely to provide calories (AHA, n.d.). **Table 3.9** provides tips on promoting increased fruit and vegetable intake.

**TABLE 3.8 Finger Foods**

- Foods should be easily dissolved.
- Serve cheese sticks, rice puffs, wagon wheels, cheese puffs, meat sticks, soft vegetable pieces, peeled apple or pear sticks, and soft fruit pieces.

Types of foods offered *must* match developmental abilities.

**TABLE 3.9** How to Promote Increased Fruit and Vegetable Intake

- Add fruit purées into mixes, such as pancakes, waffles, and muffins. Apples, pears, bananas, and blueberries are great fruits to try. Using purées avoids the child finding chunks of the fruit, which may prevent the child from eating the food. Sometimes starting with a very small amount of fruit and increasing the amount as the child becomes familiar with flavor is the best way for more sensitive children.
- Puréed fruits also can be mixed into yogurt, milk shakes, and pudding to add flavor.
- Adding dried fruits to cereal or trail mixes is a good way to increase fruit intake. Serving sizes are smaller for dried fruit, so the child does not have to eat as much. Note: This should be used for children older than age 3 years to prevent a choking risk.
- Mix vegetable purées into soups and sauces, such as spaghetti sauce or chicken noodle soup. Carrots, green beans, and sweet potatoes are good vegetables to start with. You also can mix these purées into homemade meat loaf, meatballs, or hamburgers.
- Bring the child along to the store to select a new fruit or vegetable he or she would like to try. Have the child help prepare the new food. If the child feels more involved with the selection of the food, he or she is more likely to try it.

## Planning Children's Meals

When planning meals for the growing toddler or child it is important to keep in mind the different food groups and nutrient needs. It can be difficult to meet daily recommendations for fruit and vegetable intake if one only offers these foods at the three main meals. Low intake of fruits and vegetables is correlated with suboptimal intake of many nutrients, including fiber and vitamins A and C, and also appears to be linked with higher fat intakes (Dennison et al., 1998). Fruits and vegetables are a good choice at snack time, thereby helping to meet optimal intake.

It is important to plan balanced meals, demonstrating healthy eating early while the child is developing feeding behaviors. Starting a heart healthy diet before reaching adulthood can help to reduce the prevalence of obesity (Fisher et al., 1997).

It is essential for healthcare providers to be aware that preparing and providing healthy meals can be expensive. The high cost of fruits and vegetables is often reported as the main reason a parent or caregiver does not frequently purchase these foods (Dennison et al., 1998). Parents should be educated on creative ways to provide fruits and vegetables. For example, shopping for produce at local farmers markets provides a more affordable opportunity. Furthermore, canned and frozen fruits and vegetables are often less expensive and remain an excellent source of nutrients. It is important, however, to let caregivers know to purchase fruit canned in its own juice and vegetables without added salt to avoid providing extra sugar or sodium to the child.

Children tend to eat more of foods they are familiar with, so it is important for healthy foods to always be available in the home. Patrick and Nicklas (2005) reported that "children are more likely to eat foods that are available and easily accessible." This should always be considered when planning and shopping for a meal.

## Hunger and Behavior

Children gain knowledge of dietary behaviors from watching others (Hayman, 2003; Patrick & Nicklas, 2005). As children age they begin to spend more time outside of the home, acquiring feeding behaviors from those around them. By age 3 or 4 years, eating becomes influenced by environmental cues more than the previous "deprivation-driven response" during infancy (Patrick & Nicklas, 2005).

However, regardless of behaviors developed the child should not control meal planning. Satter suggested caregivers develop a "division of labor" between caregivers and the child. In this model, the caregiver's role is to provide healthy foods that are accessible. It is the child's responsibility to decide when to eat and how much food to eat at a particular meal (Satter, 1986, 1996).

Food behaviors can also be related to the availability of certain foods and how easy they are to consume for the developing child. Baranowski, Cullen, and Baranowski (1999) found that more often children tend to choose foods that are in an accessible location and also of an appropriate size for their developmental capacity. Fruits and vegetables that are cut into sticks or bite-size pieces are more likely to be chosen than are whole fruits and vegetables simply because the child is able to handle them independently.

Growing toddlers and children should typically not be restricted at meals. Restriction of foods that are high in fat and sugar often lead to the child

"fixating on the forbidden food" and consuming more of these foods outside of the home or when full (Fisher & Birch, 2000). If seconds are requested it is beneficial to develop a habit of offering an additional serving of vegetables or fruits, though not forcing the child to take what or *only* what is offered.

## Picky Eating

Picky eating can be common in toddlers and may continue throughout childhood. Often, picky eating may represent an attempt at acquiring independence rather than a declaration of actual likes and dislikes. A caregiver may try to accommodate a picky eater or to develop techniques that may help increase amount and quality of foods eaten. However, using rewards, prodding, or punishment to encourage eating may only enforce the "picky eater phenomenon" (Pelcaht & Pliner, 1986). Caregivers should continue to provide healthy food options and not make special foods or entire meals for the picky child.

Working with a child who is exhibiting picky behaviors can certainly be challenging. Parents and caregivers may need information and strategies from their healthcare professional to increase the number of foods acceptable to their toddlers and children and to develop a sound eating plan (Carruth et al., 1998).

## Grazing

Growing toddlers and children may not be hungry on a schedule, and they often do not want to stop for a meal or snack that can interrupt their playtime. If a child asks for food or liquids more frequently than every 2 to 3 hours, it is important to encourage him or her to wait until the next meal or snack time to eat.

Grazing may produce a constant feeling of fullness and cause the child to avoid eating an appropriate amount at mealtime, therefore not expanding the stomach size. Constant snacking and early satiety may result in the child not receiving adequate calories each day, thereby ultimately leading to failure to thrive (Krugman & Dubowitz, 2003). It is important to have the child sit at a table away from distractions while eating to help him or her better understand the difference between mealtime and playtime.

## Failure to Thrive

Failure to thrive is defined by "inadequate physical growth diagnosed by observation of growth over time using a standard growth chart" (Krugman & Dubowitz, 2003). This failure to maintain adequate growth can be caused by a multitude of factors. Most often, failure to thrive is classified as organic (inability to meet calorie needs as a result of medical conditions, malabsorption, or increased metabolism with specific disease states) or inorganic (food shortage, incorrect mixing of formula, or neglect) (Krugman & Dubowitz, 2003).

Healthcare professionals should be aware of any factors that may contribute to poor growth. Taking a full diet history, including formula-mixing techniques, can help assess whether adequate calories are in fact being provided to the child. If all possible inorganic etiologies are ruled out, a medical workup for organic failure to thrive should be undertaken.

## Lactose Intolerance

According to Vesa, Marteau, and Korpela (2000), lactose is the "most important source of energy during the first year of human life." Intolerance is uncommon in healthy infants but can present as a result of a gastrointestinal illness, ultimately resolving with improvement of the illness. Symptoms of intolerance are similar to other gastrointestinal dysfunctions (Vesa et al., 2000), causing stomach pain, flatulence, and loose stools.

Hypolactasia can present early in childhood in African American and Asian children but seems to appear in late childhood or adolescence for white children (Scrimshaw & Murray, 1988). Many lactose maldigesters are able to tolerate small amounts of lactose without discomfort (Vesa et al., 2000). For example, children with lactose intolerance can often drink a small amount of regular milk. Alternatives exist, such as solid cheeses that are low in lactose, and yogurt may also be better tolerated.

More recently, lactose-free and low-lactose milks and ice creams have become readily available. Soy milk may also be a good substitute, but caregivers should be educated that whatever the variety of substituted milk, it must be fortified with calcium

and vitamin D. Careful diagnosis is important to prevent unnecessary diet restrictions that may lead to deficiencies in the essential nutrients provided by dairy products (Vesa et al., 2000). Families should be instructed on ways to meet nutrient needs if a dairy restriction is ultimately recommended.

## Television Watching and Media Influence on Food Cravings

Many children spend more time watching television than interacting with their families, creating a change in key influences. More than 50% of food advertisements on television target children (Story & French, 2004). Messages often show "junk" foods as fun, exciting, and glamorous. Young people often have difficulty discriminating between education provided in television programs and information in commercials, therefore misinterpreting advertising as lifestyle instruction. Furthermore, because commercials are brief and to the point, they target the child's short attention span, thereby conveying an efficient and powerful message.

It is suggested that television viewing contributes to obesity by one or more of three mechanisms: displacement of physical activity, an increase in calorie consumption while watching or as a direct result of the effects of advertising, and/or reducing resting metabolism (Robinson, 2001).

The AAP (2001) proposes that children should spend no more than 2 hours per day watching television or using other electronic forms of entertainment. (The AAP recommends no television watching at all for children younger than age 2 years.) Making sure that meals and snacks are not eaten while watching television may help to reduce daily television viewing. Saelens and associates (2002) found that the frequency of meals eaten while watching television was the most important predictor of long-term increase in television viewing time among children. Creating rules about eating meals in front of the television was found to decrease the probability of the child watching television for longer than 2 hours per day (Saelens et al., 2002).

Simply restricting meals while watching television may not be an entirely adequate strategy for decreasing overall inactivity. The relationship among the family environment, television viewing, and low level of activity is complex. Restricting television viewing during mealtimes may be effective, but focusing on increasing physical activity is also a component not to be overlooked (Salmon, Timperio, Telford, Carver, & Crawford, 2005; Waller, Du, & Popkin, 2003).

In a study by Burdette and Whitaker (2005) on preschool children, it was found that television viewing and outdoor play minutes were not correlated to one another or to the child's BMI. However, in an article by Gortmaker, Must, and Sobol (1996), a dose-dependent relationship between television viewing and obesity was demonstrated. Given the conflicting evidence, more research is needed regarding any possible real-world correlation between daily television viewing, activity, and excessive weight gain.

## Learning Through Participation

As children grow and mature, involving them in the preparation of the meal may help to increase variety in the diet. A child will often taste what he or she has participated in preparing. Time spent in the kitchen can also be used to talk about the different colors of fruit and vegetables, to help the child understand the healthy reasons behind eating a wider variety of foods, and to promote family bonding. Furthermore, offering a choice of fruits or vegetables at a meal may help the child feel a modicum of control and inclusion. Such a practice may also promote better intake of the chosen food during the meal.

Interestingly, many studies have shown that it can be beneficial to allow children to be involved in portioning foods onto their own plate at meals. Orlet Fisher, Rolls, and Birch (2003) showed that allowing children to serve themselves at meals may decrease the incidence of exposure to excessive portion sizes.

## Choking Prevention

Having an adult always present whenever a child is eating constitutes the ultimate in safe eating practices. Because many children do not develop the skill of chewing with a grinding motion until about 4 years of age (Dietz & Stern, 1999), foods with a firmer texture that need adequate chewing may not be appropriate for young children. To prevent choking, foods should be cut into small bite-size pieces and children should remain sitting at all times while eating. Choking is more likely to occur if the child is

running or falling with food in the mouth. The child should be encouraged to take small bites and avoid stuffing his or her mouth full of food. **Table 3.10** provides foods that may present a choking risk.

## Snacks

Snacking among children is an important source of energy (Jahns, Siega-Riz, & Popkin, 2001). Toddlers and children have a comparatively small stomach size and cannot consume large amounts at meals; therefore, snacks provide needed servings of healthy foods. However, Jahns and associates (2001) found that children obtain an inordinate percentage of their daily calories from snacks, so much so that children at times may snack to such an extent that they are not hungry at mealtime. On the other hand, many children continue to eat normal-sized meals and simply consume excess calories during the day, leading to increased weight gain.

Instead of skipping snacks, caregivers should provide healthy snack choices. Nutritious snacks include, but are not limited to, cheese slices, carrot sticks, orange slices, yogurt, apple pieces, and peanut butter on wheat crackers. Snacks should be easy to prepare and on hand to reduce the need for quick, often unhealthy, snack choices.

Caregivers should be aware of what and how much their child is eating while watching television. Sometimes television viewing may condition a child who is not hungry to snack (Epstein, Coleman, & Myers, 1996). As stated previously, it can be helpful to prohibit meals or snacks in front of the television.

## Dental Health

It is beneficial to start tooth care at a young age. Introducing a toothbrush during the toddler years can initiate a good oral hygiene routine that continues through life (Mahan & Escott-Stump, 2000). Children often snack on sugary sticky foods that may stay in the teeth and provide an ideal environment for growth of the bacteria that cause dental caries. Also, the continued exposure of the teeth to the sugary drinks, such as juice, contributes to dental caries in children (AAP Committee on Nutrition, 2001). Teaching young children to brush their teeth after meals and snacks can help to reduce the incidence of cavities.

**TABLE 3.10** Foods That May Cause Choking

- Hot dog slices
- Carrot rings
- Whole grapes
- Nuts
- Popcorn
- Hard candies
- Large beans

## Role Models

Children learn by observing the behaviors of others and then incorporating and imitating these practices. Parents modeling healthy behaviors, accessibility to sedentary pursuits at home, sibling influences, and family television viewing habits may be important determinants of a child's activity level and healthy behaviors (Rachlin, Logue, Gibbon, & Frankel, 1986). Food choices and dietary quality in the early years of life are usually influenced by parents or other primary caregivers.

As children mature they tend to eat more meals outside the home. Therefore, it is essential to model good habits while the child is at a young, impressionable age. Modeling food behaviors is one of the most effective ways to promote increased consumption of healthy foods (Dennison et al., 1998). Parents who do not eat fruits and vegetables tend to raise children who refuse these foods as well.

Negative thoughts regarding food also can begin at a young age. Preadolescent girls are becoming more at risk for picking up dieting habits from female caregivers. Girls who diet by adolescence tend to have mothers who are often overtly encouraging or unconsciously demonstrating the means to diet through their own restrictive habits (Birch & Fisher, 2000).

### CRITICAL Thinking

Evidence suggests that more young girls are becoming concerned with gaining weight because of the influence by caregivers and the media. Can you give some examples of this? What can be done to prevent this?

## Breakfast

As children enter school there becomes less time in the morning for simple things, such as eating a healthy breakfast. It is important to develop the

habit of including breakfast in the daily morning routine. Eating breakfast has been shown to increase energy and concentration at school. It also allows for a balanced metabolism, thereby aiding in the child's ability to maintain a healthy weight. Studies have shown that skipping breakfast is related to increased BMI and risk of obesity (Cho, Dietrich, Brown, Clark, & Block, 2003).

Breakfast is an excellent time to consume a needed serving of fruit, whole grains, dairy, or protein. Many schools have a breakfast program, but it is important to be aware of what foods are being offered and how much the child is consuming during this time. At breakfast, schools provide 25% or more of the daily recommended levels of key nutrients that children need (Food and Research Action Center, n.d.), but this is often not controlled to avoid excess. Children can usually make their own food choices, so education concerning appropriate portion sizes and healthy choices can be helpful.

## Nutrition at School

Many schools participate in the government-funded lunch program. The National School Lunch program is a federally assisted meal program. Regulations create a standard for school lunches to provide one-third of the Recommended Dietary Allowances for protein, vitamins A and C, calcium, iron, and calories (U.S. Department of Agriculture, Food and Nutrition Service, 2005). Meals must meet nutritional requirements, but individual schools can decide what foods to serve and how to prepare them.

The program guarantees that every child will meet at least 33% of his or her daily needs each day. It is true that many children rely on this program for needed nutrition. However, to meet the required 33% of calories, at times meal options do not contain particularly healthy choices. Less than nutritious options such as nachos, giant hoagies, and hot dogs are common at many schools. Also, many children receive a large portion of their daily calories from snacks, which often leads to excess daily calories when combined with school lunches and fast food consumption. Lunch is often provided at a discount or may be free to students, making it unrealistic to expect these children to pack lunches containing healthier options. Therefore, it seems reasonable for schools to become more adept at offering well-balanced meals.

Peer influence also plays a role in lunchtime nutrition. Food choices are often affected by what friends at school are eating. Children may be encouraged to try a new food if others around them are eating it. On the other hand, such influence may also contribute to requesting more "junk" foods that other children bring to school.

Parents and children may benefit from education on choosing healthier options at school. If available, reviewing the monthly menu with the child can prompt discussion on which foods are the healthier choices.

## Physical Activity

Physical activity is an important part of a healthy lifestyle. It promotes muscle building, increased metabolism, and disease prevention. Among children, overweight, obesity, and low levels of physical activity have been shown to be associated with increased risk of disease later in life (Baranowski, 1992). Exercise is also a vital aspect of achieving maximal peak bone mass (AAP, 1999).

With age and increased independence, physical activity often decreases. The CDC demonstrated that 61% of 9- to 13-year-olds do not participate in organized after-school activities and 23% did not participate in any physical activity during their free time (CDC, 2003). Something as simple as parental encouragement may be all some children need to become more active. Studies have shown that parents who are supportive of their children's participation in physical activities are more likely to have an active child (Sallis, Prochaska, & Taylor, 2000).

Sedentary activities such as watching television and playing video games have become the preferred after-school pastime. Parents, caregivers, health educators, and teachers should promote enjoyable alternatives to sedentary behaviors to increase children's participation in physical activity and prevent unhealthy weight gain (Salmon et al., 2005). If the safety of the neighborhood is poor, caregivers should work to create inside activities that keep the child active and away from sedentary pursuits.

The bottom line is that physical activity should be a part of everyday life. If children are cared for outside of the home after school, parents may need to discuss with day care providers or other caregivers the stated preference that their child be active and not spend the afternoon involved in sedentary

activities. Being active as a family can help everyone to stay healthy and provides an excellent means of spending quality time together.

## Excessive Weight Gain

Obesity is becoming a more common problem with young children. According the Pediatric Nutrition Surveillance report of 2009, the U.S. prevalence of obesity in children younger than the age of 5 years is 14.7%, and 16.4% for overweight (Polhamus, Dalenius, Mackintosh, Smith, & Grummer-Strawn, 2011). The rates are higher for childhood of all ages: 33.6% of children aged 2 to 19 years are overweight and 17.1% are classified as obese (CDC, 2012). Inactivity and larger portion sizes are contributors to this multifaceted and increasingly severe problem. Studies demonstrate that increased size of food portions offered can stimulate a young child's food intake, causing excess calorie consumption at meals (Orlet Fisher et al., 2003).

Obesity prevention programs focused on diet and exercise are becoming more and more available to young children. However, Etelson, Brand, Patrick, and Shirali, (2003) reported that participation in obesity prevention programs and lifestyle changes depends on the parents' ability to recognize that their child is overweight. In their study, it was determined that no parent of a child above the 75th percentile for BMI/age was able to estimate correctly how overweight their child was. These parents universally believed their children were less overweight than actually recorded. Therefore, if a parent perceives a child to be at low risk for being overweight, the intervention is likely to be delayed.

### Juice Consumption

In the past, fruit juice was consistently recommended by pediatricians as a good source of vitamin C for children and also as an additional source of hydration (AAP Committee on Nutrition, 2001). As mentioned previously, juices high in vitamin C have been shown to aid in the body's absorption of iron. However, the high carbohydrate content of juice often leads to malabsorption in children.

Over time, juice has become the preferred beverage among many children. Children aged 1 to 12 years make up about 18% of the total population but are responsible for almost one-third of the average daily intake of juice and juice drinks (AAP Committee on Nutrition, 2001).

In toddlers and children, juice often inappropriately replaces formula, milk, and water intake and is also used as a replacement for whole fruit and vegetable servings. Fruit juice accounts for about 33% of all fruit and vegetable intake among preschoolers and 50% of fruit intake by children aged 2 to 18 years (Dennison, 1996). When juice replaces formula and milk, the child often does not receive optimal calories and nutrients needed for growth. Excessive juice consumption has been shown to contribute to inorganic failure to thrive and decreased stature in some children. Juice provides empty carbohydrate calories and decreases the child's appetite at mealtime, causing overall decreased calorie intake (Dennison, 1996; Smith & Lifshitz, 1994). Conversely, it has also been shown to be associated with high caloric intake and obesity in some studies when consumed frequently throughout the day (Dennison, 1996).

Only pasteurized 100% fruit juice is recommended for growing children and is the specific type of juice provided by WIC. Juice should be watered down for the child and should not be available to be sipped throughout the day. Juice should be limited to no more than 4 to 6 oz per day for children aged 1 to 6 years (AAP Committee on Nutrition, 2001).

The Institute of Medicine (IOM) has recommended a change in the WIC package to decrease the juice content. Also, the AAP's Top Ten Resolutions for 2005 stated that their number 2 priority is to get the message out that juice is not a healthy choice for children.

## Supplements for Increased Calories

Children who are not able to consume adequate calories each day may need a high-calorie nutritional supplement. Multiple available supplements are designed specifically for growing children. Pediasure, Nutren Jr, and Boost Kid Essentials are just a few supplements available for children ages 1 through 10 years. These supplements are often packaged in child-friendly containers and come in a variety of popular flavors. After age 10, most adult supplements, such as Ensure, Boost, and Nutren 1.0, are acceptable. It is essential to note that supplements should be used under the care of a healthcare professional.

## *Role of Parents in Promoting Healthy Nutrition in Early Childhood*

*Barbara Robinson, MPH, RD, CNSC, LDN*

Social science research contributes to our understanding of why young children consume particular foods and display specific eating behaviors. Historically, nutrition professionals have focused more on the science of nutrition in childhood rather than on why children eat what they eat. Lipsitt, Crook, and Booth (1985) commented: "Research on the psychological aspects of feeding development has lagged behind the study of nutritional requirements for physiologic sustenance … the style and quality of caretaker–infant interactions are determinative of other (non-feeding) interactions and subsequent feeding behavior, including preferences and aversions." In the past decade, more

attention has been paid to the antecedents of poor eating habits in childhood, including the influence of parental feeding choices on diet quality.

Recently, the burgeoning childhood obesity problem has prompted medical and nutritional experts to focus more on food and beverage consumption in the early years of children's lives when parents are primarily in control of children's diets and eating occasions. Additionally, time spent in day care settings and television viewing are receiving some attention as contributors to early childhood obesity (Kim & Peterson, 2008). For best outcomes, a holistic view of family patterns and effectiveness of parenting provides the best foundation for administering advice on diet and feeding. Behavioral determinants of the nutritional content of children's diets can inform efforts to improve the quality of energy and nutrient intakes of U.S. children.

In 2004, the Academy of Nutrition and Dietetics, formerly known as the American Dietetic Association, and the Gerber Products Company joined together to publish the "Start Healthy Feeding Guidelines for Infants and Toddlers" (Butte et al., 2004). These are comprehensive guidelines that advise feeding based on developmental readiness rather than on age and seek to help adult caregivers understand hunger and fullness cues in addition to nutritional requirements of children. In the *Start Healthy Feeding Guidelines*, the value of repeated exposures to healthy foods and age-appropriate physical activity is addressed (Butte et al., 2004). In 2005, the American Heart Association published a scientific statement entitled "Dietary Recommendations for Children and Adolescents" (Gidding et al., 2005). As is typical in a statement such as this, specific nutrient goals are discussed and nutritional recommendations are provided. What makes this statement unique is the significant focus on the effects of parenting in relation to the nutritional content of children's diets.

The context in which complementary foods are offered can facilitate either acceptance or rejection of new flavors. There is a window of opportunity in the normal development of infant and child food preferences. To a large extent food preferences and eating behaviors are formed in the early years of a child's life and become the foundation for lifelong eating habits. "Parents are a child's first teachers" is often quoted in discussions on child development. Adult family members not only determine the content of a young child's diet, but they and older siblings are role models. There is another dimension to food acceptance: the emotional bond between the young child and the primary feeder. The influence of the family environment, including the type of foods to which a child is exposed, continues throughout childhood, but the impact of parenting on diet is greatest in the early years. The goal for this section is to discuss the relationship between the home environment and early childhood nutrition.

## Eating Is a Learned Behavior: Acquisition of Flavor and Food Preferences

Children are not born with a preference for french fries rather than green beans. As demonstrated over and over again in behavioral research, eating is a behavior that is learned through exposure and repetition (Birch & Marlin, 1982; Skinner, Carruth, Bounds, & Zeigler, 2002; Sullivan & Birch, 1990). How early in a child's life does this learning begin to take place? What impact, if any, does exposure to flavor compounds in utero have on the developing fetus? Interestingly, researchers have found that the amnion, or the fetal environment, contains flavor compounds specific to the maternal food intake (Mennella, Jagnow, & Beauchamp, 2001; Mennella, Johnson, & Beauchamp, 1995; Schall, Marlier, & Soussignan, 2000). Furthermore, it has been observed that the flavor compounds a fetus is exposed to from the maternal diet via amniotic fluid are recognized by the infant postnatally, as reflected by newborns' facial, mouthing, and orienting responses when exposed to the same flavors orally. Mennella and associates (2001) demonstrated that intake of a specific food flavor (carrot) during either pregnancy or lactation influenced liking or disliking of carrot flavor in the mothers' infants at 5 to 6 months of age. One implication from the findings about breastfeeding and the maternal diet is that infants who are breastfed are exposed to a wide variety of flavors that reflect the maternal diet. Conversely, formula-fed infants become accustomed to only one flavor, that of the formula. This exposure to a multitude of flavors is thought to help children accept a diverse array of foods as new foods are introduced throughout the first few years of a child's life.

**CRITICAL Thinking**

In a small study conducted in 1994, Sullivan and Birch (1994) found that breastfed infants aged 4 to 6 months more readily accepted a new food and ate more of it than did formula-fed infants of the same age. It is quite possible that this and other related research represents yet another advantage of feeding infants breast milk; that is, when complementary or solid foods are introduced, infants who are breastfed might find it easier to accept new flavors than bottle-fed infants do. Do you agree? Why?

## Neonatal and Early Infant Weight Gain

The Infant Growth Study, conducted by the University of Pennsylvania and the Children's Hospital of Philadelphia, showed that infants who gained weight rapidly by 4 months of age tended to be heavier in later childhood. A higher weight at 24 months of age was observed in children who were also heavier at 3 months of age and who exhibited increased nutritive sucking and increased habitual food intake (Stettler, Zemel, & Kumanyika, 2002).

The potential for long-term consequences to very early nutritional experiences could have significant implications for guidance to pregnant women and new parents. Early childhood healthcare providers are appropriately concerned when an infant or young child is failing to grow at an acceptable rate. Newer research into the potential for negative health consequences of rapid weight gain should promote careful guidance for adequate but not excessive rate of weight gain. This has important implications, as discussed by Stettler and colleagues (2002), who found a strong association between early toddler weight gain and obesity at age 7 years.

Similar results were found in a study conducted in England on children who were infants in 1991 and 1992 (Ong et al., 2006). Parents reported intake at 4 months of age and children were weighed and measured up to 10 additional times up to the age of 5 years. Among infants who were bottle-fed or who received a combination of formula and breast milk, a higher calorie intake as early as 4 months of age predicted more weight gain in early childhood and a higher BMI at ages 1, 2, 3, and 4 years.

## Development of Food Preferences

Although there seems to be an association between consumption of certain foods during both pregnancy and breastfeeding and food preferences of the infant, there has been insufficient research to determine the degree of influence that maternal diet has on an infant's flavor preference. This remains an intriguing area for further investigation. There has been more exploration into the developmental nature of how children learn what to eat. Transitioning from the suckling and sucking that occurs with breastfeeding or bottle-feeding to tasting, chewing, and selecting food flavors and textures is a developmental process that occurs in the context of the family environment. Eating is an activity that is repeated over and over again; feeding behaviors and preferences are shaped through this repetition. Because eating behaviors are shaped through repetition, parents will have the best chance of helping their children learn to like healthy foods if they continue to offer a food despite apparent expression of dislike the first few times a child tastes a new food.

Why is it typically necessary to provide several exposures before infants will accept new foods? Although infants are curious, they are also neophobic; that is, they are cautious about trying new foods and/or might not like them until they have tried them numerous times (Addessi, Galloway, Visalberghi, & Birch, 2005; Birch & Fisher, 1995). Parents should not become discouraged and should continue to offer healthy foods in the context of a pleasant feeding experience. Confidence among parents around the issue of offering new foods can be promoted by pediatric healthcare providers. Although there is no guarantee that a child will like a given food, early exposure to a variety of healthy foods appears to be a critical factor in fostering preferences for healthy foods. Birch and Marlin (1982) showed that a higher number of food exposures were necessary in children aged 4 to 5 years than in children aged 2 years, indicating that younger children are more likely to accept new flavors. Children aged 2 years accepted new foods after 10 exposures, but it took 11 to 15 exposures to a new food before children aged 4 to 5 years established a preference for that food. Skinner and associates (2002) showed that 8-year-old children preferred the same foods that they liked at the age of 4, demonstrating the durability of previously established food preferences. These findings suggest that food preferences are more difficult to change as children become older. There might be implications for confronting the increasing incidence of childhood obesity, which

is becoming evident even in very young children. Changing established food preferences is very difficult for the family and is a challenge for healthcare providers.

The conventional approach to childhood obesity can be used to illustrate this point because the emphasis has been on identification of existing childhood overweight and obesity by measuring BMI and then intervening if the BMI is over the 85th or 95th percentile. The BMI has become the standard by which practitioners screen for excessive weight status. Because BMI is not measured until 24 months of age, early excessive weight gain would not lead to a categorization of risk. It takes more training and skill to determine risk of becoming overweight based on food intake patterns, and physicians might not make a special effort to discuss an infant's or toddler's eating habits or opportunities for physical activity with parents. Therefore, risk factors for childhood obesity might be missed (Salsberry & Reagan, 2005). In 2010, the Centers for Disease Control and Prevention (CDC) recommended the use of the World Organization (WHO) growth curves for children from birth to 24 months of age. Use of these charts can reveal more cases of overweight in young children because the charts are based on growth of breastfed infants, who tend to grow more slowly after about 3 to 6 months of age (CDC, 2000).

Setting the stage for positive eating behaviors in early childhood as a vehicle for obesity prevention has received much more attention over the past decade. Data from a descriptive survey (Feeding Infants and Toddlers Study [FITS]) conducted in 2002 by Dwyer, Sutor, and Hendricks (2004) highlight for child health professionals the benefit of shifting the focus from changing established eating habits when a weight problem is identified to promoting healthy eating behaviors to parents of all infants and toddlers whether they meet risk criteria for overweight or not.

### Feeding Infants and Toddlers Studies, 2002 and 2008

The original FITS, conducted in 2002, explored the diet composition of infants and toddlers ages 4 to 24 months (Fox, Pac, Devaney, & Janowski, 2004). FITS data show that 18% to 23% of infants and toddlers consumed no vegetables and 23% to 33% consumed no fruits during a day. And by 1 year of age, white potatoes were the most commonly consumed vegetables (often in the form of french fries), whereas the deep-yellow/orange vegetables that were consumed in infancy were no longer consumed on a regular basis. Foods high in added fats and sugars, including sweetened beverages, sweetened cereals, cookies, processed meats, cakes, and pies, provided approximately 19% of calories in toddlers. By the end of the second year, more than 11% of children were consuming carbonated sodas. Such foods and beverages can be classified

### Feeding Infants and Toddlers Study

FITS was a feeding and food intake survey conducted on a day in 2003. The study population was 3,022 children aged 4 to 24 months of age. Parents of these infants and toddlers completed both a qualifying interview and a 1-day 24-hour dietary recall and reported it over the phone to trained interviewers. The sample of children represented families at all income levels, and there was not a disproportionate representation from either low-socioeconomic groups or minority ethnic groups. A second 24-hour recall was reported for a subsample of 703 infants.

Excerpted results from the FITS data are as follows:

- *Infants, younger than 6 months old:* 17% received juice despite the American Academy of Pediatrics (AAP) recommendation to refrain from offering juice until after 6 months of age.
- *Infants 7 to 8 months old:* 46% consumed some type of sweet, sweet dessert, or sweetened beverage, and this percentage increased with age.
- *Infants 7 to 24 months old:* 18% to 33% consumed no vegetables.
- *Infants 7 to 24 months old:* 23% to 33% consumed no fruits.
- *Infants 9 to 11 months old:* One of the three most often consumed vegetables was french fries.
- *Toddlers 15 to 18 months old:* French fries were the vegetable most often consumed.
- *Energy intake:* Estimated mean energy intake exceeded the Estimated Energy Requirement (EER) by 10% for infants aged 4 to 6 months, 23% for infants aged 7 to 12 months, and 31% for toddlers aged 12 to 23 months. Although this might be partially to the result of overreporting and other confounders, an excess calorie intake in this age group is consistent with the observation of increasing percentages of excessive weight in young children.
- *Calorie consumption:* Infants and toddlers aged 9 to 14 months who continued to consume deep-yellow vegetables took in fewer calories as opposed to same-age infants who consumed popular food items such as pizza, soda, french fries, candy, and other sweets.

#### REFERENCES


Briefel, R., Reidy, K., Karwe, V., Jankowski, L., & Hendricks, K. (2004). Toddlers' transition to table foods: Impact on nutrient intakes and food patterns. *Journal of the American Dietetic Association, 104*(Suppl. 1), 38–44.

Devaney, B., Kalb, L., Briefel, R., & Zavitsky-Novak, M. S. (2004). Feeding Infants and Toddlers Study. Overview of the study design. *Journal of the American Dietetic Association, 104*(Suppl. 1), S8–S13.

Devaney, B., Zeigler, P., Pac, S., Karwe, V., & Barr, S. (2004). Nutrient intake of infants and toddlers. *Journal of the American Dietetic Association, 104*(Suppl. 1), S14–S21.

Fox, M. K., Pac, S., Devaney, B., & Janowski, L. (2004). Feeding Infants and Toddlers Study. What are infants and toddlers eating? *Journal of the American Dietetic Association, 104*(Suppl. 1), S22–S30.

**competitive foods** Those foods that are less nutritious, such as high-fat high-sugar snacks, soda, and other sweetened beverages. Easy access to these foods competes with healthier food choices for attention and consumption.

as **competitive foods**, which are those foods that are less nutritious, such as high-fat high-sugar snacks, soda, and other sweetened beverages. Easy access to these foods competes with healthier food choices for attention and consumption.

In a follow-up to the original FITS, Butte and coworkers (2008) repeated a national nutritional survey of food and liquids intake of infants and toddlers and expanded the survey to include preschoolers up to age 48 months. Similar nutritional excesses and deficits were revealed with some exceptions. The 2008 FITS survey was completed by parents or primary caregivers and included questions on foods offered, child development, and knowledge and attitudes about feeding their young children. Young children continue to consume inadequate volumes and variety of fruits and vegetables and excessive volumes of sweet beverages and juices, although the intake of juices decreased slightly from the 2002 study to the 2008 study (Briefel et al., 2010).

### Parents

Parents and/or adult caregivers play a central role in the development of early childhood feeding behaviors. The 2002 and the 2008 FITS surveys describe poor quality dietary intakes of infants, toddlers, and preschoolers that are a direct reflection of the choices that parents are making for their young children. The proliferation of commercially available high-sugar high-fat food and beverages contributes to the poor diet quality, but there are other influences on parents as well. Carruth and Skinner (2000) performed multiple interviews of mothers of children aged 2 to 54 months to identify sources and types of information and advice mothers received about feeding their children and to determine whether these sources differed as infants grew into toddlers and preschoolers. Sources of nutrition and feeding information included family, especially grandmothers; professionals; magazines; books; and videotapes. (Unfortunately, the interview process did not include questions about more insidious influences such as corporate food advertising geared toward children.) Other contemporary influences on mothers' food choices for children included family composition (e.g., single-parent families versus two-parent families) and lack of time resulting from employment outside of the home.

From 1970 to 1999, the percentage of women with young children (younger than age 6 years) who were working outside the home increased from 30% to 62%. Mothers have traditionally been more involved in preparing meals, offering foods, and feeding. Skinner and colleagues (2002) found that food preferences of 2- and 3-year-olds are related to mothers' food preferences, based on foods that were liked, disliked, or never tasted by both the mother and the child. If a mother never learned to like vegetables and fruits, for example, she is less likely to prepare and offer these foods.

### Modeling

In addition to repeatedly exposing children to a variety of healthy foods, parents have another important role to play. Parents, other adults who are with a young child regularly, and older siblings all serve as models for young children in regard to eating. The effects of modeling on a child's food preferences are additive to the effects of repeated exposure. The social learning theory as described by Bandura (1972) became the foundation on which behavioral psychology experts conducted research on how children acquire food preferences. Bandura showed that in other areas of human interaction, modeling is a process that is a strong influence on behavior.

Eating is both a social experience and a repetitive experience. Other people in a young child's environment become the models for food choice, and children develop preferences for the foods they see others eating. Harper and Sanders (1975) contrasted the degree of modeling influence on young children between mothers and strangers. There were several pertinent findings. When mothers modeled eating a certain food, children were more willing to try the new food than when strangers modeled consumption of the same food. Older children were influenced by modeling but to a lesser degree than were younger children. And finally, but perhaps most important, new foods were accepted more readily by children when the adult model ate the food with the child rather than just offering the food to the child. This has implications for the value of encouraging family meals, which have become less frequent as families become busier with work and extracurricular activities.

Birch (1980) investigated whether the effects of modeling on food preferences were sustained after the modeling event. She found that preschoolers who learned to like previously "nonpreferred

foods" through modeling exposure continued to like the new food several weeks later. This was interpreted as learned behavior rather than just the conformity that would be expected when others at the table are eating the same foods. An additional finding found in other studies is that 3-year-old children are more readily influenced in their feeding behaviors than 4-year-old children are. The greater magnitude of influence seems to indicate that earlier influences on food preferences have the greatest impact.

Research by Addessi and associates (2005) showed the importance of modeling on preference of a specific food. Three groups of 2- to 5-year-old children were offered pasta with very strong food flavors added. The added flavors were cumin, caper paste, and anchovy paste. These food flavors were considered to be new or "novel" to the children. The respective flavors of the semolina were colored yellow, green, and red. Familiar adults served as eating models. In one group, the children were served the food and the model was present but was not eating. In the second group, the children were served yellow pasta, but the adult model was eating different colored pasta. Children and the adult model in the third group were both given the same color pasta served in clear containers. When each of the trials was over, the children switched groups after a "break period" of 1 week. The pasta for the adult models was flavored to be palatable so they would eat with enthusiasm. The result of this simple experiment was very informative. Children who were given the same food (children identified it by color) as the adult model ate the most, seemed to enjoy it the most, and had the least hesitation when first trying the food. The authors of this study suggested that children might learn to like more foods if day care and preschool teachers routinely and enthusiastically ate with young children. However, a 2002 study found that preschool teachers typically eat very few of the foods that are offered to children (Hendy, 2002).

Historically, efforts at improving nutrition and activity behaviors have targeted school-aged children, and children have been regarded as the "agents of change." More recently, however, settings where young children are together have been targeted as well. Broccodile the Crocodile was a 9-month study of 307 children ages 3 to 5 (and their parents) in 18 day care/preschools to compare changes over the 9 months in BMI z-scores for appropriate weight gain in control and intervention groups. The intervention promoted healthier food intake, more activity, and less television and video viewing. The results were positive for all the intended behaviors and for no increase in the BMI z-score in the intervention group as opposed to a slight increase in the control group. Parents and daytime caregivers were considered to be the agents of change, that is, the people who intentionally changed their behaviors to achieve the desired outcome (Dennison, 2002).

### Self-Regulation

Flavor and food preferences are learned and can be modified through repeated exposure and modeling. Conversely, the ability to regulate volume of food intake is believed to be a normal human newborn behavior. Based on social science research, many child development experts believe that infants are innate "self-regulators" and possess the ability to respond to their own internal cues of hunger and satiety and that they communicate these feelings to feeders (Shea, Basch, Contento, & Rand Zybert, 1992). In young infants, crying, excited arm and leg movements, and moving forward as the breast or bottle is offered with mouth open can be signs of hunger. Signs of satiety can include fussing during feeding, slowing down the pace of eating, falling asleep, or spitting out or refusing the nipple.

**Learning Point** Some parents are not skilled at interpreting infant "cues" or signals, and without proper guidance parents' behaviors can disrupt this natural regulation and children can develop responses that manifest in disordered eating.

One of the many reasons breastfeeding is encouraged is because it allows infants to choose their own portion sizes. The infant guides the feeding because mothers typically nurse until the infant indicates he or she is satisfied. The quantity of breast milk consumed by the infant is primarily under the infant's control (Dewey, Heinig, Nommensen, & Lonnerdal, 1991). The feeder does not teach overconsumption by expecting the infant to finish the bottle. After the first few months of an infant's life, because of the belief that their infant is not being satisfied by infant formula or for other reasons, some parents are eager to begin solid foods before an infant is ready. The AAP supports exclusive breastfeeding for approximately 6 months but recognizes that infants are often developmentally ready to accept complementary (solid) foods between 4 and 6 months (Kleinman,

2004). Developmental readiness should be the guide for when to begin offering complementary foods. Control of the tongue, which is important for swallowing, chewing, and handling of foods, and the ability to hold the head up are two developmental indicators of readiness. Fomon and Bell (1993) advised that when an adult spoons food into the mouth of an infant who does not yet possess the skills to show that he or she is no longer hungry, this adult behavior might increase the likelihood that the infant will learn to overeat.

### Disordered Eating

Disordered eating describes a full spectrum of feeding behaviors leading to a poor-quality diet. Disordered eating can manifest in nonorganic failure to thrive or in excessive weight or obesity in children. Although the following advisory was provided in the context of feeding children with developmental disabilities, they are useful words of wisdom for parents of any young child. Stein (2000) quotes the Kennedy Krieger Institute's Pediatric Feeding Disorders Program in Baltimore, Maryland, as recommending the following:

- Parents should feed their children a wide range of foods before the age of 15 to 18 months.
- Parents should keep a ready supply of healthful foods in the house and make unhealthy foods less available.
- Parents should teach children to communicate hunger by relating food intake to appetite.
- Parents should reinforce good mealtime behaviors and not the amount of food eaten.
- Parents should maintain ample time and effort for meals so children can relax during mealtimes and therefore attend to hunger cues, digest food better, and attain more enjoyment from eating.
- Parents should avoid power struggles, threat, and force-feeding.

When feeding their infants, some parents react to fussing or crying by persisting in offering food when a child is not actually hungry and is expressing something entirely different from hunger. At other times, parents do not respond to signs of fullness and continue to feed beyond a child's hunger. Starting at around the age of 6 months, infants display more overt signs of interest or disinterest in eating. Children who are being spoon-fed will close their lips and turn away when offered food if they are not hungry. Quite simply, infants seem to eat when hungry and stop when full. Although it appears that infants possess the innate ability to self-regulate, childhood eating behaviors are formed in a social context, usually within a family. Many parents do not practice healthy eating themselves and/or may not understand the value of repeated exposures to healthy foods for young children or that their infants and young children know when they are hungry and full.

Parents' expectations that infants will immediately either accept or reject a new food can be a barrier to successful feeding. It has long been known that repeated exposures to new foods are typically necessary for acceptance (Birch & Marlin, 1990). Furthermore, when parents do not accurately interpret cues or messages from their infants and are also concerned that their children are eating too much or too little, they might resort to coercion of a child at mealtimes to respond to a meal or snack according to guidelines imposed by the parents. Parents often believe they have to motivate their infants to eat. However, children are naturally curious about trying new foods and will consume a varied diet if the foods are made available to them in the context of a pleasing and positive feeding experience. Children should be allowed to use their own hunger and satiety cues to initiate and terminate eating. Repeated opportunities to taste a new food are positively correlated with eventual preference, but coaxing, coaching, urging, or bribing, even when conducted in a pleasant fashion, interferes with the learning process of exploring and ultimately enjoying new foods, which comes from within the child.

Both coercing a child to eat a defined amount or limiting access to foods can promote disordered eating (i.e., lack of response to internal cues and overeating or undereating). Some parents become overly controlling in regard to what foods their young children are expected to eat. This has the unintended effect of promoting disordered eating in children, which persists into adulthood. Many pediatric nutritionists are familiar with the work of Ellyn Satter, who has written extensively on the feeding relationship between parent and child. Satter (1999) advised that parents should offer healthy foods at regular times during the day but that children should be the ones to determine whether and how much they will eat. Carper, Fisher, and Birch (2000) developed the Kids' Child-Feeding Questionnaire and used it to assess the relationship between controlling parental behavior during feeding and diminished use of their own hunger and satiety cues by 5-year-old girls. These researchers found that girls who perceived they were pressured to eat (parents' urging) engaged in both restrained eating and disinhibited eating. Cutting and colleagues (2000) found an association between the overeating styles of mothers who engaged in disinhibited eating and their daughters, who exhibited the same tendencies in an experimental environment.

Some social scientists believe that children can relearn self-regulation of energy intake through training to focus on internal cues to hunger and satiety. In an intriguing study, Johnson (2000) used dolls with detachable stomachs of three sizes to correspond with an empty, in-between, and full stomach. Over the course of 6 weeks, children learned to focus on feelings of hunger and fullness based on the three stomach sizes.

Although some individuals continue to self-regulate their calorie intake into adulthood, many people do not, as demonstrated by the work of Engell and associates (1995) and Booth, Fuller, and Lewis (1981). In general, when does the shift away from self-regulation occur? There is evidence that this process begins early. In a small study of 32 children, Rolls and coworkers (2000) demonstrated that children at the age of 3½ years responded well to internal cues and ate only the amounts they were accustomed to eating, but 5-year-old children ate larger amounts when the portions served were larger than their usual portions. The larger portions responded to by the 5-year-old children were an "external" cue. The findings of a similar study were interesting in that when 3- to 5-year-old children were repeatedly given a larger portion, they consumed about 25% more calories. However, when the same children were allowed to serve themselves they did not eat a larger than usual portion even though more food was available. This study suggests that the difference in response to more available food is that although parents offered a particular food, the children chose how much to eat and were better able to self-regulate their energy intake.

The impact of parenting related to feeding can be seen in eating styles of the children; the strongest association has been seen between mothers and their daughters. Birch and Fisher (2000) examined the influence on daughters' eating habits of maternal-child feeding practices. Mothers who reported they tended to restrict their own food consumption also restricted their daughters' food consumption. This is an example of how the inborn ability to self-regulate can be disrupted. Birch and Fisher also looked at short-term energy regulation of 3- to 6-year-old children after a meal. Daughters of mothers who scored higher on a scale measuring **dietary disinhibition** ate more when they had free access to food after they had already eaten a meal than did the children of mothers who were able to self-regulate. Although fathers and sons were also evaluated in this study, no relationship was found between dietary disinhibition between fathers and daughters, fathers and sons, or mothers and sons. Both maternal excessive weight and maternal dietary disinhibition were correlated with excessive weight in 3- to 6-year-old daughters. "Emotional" eating is commonly used to represent the phenomenon of disinhibited eating.

**dietary disinhibition** Unrestrained eating without regard to hunger or satiety.

### Parenting Styles

Parenting styles affect all areas of children's behavior and development, including eating behaviors. Baumrind (1998) defined four different parenting styles:

1. *Authoritative:* Parents exhibit consistent firm regulations and control; however, they give a clear explanation to their child for their standards. Authoritative parents are loving and supportive. In addition, these parents believe in autonomy for their children.
2. *Authoritarian:* Parents are very demanding of their child and are not strong believers of giving children a response and providing explanations. They discourage give-and-take feedback with their child. A parent who exhibits this style of parenting believes that it is "his/her way or no way." They tell their child what they should do and do not expect any feedback from the child.
3. *Permissive:* Parents demand very little of their children in regard to following rules, and they allow their children to do what they want. They provide minimal guidance. They are not neglectful, but they do not provide the structure of rules and consequences for behaviors.
4. *Rejecting-neglecting:* Parents do not monitor their child's behavior and tend to be disengaged from their child. They do not set limits for what the child does. Also, they are not responsive, so they do not provide any type of warmth to their child.

Are there some specific parenting styles that might be more likely to foster disordered eating in children? Baumrind (1998) described four different parenting styles. Rhee and colleagues (2006) examined the relationship between each of four parenting styles and excessive weight status in first-graders. In this study, parenting style was classified when the children were 4½ years old and the children were weighed 2 years later. The results of the study showed that a strict environment coupled with a lack of emotional responsiveness (authoritarian style) is most strongly associated with excessive weight in children. The kind of parent who is insensitive to the child's emotional needs and development might, for example, create a structure that requires a child to finish his or her entire meal, regardless of hunger. In this scenario an external cue would be imposed, disturbing the child's innate self-regulation. In the study by Rhee and coworkers, first-graders with authoritarian parents were found to have almost five times the odds of being overweight. The odds for children with permissive

or neglectful parents were also higher than those with authoritative parents by almost twice as much. Children whose parents displayed an authoritative parenting style (firm but loving) had the lowest prevalence of excessive weight.

Even well-meaning parents might not appreciate the value of eating meals with their young children. Mothers of infants might still be struggling to lose weight gained during pregnancy and might be skipping meals. Or parents might want a quiet mealtime together after their young children have had their meals. The opportunity for modeling healthy eating behaviors is thus lost.

### Anticipatory Guidance

Well-child visits to primary care providers (i.e., pediatricians, family practitioners, pediatric nurse practitioners) represent a missed opportunity for helping parents to promote healthy eating behaviors in their very young children. Well-child visits are most frequent in the first 2 years of a child's life, coinciding with the time of establishment of food preferences and eating behaviors. The concept of anticipatory guidance, or the provision of accurate messages before a practice is established, is well known in pediatric primary care medicine. Conceptually, enhancing the nutrition information that is usually discussed with behavioral strategies for promoting healthy eating makes sense. The reality is that primary care physicians are concerned about the lack of time they have for each well-child visit and, also, parents might suffer from information overload if too many topics are discussed on visits (Barkin et al., 2005). Nevertheless, physician–parent encounters at well-child visits present a potential opportunity for primary care providers to use age-appropriate "talking points" as a way to discuss early childhood nutrition. Some key messages, such as fruit is a more healthy choice than juice, could be reiterated at several well-child visits at sequential visits. If supported by teaching of primary care physicians to make them more adept at providing anticipatory guidance counseling and age-appropriate handouts that parents can take home, such discussions with parents of all children might foster healthier eating habits and might have a secondary effect of reducing the incidence of new cases of childhood obesity.

To accompany the preceding discussion between healthcare providers and parents, the parents can be provided with a handout to take home such as the one shown here:

#### Talking Points

Talking points for primary care practitioners are to be used when discussing nutrition on a well-child visit. These age-appropriate talking points could be discussed at every well-child visit and the information would build on previous information. The following example is for the 12-month checkup:

1. *Feeding routine: When and where does your child eat?* Your child should have a regular feeding routine including time and location, and the television should be off during meals.
2. *Dietary variety: What kinds of foods does your child like to eat?* Offer soft veggies, fruits, meats, pasta, and so on so your child can practice picking up and chewing. Limit juice to 4 oz daily. Did you know that fruit is a healthier choice that juice?
3. *Pleasant mealtimes: Does your child enjoy mealtimes?* Mealtime should be a pleasant and positive eating experience for both you and your child. I can help you with this if you want some ideas on how to make this happen.
4. *Competing foods: Does your child receive fatty or sweetened foods and drinks?* If a young child is offered foods such as french fries, chips, donuts, desserts, and soft drinks, he or she will learn to like and expect these foods instead of healthier choices such as whole fruit, vegetables, and milk. An example of this would be instead of giving a Pop-Tart for breakfast, you can offer whole-wheat toast with jam.

Used with permission from DeLessio, D., Robinson, B. B., & Shalon, L. S. (2003). *Physician education for prevention of childhood overweight*. Unpublished manuscript.

#### Helping Your Child Stay Healthy at 12 to 15 Months

What does your child drink? Now is a good time to offer whole milk and water in a cup.

- Soda, fruit drinks, and Kool-Aid don't help your child's body. Why not?
- They are easy to drink and taste so sweet that your child might not learn to like flavors of other foods, such as fruits and vegetables.
- They add extra calories but not many nutrients. Milk is more nutritious.

Your child is learning about her world, including what foods she likes.

- She will learn to eat healthy foods by watching you eat them.

Children learn to like foods they are given, especially before age 2 years.

- Now is the time to offer a wide variety of healthy foods so your child learns good eating habits for life.
- Did you know that children do best when they eat at a regular time and place?
- Make mealtimes pleasant. Turn off the television. Enjoy one another!

What are some healthy foods for children of this age?

- All fruits and vegetables (grapes cut in half and carrots cut lengthwise) are good choices.
- Soft beans, such as kidney beans, make good finger foods.
- Yogurt, cheese, and whole-wheat toast with jam are healthy snacks for your child.
- Sweet potato, broccoli, mango, oatmeal, beans, watermelon, cantaloupe, and milk are so nutritious that some experts call these "super foods."

- Do you know that children will learn to prefer less healthy foods such as french fries, doughnuts, candy, and other sweets if they are given them often?

**ACTIVITY**

- Have fun together inside and outside! Enjoy active playtime together such as walking, running, jumping, kicking and throwing a ball, or dancing.
- This is a great age for visiting zoos, beaches, and parks. Babies love swings.
- Your child will learn to be active if it is fun and if she sees that you enjoy being active.

**GOOD NUTRITION FOR YOUR 1-YEAR-OLD CHILD**

- Encourage your child to drink water during the day.
- Limit juice to 4 ounces a day.
- Serve milk with meals and snacks.

These foods are healthy and just right for your 1-year-old

**Breakfast**

½ cup of plain Cheerios
½ cup whole milk (red top)
½ banana, sliced

**Snack**

½ cup of yogurt
2 to 3 slices of fresh or canned peaches (drain juice from can)

**Lunch**

¼ cup rice
½ cup kidney beans cooked with tomatoes and a small amount of oil
¼ cup soft melon slices (no seeds)

**Snack**

½ cup whole milk
1 to 2 graham crackers
2 teaspoons peanut butter

**Dinner**

¼ cup meatloaf
¼ cup mashed potatoes
¼ cup cooked broccoli, cut up
1 small piece of corn bread
1 fresh orange, cut up, no seeds
½ cup whole milk

Used with permission from Shalon, L. S., Robinson, B. B., & DeLessio, D. (2003). Anticipatory guidance in early childhood for overweight prevention.

## Summary

It is undeniable that to provide essential nutrients for optimal growth and development, toddlers and young children must consume a wide variety of foods. This varied diet, coupled with physical fitness, is an excellent beginning for these young lives.

Growing toddlers and children rely on caregivers to provide them with adequate nutrition and guide them in the development of proper mealtime behaviors, ultimately resulting in healthy lifestyles into the adult years. However, outside influences, including the media and the food and beverage industries, make this challenge ever more difficult. Healthcare professionals must provide support for each family, with the goal of fostering understanding regarding the importance of good nutrition and consistent physical activity, and they must help parents acquire the necessary parenting skills to put their beliefs into practice when raising their young children.

There are many influences on the diet quality of the young child. As detailed in this chapter, constructing an environment for promotion of healthy eating in early childhood includes authoritative parenting style, repeated and early exposure to healthy foods, family dining, keeping competitive foods out of the home, and provision of sound advice before childhood eating habits are established (anticipatory guidance).

These are guidelines for parents that encompass the domains of infant and toddler development, parent–child interaction, the feeding relationship, nutritional requirements, development of food preferences, and eating behaviors as well as television viewing and physical activity during infancy and the toddler years. Central to the recommendations is the concept put forth in the guidelines as follows: "Responsive parenting is at the core of a healthy infant–parent feeding relationship" (Butte et al., 2004).

## Case Study 1

### Lactose Intolerance in Children

Jessica Brie Leonard, RD

1. Pediatrician Office Visit (MD). CT is a 6-year-old Asian American female who presents with stomach cramps, nausea, and intermittent diarrhea times approximately 2 and a half weeks.

Her anthropometrics are as follows:

*Weight:* 44 lb (20 kg)
*Height:* 45 inches (115 cm)
*Comparative standards:* 50th percentile weight for age, 50th percentile height for age (WHO growth charts)

This is CT's first visit to the medical doctor (MD) to address these symptoms. Her mother is present and expresses concern that CT's stomach cramps have not resolved on their own and her discomfort is affecting her appetite and mealtime consumption. CT has stated on several occasions that her "stomach hurts," but it is hard to discern the source of the pain. When she is asked what it feels like, CT states, "She feels full" and "like she needs to go to the bathroom."

On interview, the MD questions CT's mother about any recent illness and discovers CT had a recent bout of strep throat for which she was prescribed antibiotics, which she finished approximately 10 days ago. The MD explains to CT's mother that her symptoms are most likely the result of the antibiotics and that symptoms can occur even after the prescription has been finished. He advises her to make sure CT is drinking plenty of fluids when she is having diarrhea and recommends she offer CT her favorite foods to encourage increased mealtime intake.

2. Phone Follow-Up (RD referral). Four days after visiting the MD, CT's mother calls the office to say that her symptoms have not resolved and seem to be getting worse. She has tried offering CT her favorite foods to keep her eating, but that seems to be making things worse. The MD refers CT to a dietitian to review her dietary intake to determine whether the stomach cramps, nausea, and diarrhea are being caused by her diet.

3. Nutrition Assessment (RD). CT's mother meets with the registered dietician (RD) and explains that the stomach cramps, diarrhea, and nausea have been going on for about 3 weeks. She says that her child's diarrhea seems to be the worst in the morning and that CT has stayed home from school for the last couple of days for fear that she'll have diarrhea at school. The RD does a 24-hr recall with CT and her mother that reveals the following:

**Breakfast**

Cheerios with 1% milk
Orange juice

**Snack**

Vanilla pudding
Water

**Lunch**

Instant mac and cheese
Baby carrots
Peanut butter
1% milk

**Snack**

Apple

**Dinner**

Cream of broccoli soup
Crackers
1% milk
Ice cream

CT's mother says that her symptoms seem to be better in the afternoon but are usually bad again around bedtime.

The RD suspects that CT's symptoms are to the result of lactose intolerance given that her symptoms are worst in the morning when both her breakfast and morning snack are high in dairy. The RD shares her findings with the MD, who orders a lactose breath test for CT. The test reveals that CT has lactose intolerance, so she and her mother return to the RD for education on diet strategies to manage her symptoms.

4. Nutrition Follow-up (RD). At CT's follow-up visit, the RD explains that CT's body doesn't make enough lactase, the enzyme that enables people to digest lactose, and that this has been causing her stomach cramps, nausea, and diarrhea when she eats dairy foods. CT's mother is concerned that she did something wrong when CT was a baby to make this happen. The RD explains that lactose intolerance is genetic and she didn't do anything wrong. The next step is going to be to remove foods with lactose from CT's diet. Lactose is found in milk and any food that is made with milk. Some substitutions the RD recommends based on CT's 24-hr food recall are the following:

- Lactaid milk or soy milk instead of 1% milk
- Calcium-fortified orange juice instead of orange juice
- Soy pudding or soy yogurt instead of pudding
- Pasta with tomato sauce instead of mac and cheese
- Grilled chicken and steamed broccoli instead of cream of broccoli soup

CT may be able to tolerate some lactose, but to start the best approach will be to do a lactose-free diet. The RD explains that Lactaid milk has lactase added to it so CT can digest it. They also review that it will be important to make sure CT's diet includes alternate sources of calcium and vitamin D to maintain growth and development of her bones.

The long-term goal for CT's nutrition care will be to determine whether CT can tolerate low levels of lactose in her diet. This will not be explored until CT's stools become more formed, and her tolerance should be monitored. It also will be recommended that if CT is going to eat foods with lactose, that she eats them with fat, protein, or fiber, which will slow their absorption and reduce CT's intolerance symptoms.

**Questions**

1. Name five ingredients that will be important for CT to avoid eating.
2. Name three additional nutrients, besides calcium and vitamin D, that will be missing, or decreased, on CT's lactose-free diet.
3. What are some alternative sources of calcium and vitamin D?
4. Should CT consume the following foods on her lactose-free diet? Indicate Yes or No for each.
   Red velvet cupcake with cream cheese frosting
   Grilled chicken on green salad with Italian dressing
   Strawberry soy yogurt
   Creamed spinach
   Lime gelatin
   Vegetable quiche
   Sherbet
   Sorbet
5. What two ethnicities have the highest prevalence of lactose intolerance?

## Case Study 2

### Picky Eater

Jennifer Sabo, RD, LDN, CNSD

Anna is a 2½-year-old girl. Her mother considers her a picky eater. She loves milk and sometimes will only drink at a meal and pick at the other foods. Her favorite foods are oatmeal, yogurt, dried cereal, macaroni and cheese, and pasta with tomato sauce. She doesn't really like to sit down for a meal. Her mom leaves snacks out so she can grab a bite or two while she is playing and then continue to play. She carries a sippy cup of milk around with her too in case she gets thirsty. Her mom is concerned that she is not getting enough nutrients. Her growth has started to slowly fall off her normal curve, and she also seems pale and more tired during the day.

#### Questions

1. What are some nutritional concerns you may have for Anna?
2. Why might her growth be inadequate?
3. What suggestions could you make to her mother?

## Case Study 3

### Snack Foods

Jennifer Sabo, RD, LDN, CNSD

Mark is a 5-year-old male who loves snack foods. He doesn't really like to sit down for a meal. His mom will leave the snacks out so he can grab a bite or two while he is playing and then continue to play. He carries his sippy cup of juice around with him too in case he gets thirsty. He has not been growing well, but his mom says that once he grows out of this phase, he will catch up.

#### Questions

1. Why might Mark's growth be inadequate?
2. What changes to Mark's current eating habits would you recommend?

## Case Study 4

### Obesity

Jennifer Sabo, RD, LDN, CNSD

Tony is an 11-year-old male whose favorite activity is his Playstation system. He loves to come home from school and start playing right away. He will usually grab a large bag of potato chips and a soda from the fridge and snack while he plays. He will take a break for dinner, but then he heads right back to the game. Tony has always plotted around the 50th percentile for weight and height for age, but lately he has been gaining weight and is now greater than the 95th percentile for weight. His father is not concerned, reporting that he was the same way at his age but "thinned out" as he got older.

#### Questions

1. Should Tony's weight gain be a concern, despite his family history?
2. What recommendations could be made to the family?

## References


Addessi, E., Galloway, A. T., Visalberghi, E., & Birch, L. L. (2005). Specific social influences on the acceptance of novel foods in 2–5 year-old children. *Appetite*, *45*, 264–271.

American Academy of Pediatrics. (1999). Calcium requirements of infants, children, and adolescents. *Pediatrics*, *104*, 1152–1157.

American Academy of Pediatrics. (2001). Children, adolescents, and television. *Pediatrics*, *107*, 423–426.

American Academy of Pediatrics. (2008). Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. *Pediatrics*, *122*, 1141–1153.

American Academy of Pediatrics Committee on Nutrition. (1992). Statement on cholesterol. *Pediatrics*, *90*, 469–473.

American Academy of Pediatrics Committee on Nutrition. (1988). *Cholesterol in childhood*. Elk Grove, IL: Author.

American Academy of Pediatrics Committee on Nutrition. (2001). The use and misuse of fruit juice in pediatrics. *Pediatrics*, *107*, 1210–1213.

American Heart Association. (2013). Dietary recommendations for healthy children. Retrieved from www.heart.org/HEARTORG/GettingHealthy/Dietary-Recommendations-for-Healthy-Children_UCM_303886_Article.jsp

American Heart Association. (n.d.). AHA scientific position: Dietary guidelines for healthy children. Retrieved from www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/Dietary-Recommendations-for-Healthy-Children_UCM_303886_Article.jsp

Bandura, A. (1972). Modeling theory: Some traditions, trends, and disputes. In R. D. Parke (Ed.), *Recent trends in social learning theory.* New York, NY: Academic Press.

Baranowski, T. (1992). Assessment, prevalence, and cardiovascular benefits of physical activity and fitness in youth. *Medicine and Science in Sports and Exercise*, *24*, S237–S247.

Baranowski, T., Cullen, K. W., & Baranowski, J. (1999). Psychosocial correlates of dietary intake: Advancing dietary intervention. *Annual Review of Nutrition*, *9*, 17–40.

Barkin, S. L., Scheindlin, B., Brown, C., Ip, E., Finch, S., & Wasserman, R. C. (2005). *Ambulatory Pediatrics*, *5*, 372–356.

Baumrind, D. (1998). Rearing competent children. In W. Damon (Ed.), *Child development today and tomorrow*. San Francisco, CA: Jossey-Bass.

Birch, L. L. (1980). Effects of peer models' food choices and eating behaviors on preschoolers' food preferences. *Child Development*, *5*, 489–496.

Birch, L. L., & Fisher, J. A. (1995). Appetite and eating behavior in children. *Pediatric Clinics of North America*, *42*, 931–935.

Birch, L. L., & Fisher, J. O. (1998). Development of eating behaviors among children and adolescents. *Pediatrics*, *101*, 539–549.

Birch, L. L., & Fisher, J. O. (2000). Mothers' child-feeding practices influence daughters' eating and weight. *American Journal of Clinical Nutrition*, *71*, 1054–1061.

Birch, L. L., & Marlin, D. W. (1982). "I don't like it; I never tried it": Effects of exposure on two-year-old children's food preferences. *Appetite*, *3*, 353–360.

Booth, D., Fuller, J., & Lewis, V. (1981). Human control of body weight: Cognitive or physiological? Some energy-related perceptions and misperceptions. In L. Cioffi, W. James, & T. van Italie (Eds.), *The body weight regulatory system: Normal and disturbed mechanisms* (pp. 305–314). New York, NY: Raven Press.

Briefel, R., Hanson, C., Fox, M. K., Novak, T., & Ziegler, P. (2006). Feeding Infants and Toddlers Study: Do vitamin and mineral supplements contribute to nutrient adequacy or excess among US infants and toddlers? *Journal of the American Dietetic Association*, *1*(Suppl.), S52–S65.

Briefel, R. R., Kalb, L. M., Condon, E., Deming, D. M., Clusen, N. A., Fox, M. K., … Reidy, K. C. (2010). The Feeding Infants and Toddlers Study 2008: Study design and methods. *Journal of the American Dietetic Association*, *110*(12 Suppl.), S16–26.

Burdette, H. L., & Whitaker, R. C. (2005). A national study of neighborhood safety, outdoor play, television viewing, and obesity in preschool children. *Pediatrics*, *116*, 657–662.

Butte, N. (2000). Fat intake of children in relation to energy requirements. *American Journal of Clinical Nutrition*, *72*(Suppl.), 1246S–1252S.

Butte, N. F., Cobb, K., Dwyer, J., Graney, L., Heird, W., Richard, K., … Gerber Products Company. (2004). The Start Healthy Feeding Guidelines for Infants and Toddlers. *Journal of the American Dietetic Association*, *104*, 443–454.

Butte, N. F., Fox, M. K., Briefel, R. R., Siega-Riz, A. M., Dwyer, J. T., Deming, D. M., & Reidy K. C. (2010). Nutrient intakes of US infants, toddlers, and preschoolers meet or exceed dietary reference intakes. *Journal of the American Dietetic Association*, *110*(12 Suppl.), S27–S37.

Butte, N. F., Wong, W. W., Hopkinson, J. M., Heinz, C. J., Mehta, N. R., & O'Brian Smith, E. (2000). Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. *American Journal of Clinical Nutrition*, *72*, 1558–1569.

Carper, J. L., Fisher, J. O., & Birch, L. L. (2000). Young girls' emerging dietary restraint and disinhibition are related to parental control in child feeding. *Appetite*, *35*, 121–129.

Carruth, B. R., & Skinner, J. (2000). Preschoolers food product choices at a simulated point of purchase and mothers' consumer practices. *Journal of Nutrition Education*, *33*, 143–147.

Carruth, B. R., Skinner, J., Houck, K., Moran, J., Coletta, F., & Ott, D. (1998). The phenomenon of "picky eater": A behavioral marker in eating patterns of toddlers. *Journal of the American College of Nutrition*, *17*, 180–186.

Carter, L. M., Whiting, S. J., Drinkwater, D. T., Zello, G. A., Faulkner, R. A., & Baily, D. A. (2001). Self-reported calcium intake and bone mineral content in children and adolescents. *Journal of the American College of Nutrition*, *20*, 502–509.

Centers for Disease Control and Prevention. (2000). Tracking Healthy People 2010. Retrieved from www.cdc.gov/nchs/healthy_people/hp2010/hp2010_thp.htm

Center for Disease Control and Prevention & National Center for Health Statistics. (2000). Growth Charts. Retrieved from www.cdc.gov/growthcharts/2000growthchart-us.pdf

Centers for Disease Control and Prevention. (2001). Recommendations for using fluoride to prevent and control dental caries in the United States. *Morbidity and Mortality Weekly Report*, *50*(RR-14), 26–27.

Centers for Disease Control and Prevention. (2003). Physical activity levels among children aged 9–13 years: United States, 2002. *Morbidity and Mortality Weekly Report*, *52*, 785–788.

Centers for Disease Control and Prevention. (2012). Childhood overweight and obesity. Retrieved from www.cdc.gov/obesity/childhood/index.html

Cho, S., Dietrich, M., Brown, C. J., Clark, C. A., & Block, G. (2003). The effect of breakfast type on total daily energy intake and body mass index: Results from the Third National Health and Nutrition Examination Survey (NHANES III). *Journal of the American College of Nutrition*, *22*, 296–302.

Cutting, T. M., Fisher, J. O., Grimm-Thomas, K., & Birch, L. L. (1999). Like mother, like daughter: Familial patterns of overweight are mediated by mothers' dietary disinhibition. *American Journal of Clinical Nutrition*, *69*, 608–613.

Dennison, B. A. (1996). Fruit juice consumption by infants and children: A review. *Journal of the American College of Nutrition*, *15*(Suppl. 5), 4S–11S.

Dennison, B. A., Rockwell, H. L., & Baker, S. L. (1998). Fruit and vegetable intake in young children. *Journal of the American College of Nutrition*, *17*, 371–378.

Dewey, K. G., Heinig, J., Nommensen, L. A., & Lonnerdal, B. (1991). Maternal versus infant factors related to breast milk intake and residual milk volume. *Pediatrics*, *87*, 829–837.

Dietz, W. H., & Stern, L. (1999). *American Academy of Pediatrics: Guide to your child's nutrition* (pp. 41–60). New York, NY: Random House.

Dwyer, J. T., Sutor, C. W., & Hendricks, K. (2004). FITS: New insights and lessons learned. *Journal of the American Dietetic Association*, *104*(Suppl. 1), s25–s27.

Engell, D., Kramer, M., Zaring, D., Birch, L. L., & Rolls, B. J. (1995). Effects of serving size on food intake in children and adults. *Obesity Research*, *3*(Suppl. 3), 381s.

Epstein, L. H., Coleman, K. J., & Myers, M. D. (1996). Exercise in treating obesity in children and adolescents. *Medicine and Science in Sports and Exercise*, *28*, 428–435.

Etelson, D., Brand, D. A., Patrick, P. A., & Shirali, A. (2003). Childhood obesity: Do parents recognize this health risk? *Obesity Research*, *11*, 1362–1368.

Fisher, E. A., Van Horn, L., & McGill, H. C. (1997). Nutrition and children: A statement for healthcare professionals from the Nutrition Committee, American Heart Association. *Circulation*, *95*, 2332–2333.

Fisher, J. O., & Birch, L. L. (1999). Restricting access to palatable foods affects children's behavioral response, food selection, and intake. *American Journal of Clinical Nutrition*, *69*, 1264–1272.

Fisher, J. O., & Birch, L. L. (2000). Parent's restrictive feeding practices are associated with young girls' negative self-evaluation of eating. *Journal of the American Dietetic Association*, *100*, 1341–1346.

Fomon, S. J., & Bell, E. F. (1993). *Nutrition of normal infants: Energy*. St. Louis, MO: Mosby-Year Book.

Food and Agricultural Organization, World Health Organization, & United Nations University Expert Consultation. (1985). Energy and protein requirements. *World Health Organization Technical Report Series*, *724*, 1–206.

Food Research and Action Center. (n.d.).School breakfast program. *Child Nutrition Fact Sheet*. Washington, DC: Author. Retrieved from www.frac.org/pdf/cnsbp.PDF

Fox, M. K., Pac, S., Devaney, B., & Janowski, L. (2004). Feeding Infants and Toddlers Study. What are infants and toddlers eating? *Journal of the American Dietetic Association*, *104*(Suppl. 1), S22–S30.

Fox, M. K., Reidy, K., Novak, T., & Ziegler, P. (2006). Sources of energy and nutrients in the diets of infants and toddlers. *Journal of the American Dietetic Association*, *1*(Suppl.), S28–S42.

Freedman, D. S., Thornton, J. C., Mei, Z., Wang, J., Dietz, W. H., Pierson, R. N., & Horlick, M. (2004). Height and adiposity among children. *Obesity Research*, *12*, 846–853.

Gidding, S., Dennison, B., Birch, L. L., Daniels, S., Gilman, M. W., Lichtenstein, A. H., … Van Horn, L. (2005). Dietary recommendations for children and adolescents: A guide for practitioners. Consensus Statement from the American Heart Association. *Circulation*, *112*, 2061–2075.

Gortmaker, S. L., Must, A., & Sobol, A. M. (1996). Television viewing as a cause of increasing obesity among children in the United States. *Archives of Pediatric and Adolescent Medicine*, *150*, 356–362.

Grantham-McGregor, S., & Ani, C. (2001). A review of studies on the effect of iron deficiency on cognitive development in children. *Journal of Nutrition*, *131*, 649S–668S.

Harper, L. V., & Sanders, K. M. (1975). The effect of adults' eating on young children's acceptance of unfamiliar foods. *Journal of Experimental Child Psychology*, *20*, 206–214.

Hayman, L. L. (2003). The Dietary Intervention Study in Children (DISC): Progress and prospects for primary prevention. *Progress in Cardiovascular Nursing*, *18*, 4–5.

Hendy, H. M. (2002). Effectiveness of trained peer models to encourage food acceptance in preschool children. *Appetite*, *39*, 217–225.

Holick, M. F., Shils, M. E., Olsen, J., Shike, M., & Ross, C. A. (Eds.). (1999). *Modern nutrition in health and disease* (9th ed., p. 329). Baltimore, MD: Williams & Wilkins.

Institute of Medicine, Food and Nutrition Board. (2002/2005, 2011). Dietary Reference Intakes tables and applications. Retrieved from www.iom.edu/Activities/Nutrition/SummaryDRIs/DRI-Tables.aspx

Jahns, L., Siega-Riz, A. M., & Popkin, B. M. (2001). The increasing prevalence of snacking among U.S. children from 1977 to 1996. *Journal of Pediatrics*, *138*, 493–498.

Johnson, S. L. (2000). Improving preschoolers' self-regulation of energy intake. *Pediatrics*, *106*, 1429–1435.

Johnston, C. C., Miller, J. Z., Slemenda, C. W., Reister, T. K., Hui, S., Christian, J. C., & Peacock, M. (1992). Calcium supplementation and increases in bone mineral density in children. *New England Journal of Medicine*, *327*, 82–87.

Kaiser, L. (2004). What are infants and toddlers eating? Maternal and Infant Briefs: Jan/Feb edition. Retrieved from http://ucanr.org/blogs/minbblog

Kim, J., & Peterson, K. (2008). Association of infant child care with infant feeding practices and weight gain among US infants. *Archives of Pediatric and Adolescent Medicine*, *162*, 627–633.

Kleinman, R. E. (Ed.). (2004). *Pediatric nutrition handbook* (p. 105). Elk Grove, IL: American Academy of Pediatrics.

Krugman, S., & Dubowitz, H. (2003). Failure to thrive. *American Family Physician*, *68*, 879–884.

Lipsitt, L. P., Crook, C., & Booth, C. (1985). The transitional infant: Behavioral development and feeding. *American Journal of Clinical Nutrition*, *41*, 485–496.

Mahan, L. K., & Escott-Stump, S. (2000). *Krause's food, nutrition, and diet therapy* (10th ed.). Philadelphia, PA: W. B. Saunders.

May, A.L., & Dietz, W. H. (2010). The Feeding Infants and Toddlers Study 2008: Opportunities to assess parental, cultural, and environmental influences on dietary behaviors and obesity prevention among young children. *Journal of the American Dietetic Association*, *110*, S11–S15.

Mennella, J. A., Jagnow, C. J., & Beauchamp, G. K. (2001). Pre- and postnatal flavor learning by human infants. *Pediatrics*, *107*, 88–93.

Mennella, J. A., Johnson, A., & Beauchamp, G. K. (1995). Garlic ingestion by pregnant women alters the odor of amniotic fluid. *Chemical Senses*, *20*, 207–209.

National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion. (2000). Retrieved from www.cdc.gov/growthcharts

National Institutes of Health. (n.d.). Body mass index table. Retrieved from www.nhlbi.nih.gov/guidelines/obesity/bmi_tbl.pdf

National Institutes of Health. (2004). Site specific approaches to prevention or management of pediatric obesity. RFK No. RFA-DK-04-013. Retrieved from http://grants.nih.gov/grants/guide/rfa-files/RFA-DK-04-013.html

Ogden, C. L., Flegal, K. M., Carroll, M. D., & Johnson, C. L. (2002). Prevalence and trends in overweight among US children and adolescents, 1999–2000. *Journal of the American Medical Association*, *288*, 1728–1732.

Ong, K. K., Emmett, P. M., Noble, S., Ness, A., Dunger, D. B., & The ALSPAC Study Team. (2006). Dietary energy intake at the age of 4 months predicts postnatal weight gain and childhood body mass index. *Pediatrics*, *117*, 503–508.

Orlet Fisher, J., Rolls, B. J., & Birch, L. L. (2003). Children's bite size and intake of an entrée are greater with large portions than with age-appropriate or self selected portions. *American Journal of Clinical Nutrition*, *77*, 1164–1170.

Patrick, H., & Nicklas, T. A. (2005). A review of family and social determinants of children's eating patterns and diet quality. *Journal of the American College of Nutrition*, *24*, 83–92.

Pelcaht, M. L., & Pliner, P. (1986). Antecedents and correlates of feeding problems in young children. *Journal of Nutrition Education*, *18*, 23–29.

Perrin, E., Armstrong, S., & Dolinsky, D. (2011, January 1). Recognizing and preventing childhood obesity. Part 1 of 2. *ContemporaryPediatrics*.Retrievedfromhttp://contemporary-pediatrics.modernmedicine.com/contemporary-pediatrics/news/modernmedicine/modern-medicine-feature-articles/recognizing-and-prevent

Polhamus, B., Dalenius, K., Mackintosh, H., Smith, B., & Grummer-Strawn, L. (2011). *Pediatric nutrition surveillance 2009 report*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Rachlin, H., Logue, A. W., Gibbon, J., & Frankel, M. (1986). Cognition and behavior in studies of choice. *Psychology Review*, *93*, 33–45.

Rhee, K. E., Lumeng, J. C., Appugliese, D. P., Kaciroti, N., & Bradley, R. H. (2006). Parenting styles and overweight status in first grade. *Pediatrics*, *117*, 2047–2054.

Robinson, T. N. (2001). Television viewing and childhood obesity. *Pediatric Clinics of North America*, *48*, 1017–1025.

Saelens, B. E., Sallis, J. F., Nader, P. R., Broyles, S. L., Berry, C. C., & Taras, H. L. (2002). Home environment influences on children's television watching from early to middle childhood. *Journal of Developmental and Behavioral Pediatrics*, *23*, 127–132.

Sallis, J. F., Prochaska, J. J., & Taylor, W. C. (2000). A review of correlates of physical activity of children and adolescents. *Medicine and Science in Sports and Exercise*, *32*, 963–975.

Salmon, J. O., Timperio, A., Telford, A., Carver, A., & Crawford, D. (2005). Association of family environment with children's television viewing and with low level of physical activity. *Obesity Research*, *13*, 1939–1951.

Salsberry, P. J., & Reagan, P. B. (2005). Dynamics of early childhood overweight. *Pediatrics*, *116*, 1329–1338.

Satter, E. M. (1986). The feeding relationship. *Journal of the American Dietetic Association*, *86*, 352–356.

Satter, E. M. (1996). Internal regulation and the evolution of normal growth as the basis for prevention of obesity in children. *Journal of the American Dietetic Association*, *96*, 860–864.

Satter, E., M. (1999). *Secrets of feeding a healthy family*. Madison, WI: Kelcy Press.

Schall, G., Marlier, L., & Soussignan, R. (2000). Human fetuses learn odours from their pregnant mothers' diet. *Chemical Senses*, *25*, 729–737.

Schneider, J. M., Fujii, M. L., Lamp, C. L., Lonnerdal, B., Dewey, K. G., & Zidenberg-Cherr, S. (2005). Anemia, iron deficiency, and iron deficiency anemia in 12-36-mo-old children from low-income families. *American Journal of Clinical Nutrition*, *82*, 1269–1275.

Scrimshaw, N. S., & Murray, E. B. (1988). Prevalence of lactose maldigestion. *American Journal of Clinical Nutrition*, *48*(Suppl.), 1086–1098.

Shea, S., Basch, C. E., Contento, I., & Rand Zybert, P. (1992). Variability and self-regulation of energy intake in young children in their everyday environment. *Pediatrics*, *90*, 542–546.

Skinner, J. D., Carruth, B. R., Bounds, W., & Zeigler, P. J. (2002). Children's food preferences: A longitudinal analysis. *Journal of the American Dietetic Association*, *102*, 1638–1647.

Smith, M. M., & Lifshitz, F. (1994). Excess fruit juice consumption as a contributing factor in nonorganic failure to thrive. *Pediatrics*, *93*, 438–443.

Specker, B. L., Valanis, B., Hertzberg, V., Edwards, N., & Tsang, R. (1985). Sunshine exposure and serum 25-hydroxyvitamin D concentrations in exclusively breast-fed infants. *Journal of Pediatrics*, *107*, 372–376.

Stein, K. (2000). Children with feeding disorders: An emerging issue. *Journal of the American Dietetic Association*, *100*, 1000–1001.

Storey, M. L., Forchee, R. A., & Anderson, P. A. (2004). Associations of adequate intake of calcium with diet, beverage consumption, and demographic characteristics among children and adolescents. *Journal of the American College of Nutrition*, *23*, 18–33.

Stettler, N., Zemel, B. S., & Kumanyika, S. (2002). Infant weight gain and childhood overweight status in a multicenter, cohort study. *Pediatrics*, *109*, 194–199.

Story, M., & French, S. (2004). Food advertising and marketing directed at children and adolescents in the US. *International Journal of Behavioral Nutrition and Physical Activity*, *1*, 1–17.

Sullivan, S. A., & Birch, L. L. (1990). Pass the sugar, pass the salt: Experience dictates preference. *Developmental Psychology*, *26*, 546–555.

Sullivan, S. A., & Birch, L. L. (1994). Infant dietary experience and acceptance of solid foods. *Pediatrics*, *93*, 271–277.

Taveras, E. M., Sandora, T. J., Shih, M. C., Ross-Degnan, D., Goldmann, D. A., & Gillman, M. W. (2006). The association of television and video viewing with fast food intake by preschool-aged children. *Obesity*, *14*, 2034–2041.

Tomashek, K. M., Nesby, S., Scanlon, K. S., Cogswell, M. E., Powell, K. E., Parashar, U. D., … Dietz, W. H. (2001). Nutritional rickets in Georgia. *Pediatrics*, *107*, e45.

U.S. Department of Agriculture, Food and Nutrition Service. (n.d.). About WIC. Retrieved from www.fns.usda.gov/wic/aboutwic/default.htm

U.S. Department of Agriculture, Food and Nutrition Service. (2005). National school lunch program (NSLP). Retrieved from www.fns.usda.gov/nslp/national-school-lunch-program

U.S. Department of Health and Human Services. (2000). Healthy People 2010. Retrieved from www.healthypeople.gov/2020/default.aspx

Vesa, T. H., Marteau, P., & Korpela, R. (2000). Lactose intolerance. *Journal of the American College of Nutrition*, *19*, 165S–175S.

Waller, C. E., Du, S., & Popkin, B. M. (2003). Patterns of overweight, inactivity, and snacking in Chinese children. *Obesity Research*, *11*, 957–961.

Weisberg, P., Scanlon, K. S., Li, R., & Cogswell, M. E. (2004). Nutritional rickets among children in the United Stated: Review of cases reported between 1986 and 2003. *American Journal of Clinical Nutrition*, *80*(Suppl.), 1697S–1705S.

Zlotkin, S. H. (1996). A review of the Canadian nutrition recommendations update: Dietary fat and children. *Journal of Nutrition*, *126*(Suppl.), 1022S–1027S.

# CHAPTER 4 Normal Adolescent Nutrition

*Pamela S. Hinton, PhD*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Understand nutritional regulation of the hormones that moderate growth and sexual maturation.
2. Describe gender differences in growth and development and in nutrient requirements.
3. Appreciate how psychosocial development during adolescence affects health-related behaviors, including dietary patterns.
4. Identify and describe sociodemographic factors affecting dietary patterns.
5. Describe trends in chronic disease incidence among adolescents.

Adolescence is a period of rapid linear growth, altered body composition, reproductive maturation, and psychosocial development. Nutrient requirements are increased to meet the demands of growth and development. There are significant gender differences in the timing and rate of peak linear growth, puberty, and sexual development, resulting in divergent nutrient needs. Normal growth and development are influenced by nutritional status because the hormones responsible for linear growth, alteration in body composition, and sexual development are nutritionally regulated. Food intake during adolescence is influenced by psychosocial factors; peers and popular culture, including the mass media and advertising, significantly affect dietary patterns.

## Growth and Development

### Physical Growth

This section begins with a focus on physical changes of adolescence. In particular, height, weight, and sexual maturation are addressed.

#### Peak Height Velocity

The adolescent growth spurt takes 2 to 4 years to complete and is generally longer in boys than in girls. The average height velocity is 5 to 6 centimeters (cm) per year during adolescence; peak height velocity (maximal rate of linear growth) is 8 to 10 cm/year. Girls, on average, begin their pubertal growth spurt at age 9 years (Veldhuis et al., 2005) and achieve their peak height velocity at an average chronologic age of 11.5 years, corresponding to Tanner breast stages 2 and 3. The onset of the pubertal growth spurt occurs at age 11 years in boys, and peak height velocity occurs at age 13.5 years, normally Tanner genital stages 3 and 4. The onset of the adolescent growth spurt is more closely associated with bone age than with chronologic age. Boys, because their peak height velocity is greater than that of girls, 9.5 cm/yr versus 8.3 cm/yr, and because peak height velocity occurs later in boys than in girls, are on average 13 cm taller than girls. Interindividual variation in the onset and rate of pubertal growth and development is significant as a result of interaction between a child's genetic potential and his or her environment.

#### Body Composition

Normative body composition data from prospective population-based samples are lacking in adolescent populations, especially ethnic and gender-specific data. During adolescence, girls gain fat mass at an average rate of 1.14 kilograms (kg) per year. In contrast, boys do not experience a significant increase in absolute fat mass. Boys also gain fat-free mass at a greater rate and for a longer period of time than girls do; as a result, boys are relatively leaner than girls after puberty. At ages 8 to 10 years, boys, on average, have 15% body fat and 24 kg fat-free mass. At the end of puberty, ages 18 to 20 years, young men have 13% body fat and 60 kg fat-free mass. In contrast, girls have 20% body fat and 24 kg fat-free mass at ages 8 to 10 years. By ages 18 to 20 years, young women have 26% body fat and 44 kg fat-free mass (Guo, Chunlea, Roche, & Siervogel, 1997).

#### Bone Growth and Mineralization

In the immature skeleton, the ends of the long bones (**epiphyses**) are separated from the shafts of the long bones (**diaphyses**) by

**epiphyses** Ends of long bones.

**diaphyses** Shafts of long bones.

the growth plate. After puberty the growth plate becomes mineralized and the epiphyses fuse. Thus, the mature skeleton is incapable of additional growth in height or length.

Bone mass doubles between the onset of puberty and young adulthood. Bone growth is greatest approximately 6 months after peak height velocity (Whiting et al., 2004); approximately 25% of peak adult bone mass is acquired during the 2 years of peak adolescent skeletal growth. Growth of the skeleton occurs via modeling, which changes both the size and shape of the bones. Bones increase in length by ossification of the growth plates and in diameter by **periosteal apposition** and **endosteal resorption**. When the growth plates fuse after puberty, bone mass density (BMD) is 90% to 95% of peak BMD (Riggs, Khosla, & Melton, 2002). Boys are, on average, 10% taller and have 25% greater peak bone mass than do girls because of their later pubertal onset and longer growth spurt (Riggs et al., 2002).

**periosteal apposition**
Deposition of bone on the outer surface.

**endosteal resorption**
Removal of bone from the inner surface.

## CRITICAL Thinking

After reading the statement "Thus, the mature skeleton is incapable of additional growth in height or length," describe how you think nutrition in childhood affects skeletal growth.

### Calcium Absorption and Retention

The maximal rate of calcium accretion in the skeleton occurs at ages 11 to 14 years in girls and 16 to 18 years in boys. During the 2 years of peak bone growth, boys gain approximately 400 grams (g) of bone mineral and girls deposit approximately 325 g. Fractional calcium absorption is increased to approximately 40% during peak bone mineral deposition (Molgaard, Thomsen, & Michaelsen, 1999). In adolescents, there is a linear relationship between dietary calcium intake and calcium retention. During adulthood, fractional absorption decreases with increasing dietary calcium. In contrast, increasing daily calcium consumption to approximately 1,900 milligrams (mg; 47.4 mmol) did not reduce fractional calcium absorption compared with 850 mg (21.2 mmol)/day in adolescent girls (Wastney et al., 2000; Weaver, 2000). Because the additional calcium suppressed bone resorption, calcium retention was significantly increased by the higher calcium intake (Weaver, 2000). In a calcium balance study of adolescent girls, African Americans were found to have increased calcium retention, bone formation relative to bone resorption, fractional calcium absorption, and decreased urinary excretion compared with whites (Bryant et al., 2003).

### Bone Turnover Markers

Biochemical markers of bone formation and resorption also are increased during puberty and generally parallel the rate of linear growth (Federico, Baroncelli, Vanacore, Fiore, & Saggese, 2003). Bone turnover during puberty is up to 10-fold higher than during adulthood (Eastell, 2005): The rate of remodeling is increased, linear growth occurs at the epiphyseal growth plates, and there is modeling of bone, which changes the shape of the bones by periosteal or endosteal apposition. Longitudinal studies demonstrate that bone turnover markers are greatest early in puberty, whereas maximal mineral deposition occurs late in puberty when bone turnover markers in serum are decreasing (Eastell, 2005). Mechanical stress on the skeleton is critical to all these processes; the growing skeleton is more sensitive to loading than is the mature skeleton. Thus, physical activity during adolescence is necessary to maximize peak bone mass.

### Peak Bone Mass and Prevention of Osteoporosis

Loss of bone mass during adulthood is inevitable; thus, prevention of osteoporosis depends, in part, on maximizing peak bone mass, which is achieved in young adulthood. Because a large fraction of adult bone mass is acquired during adolescence, it is important to optimize skeletal growth during this stage in the life cycle. Strategies to maximize peak bone mass include adequate nutrition, regular weight-bearing exercise, and normal endocrine function to retain calcium and stimulate bone growth (Weaver, 2000).

## Hormonal Mediators of the Adolescent Growth Spurt

In healthy individuals, the timing of puberty onset and the adolescent growth spurt is genetically determined. However, multiple environmental factors, including nutrient availability, affect the rate at which puberty progresses. Normal physical growth and development during puberty depend on the integration of the growth hormone (GH)–insulin-like growth factor (IGF) and gonadotropin axes. Nutritional and metabolic signals, in part, control these hormonal systems by acting on the hypothalamus and pituitary gland.

### Fracture Risks

Although 75% to 85% of the variance in bone strength can be attributed to bone mass, the fragility of a bone also is determined by the shape of the bone, remodeling rates, and chemical properties of the bone. Most fractures occur later in life; thus, fracture risk depends not only on bone mass but on bone geometry, bone turnover, and risk of falling. Furthermore, the rate of bone loss during adulthood is accelerated by poor nutrient intake and lack of regular weight-bearing exercise (see Figure A). Therefore, acquisition of peak bone mass during adolescence does not guarantee that an individual will be protected against fractures during late adulthood (Schonau, 2004).

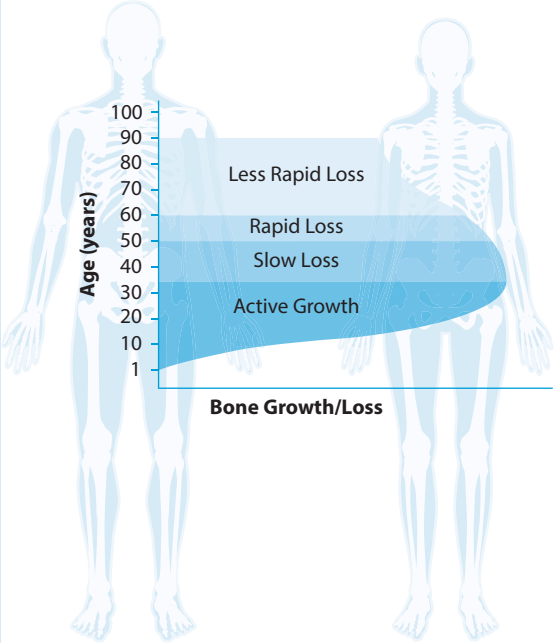



**FIGURE A** Bone loss over time

Adapted from U.S. Department of Health and Human Services. *The Surgeon General's Report on Bone Health and Osteoporosis: What It Means To You.* U.S. Department of Health and Human Services, Office of the Surgeon General, 2012.

### Pattern of Hormone Secretion

Pulsatile secretion of gonadotropin-releasing hormone (GnRH) occurs throughout the life span but is much more active during the perinatal period and adolescence. It is the pulsatile release of GnRH that stimulates pituitary release of leutinizing hormone (LH) and follicle-stimulating hormone (FSH), which in turn stimulate gonadal steroid hormone production in the testes and ovaries. During childhood, the activity of the hypothalamic-pituitary-gonadal axis is suppressed by the central nervous system. Animal data suggest that in females gamma-aminobutyric acid is responsible for the juvenile pause; in males **neuropeptide Y** prevents the onset of puberty. At puberty, excitatory neurotransmitters stimulate the release of GnRH in the hypothalamus. As a result, the pulse amplitude of the **gonadotropins**, LH and FSH, increases and the nocturnal rise in LH secretion is amplified. Pulse frequency, however, is unchanged during puberty. This nocturnal rhythm is specific to puberty and disappears in adulthood. Increased circulating concentrations of LH and FSH stimulate development of the gonads and production of sex steroid hormones. Androgens are synthesized in the Leydig cells of the testes, to a lesser degree in the ovaries, and are secreted in response to LH. Testosterone stimulates sperm protein synthesis and development of secondary sex characteristics. FSH stimulates release of estrogens from the ovaries. Progesterones are synthesized in the **corpus luteum** and secreted in response to LH. Estrogen and progesterone control the menstrual cycle and the development of the female secondary sex characteristics.

**neuropeptide Y** A 36-amino-acid peptide neurotransmitter found in the brain and autonomic nervous system; it augments the vasoconstrictor effects of noradrenergic neurons.

**gonadotropin** Hormones secreted by the pituitary gland that affect the function of the male or female gonads.

**corpus luteum** A yellow glandular mass in the ovary formed by an ovarian follicle that has matured and released its egg; secrets progesterone.

Prior to puberty, there is no sex difference in the activity of the GH–IGF-I axis. GH secretion is pulsatile, with the largest peak amplitudes during the early hours of sleep. A dramatic increase in spontaneous GH secretion precedes the onset of puberty. GH peak amplitude per pulse is elevated, and integrated 24-hour GH secretion (Styne, 2003) is increased 1.5- to 3-fold (Leung, Johannsson, Leong, & Ho, 2004). GH release stimulated by insulin, arginine, and other secretagogues also is elevated during puberty. Serum concentrations of IGF-I increase in parallel with increased GH secretion and are elevated threefold above prepubertal levels (Leung et al., 2004). Because the secretion of the acid-labile subunit and IGF binding protein 3 (IGFBP-3) do not match the increased production of IGF-I, free IGF-I concentrations also are elevated.

Sex differences in activation of the GH–IGF-I axis account for differences in peak height velocity. Girls show an increase in GH secretion at Tanner breast stage 2 with maximal GH at Tanner breast stage 3 to 4. By contrast, boys experience maximal GH secretion later during development—at Tanner genital stage 4. After sexual maturation is complete, but prior to attainment of adult body composition, GH and IGF-I production decreases (Rogol, 2010). GH-deficiency diseases illustrate the importance of the somatotropic axis in pubertal growth. Children

with GH deficiency are of short stature and have reduced growth velocity and delayed pubertal onset. Thus, normal function of the GH–IGF axis is needed for onset and maintenance of puberty and sexual development.

### Gender Hormones

The sex steroids, estrogen and testosterone, increase spontaneous and stimulated GH secretion. Estrogen appears to be primarily responsible for increased GH secretion associated with the pubertal growth spurt. The effects of testosterone are likely mediated through conversion to estradiol by the enzyme aromatase. Sex steroids also modulate IGF-I bioactivity via regulation of IGFBPs and proteases. IGF-I enhances gonadotropin-releasing hormone secretion in some species and augments the effects of LH and FSH on sex steroid production in the ovaries and testes both in vitro and in vivo (Veldhuis et al., 2005).

Leptin, a peptide hormone secreted by adipocytes, plays an essential role in pubertal development. Leptin does not initiate puberty but plays a permissive role, allowing pubertal development to proceed (Elias, 2012). Although the exact mechanism is unclear at this time, leptin is known to act on the GnRH neuronal system in the hypothalamus. Because leptin concentrations are proportional to body fat, leptin allows nutrient modulation of puberty. Evidence for the ability of leptin to activate the hypothalamic-pituitary-gonadal axis comes from administration of leptin to leptin-deficient individuals. In postmenarcheal women with hypothalamic amenorrhea, leptin treatment increased secretion of LH, FSH, and estradiol and stimulated follicular development independent of changes in body weight (Welt et al., 2004). In addition, individuals who are deficient in leptin or who have leptin receptor defects fail to go through puberty. Boys have lower serum leptin concentrations than do girls, and this gender difference increases throughout puberty. The lower leptin concentrations in males compared with females at a given body mass index (BMI) may be the result of the suppressive effects of androgens on leptin expression (Federico et al., 2003).

#### Hormone Actions on Skeletal Muscle

GH, IGF-I, and testosterone apparently have synergistic effects on anabolism in skeletal muscle, although the cellular mechanisms are not completely understood. Administration of GH or IGF-I to individuals with GH deficiency increases lean body mass by inhibition of protein breakdown and stimulation of amino acid uptake and protein synthesis. Testosterone replacement therapy at physiologic doses in hypogonadal boys and men causes an increase in lean tissue mass because of enhanced rates of protein synthesis and suppression of proteolysis. Testosterone reduces **myocyte** apoptosis via induction of **myostatin**. Some of the anabolic effects of testosterone are the result of induction of IGF-I and inhibition of IGFBP-4 expression in skeletal muscle. IGF-I promotes myoblast differentiation from satellite cells and causes hypertrophy of existing myocytes. Endogenous androgens likely drive muscle anabolism in pubertal girls, as well as in pubertal boys.

**myocyte** A single muscle cell.

**myostatin** A growth factor that limits muscle tissue growth.

#### Hormone Actions on Adipose

Total fat mass is under control of multiple hormones and neurotransmitters, including the sex steroids, GH, insulin, glucocorticoids, and **adrenergic agonists**. Testosterone, GH, and adrenergic agonists stimulate lipolysis; estrogen and insulin cause lipogenesis. Regional fat distribution is determined by tissue distribution of the sex steroid receptors. Adipocytes in the mammary gland and gluteofemoral region have estradiol receptors; intra-abdominal fat cells have androgen receptors. Testosterone has many lipolytic effects that may contribute to the changes in regional fat distribution that occur during puberty. Testosterone enhances the actions of GH and adrenergic agonists, stimulates androgen receptor expression, and reduces the lipogenic effects of insulin and lipoprotein lipase. In contrast, estrogen increases expression of insulin receptors in adipose tissue and may attenuate the lipolytic effects of GH.

**adrenergic agonists** Bind to and activate adrenergic receptors.

Exogenous female reproductive hormones administered orally or as intramuscular injections affect body composition in adolescent girls. Oral contraceptive agents and depot **medroxyprogesterone acetate** promote excessive adiposity in adolescent girls. In a longitudinal study of adolescents, depot medroxyprogesterone acetate use resulted in significantly greater weight gain after 18 months compared with oral contraceptives and no hormonal contraception in obese girls. Among nonobese teens, either form of hormonal contraception resulted in a greater incidence of obesity compared with control subjects (Bonny et al., 2006; Eastell, 2005).

**medroxyprogesterone acetate** A synthetic progestin.

### Hormone Actions on Bone

The sex steroids have opposing actions on bone. Androgens and estrogens stimulate both bone growth and fusion of the epiphysis. Individuals with subnormal levels of estrogen or testosterone at puberty have reduced peak bone mass, resulting in osteopenia or osteoporosis and increased fracture risk during adulthood. Some of the osteogenic effects of testosterone and estrogen are likely the result of increased pulsatile GH secretion, which is the primary stimulus of longitudinal bone growth; IGF-I synthesis in bone; increased intestinal absorption of calcium and magnesium; and increased osteoblast activity. Androgen receptors are present on osteoblasts and osteocytes. In vitro studies demonstrated that testosterone inhibits apoptosis of osteoblasts and osteocytes, suppresses osteoclastogenesis, and causes deposition of mineral on periosteal surfaces of cortical bone. The effects of testosterone may be partially mediated by estradiol via conversion of testosterone via aromatase. Aromatase deficiency in humans and animals causes osteoporosis, and aromatase antagonists increase bone resorption and impair pubertal bone mineralization.

Estrogens are important regulators of skeletal effects, exerting direct effects on bone and indirect effects via the GH–IGF-I system. The effects of estrogen on bone are biphasic: Low concentrations of estrogen stimulate bone growth and high concentrations reduce growth and cause closure of the growth plate (Wit & Camacho-Hubner, 2011). Thus, estrogen is responsible for the sexual dimorphism of bone, explaining why females have smaller skeletons relative to males. Estrogen, like testosterone, promotes bone growth by suppressing bone resorption and enhancing bone deposition. Estrogen stimulates proliferation and differentiation of osteoblasts as well as osteoblast secretion of osteoprotegerin. Osteoprotegerin is a glycoprotein that inhibits osteoclastogenesis. Estrogen also increases intestinal absorption of calcium, thereby indirectly enhancing bone mineralization. Another indirect effect of estrogen on bone growth is that it stimulates GH secretion, thereby also increasing IGF-I (Wit & Camacho-Hubner, 2011).

Age at **menarche** is associated with BMD and fracture risk. Delayed menarche is associated with low adult BMD and fracture risk. The relative risk of hip fracture was 2.1 in women who experienced menarche after age 17 years (Fujiwara, Kasagi, Yamada, & Kodama, 1997); the relative risk of vertebral fractures was 1.8 in women who began menstruating after age 16 years (Roy et al., 2003). There are two possible explanations for this finding (Eastell, 2005). The first hypothesis is that lower lifetime exposure to estrogen reduces BMD. The second explanation is that both early menarche and BMD result from greater body weight per height, and body weight is a strong predictor of BMD.

**menarche** First menstrual period.

**Learning Point** There are ethnic differences in BMD; African American teenagers have higher BMD than do white teenagers.

The IGF regulatory system, which includes GH, IGF-I, IGF-II, and IGFBPs, regulates bone modeling and growth. IGF-I concentrations are highest during puberty concurrent with the maximal rate of bone growth. GH stimulates IGF-I synthesis in bone, proliferation of prechondrocytes, and hypertrophy of osteoblasts. Hepatic IGF-I, which is stimulated by GH, is the primary source of IGF in circulation. Circulating IGF-I is important for longitudinal growth and bone modeling via periosteal expansion (Kawai & Rosen, 2012). In vitro experiments demonstrated that GH reduces production of IGFBP-4 in bone and increases synthesis of IGFBP-2, -3, and -5. IGFBP-4 inhibits the actions of IGF-I in bone, whereas IGFBP-2, -3, and -5 augment the effects of IGF-I.

## Assessment of Growth and Development

### Growth

Serial measurements of height and weight plotted on height for age, weight for age, and weight for height growth charts from the Centers for Disease Control and Prevention (2000) are used to evaluate growth statistics. Height growth potential is calculated from parental height. Skeletal age is assessed using radiography of the left hand and wrist. An open epiphysis indicates skeletal immaturity and potential for additional growth. Stature of pregnant adolescents should be assessed using a knee height–measuring device to determine the length of the lower leg. Vertebral compression during pregnancy caused by changes in posture (increased **lordosis**) and gestational weight gain makes stature an invalid measure of growth.

**lordosis** An abnormal forward curvature of the lumbar spine.

Height and weight are used to calculate body mass index (BMI, kg/m$^2$); BMI for age growth charts also are available. Obesity in adolescents is

defined as BMI greater than or equal to the 95th percentile; adolescents with BMI in the 85th to 95th percentiles are classified as overweight (Ogden & Flegal, 2010). Underweight is defined as BMI less than the 5th percentile.

#### Reproductive Development

Reproductive development is assessed using Tanner stages that characterize pubic hair and breast (female) and genital (male) maturation. Progression from Tanner stages 2 to 5 typically takes 2.5 to 5 years. In girls, Tanner staging of breasts and pubic hair usually occurs in parallel; for boys, genital staging precedes pubic hair staging. Menarche typically occurs at Tanner stage 4. Pubertal onset, including age at menarche, is in part genetically determined. The normal ages of pubertal onset are 8 to 16 years in girls and 9 to 17 years in boys.

### Adolescent Growth Disorders

#### Identification of Normal Short Stature

It is important to differentiate between an adolescent who is small relative to his or her peers and a teenager who is growing poorly. To make this distinction and to evaluate growth, serial determinations of height and weight are required. Two benign conditions that result in short stature are familial short stature and constitutional/maturational delay. Parental stature is used to assess growth potential. A child with constitutional delay experiences retarded growth at ages 3 and 11 to 12 years; this condition is often familial. Bone age, height age, and growth velocity can be used to differentiate familial short stature and constitutional delay. Adolescents with constitutional delay have delayed bone and height ages and slowed growth, whereas those with familial short stature have normal bone age, height age less than bone age, and normal growth (Simm & Werther, 2005). Chronologic age and bone age may differ by as much as 2 years and still be within the normal range. Puberty can be induced in boys older than 14 years by testosterone administration without compromising linear growth. Girls with extreme constitutional delay may be treated with low-dose estrogen. Because estrogen causes fusion of the growth plates, adult height may be compromised.

#### Pathologic Causes of Short Stature

Changes in weight trajectories also can be used to determine the cause of short stature because weight gain is affected before linear growth. A teenager whose weight percentile is declining may be suffering from chronic illness or poor dietary intake, possibly of psychosocial etiology. Other pathologic causes of short stature include endocrine disorders such hypothyroidism and GH deficiency or other disorders of the GH–IGF-I axis; intrauterine growth retardation; chromosomal defects, Turner, Down, and Prader-Willi syndromes; and skeletal dysplasia. Diagnosis of these disorders requires thyroid function, bone age, karyotype, and provocative GH testing. Some catch-up growth usually occurs after the underlying pathology is resolved. GH deficiency and Turner syndrome can be treated with GH.

#### Pathologic Causes of Tall Stature

Tall stature in adolescents is rare compared with short stature; most cases of tall stature are benign familial tall stature (Simm & Werther, 2005). Pathologic tall stature must be diagnosed with thyroid function tests, bone age, karyotyping, and determination of serum IGF-I concentrations. Endocrine causes of tall stature include hyperthyroidism, precocious puberty, and GH-secreting tumors. Adolescents with precocious puberty end up with compromised adult height because estrogen and androgen levels peak early, causing premature fusion of the growth plates. Klinefelter, Marfan, Sotos, and Beckwith-Widermann syndromes are rare genetic disorders that result in tall stature. Most adolescents with tall stature do not require medical intervention. Sex steroid treatment can be used to induce premature fusion of the epiphyseal growth plates, thereby reducing final height. However, estrogen treatment of tall stature may impair fertility; thus, it rarely is used (Simm & Werther, 2005).

## Cognitive and Psychosocial Development During Adolescence

### Cognitive and Affective Development

The brain develops during puberty; in particular, areas involved in regulation of behavior and emotion and in perception and evaluation of risk and reward undergo considerable change (Steinberg, 2005). Cognitive development during adolescence results in increased self-awareness, self-direction, and self-regulation. However, the changes in arousal and motivation that take place during puberty occur before acquisition of the ability to regulate **affect** (Steinberg, 2005).

**affect** Emotion.

The limbic areas of the brain associated with emotion increase in volume during puberty. The cortical gray area is reduced in volume, and there is localized synaptic pruning that increases the efficiency of information processing. Frontal brain activity, the location of abstract thought, planning, and attention, is increased (Waylen & Wolke, 2004). There is increased connectivity among brain regions via myelination of nerve fibers; in particular, increased connections between the cortex and the **limbic systems** augment the ability to evaluate and make decisions regarding risk and reward.

**limbic system** A group of brain structures and their connections with each other as well as their connections with the hypothalamus and other areas; largely associated with emotions.

During early adolescence, teenagers improve their deductive reasoning, information processing, and specialized knowledge. The capacity for abstract, multidimensional, planned, and hypothetical thought increases into middle adolescence. Although adults and adolescents have similar reasoning capacities, adolescents and adults differ in social and emotional factors, leading to divergent behaviors with age. For example, adolescents are more susceptible to peer influences than are adults, and their reasoning about real-life situations or personal choices is not as sound as their reasoning about hypothetical or ethical questions.

Age and life experience are associated with most aspects of cognitive development during adolescence; however, maturation in arousal, motivation, and emotion are more closely linked with the onset of puberty. Emotional intensity and reactivity and reward-seeking and risk-taking behaviors also are influenced by puberty and not by chronologic age. Vulnerability to social status appears to increase at pubertal onset, coinciding with increased risk-taking behaviors. Cognitive development enhances the ability to regulate affect; conversely, emotion alters decision-making thought processes and behavior. Because teenagers are more likely to engage in high-risk behaviors (e.g., drug use, unprotected sex), it previously was thought that their ability to accurately evaluate risk was lacking. However, there is evidence that adolescents can understand risk but make poor decisions, regardless. Thus, decision-making behaviors that affect health, including diet and exercise, are highly influenced by emotion and social influences, despite understanding of associated risks and benefits.

### Psychosocial Development

Adolescence is the transition from childhood to adulthood, requiring maturation of psychosocial functioning. During the teenage years, individuals move from same- to mixed-sex peer groups, develop romantic relationships, and transfer relational dependence and modeling of behaviors from parents to peers (Waylen & Wolke, 2004). Adolescence also is a time of increased independence with regard to academic performance and economic self-sufficiency. In the United States and other Western societies, physical maturity precedes achievement of these developmental tasks; this period of prolonged adolescence has been termed the "maturity gap." The length of the maturity gap differs significantly between genders and among individuals; the maturity gap is larger for boys than for girls.

The cognitive and affective development that occurs during adolescence changes a teenager's self-concept and self-esteem. Adolescents are concerned with the identity they project to others (Waylen & Wolke, 2004). The discrepancy between an adolescent's self-identity and the expectations of others may be problematic. Rapid pubertal development or pubertal onset that deviates from one's peers also may result in maladaptive behaviors. In girls, early maturation is associated with increased affective disorders, delinquency and drop-out rates, and pregnancy. In boys, early physical maturation has mostly positive consequences, namely, increased social status and high self-esteem. In contrast, males with late pubertal onset are more likely to engage in status-seeking antisocial behaviors. Thus, biological and social factors interact to affect behaviors, including those with long-term effects on health.

**Learning Point** Tobacco use, poor diet/inactivity, alcohol abuse, and illicit drug use are among the leading modifiable causes of death in the United States (McGinnis, 1993; Mokdad et al., 2000). Unfortunately, the prevalence of these risk factors is high among adolescents.

Drug use is one maladaptive coping mechanism that adolescents may adopt to deal with stress. Puberty is associated with the onset of mental health disorders in girls; in particular, the incidence of anxiety and depressive disorders, eating disorders, and smoking is increased in female adolescents. Among U.S. girls participating in the

National Longitudinal Study of Adolescent Health (Harris, Gordon-Larsen, Chantala, & Udry, 2006), 10% to 15% reported feelings of depression and 13% to 33% had suicidal thoughts. In contrast, boys were less likely to feel depressed (6% to 9%) or to report suicidal ideation (8% to 14%). Girls also are more likely to attempt suicide (Grunbaum et al., 2002), although males are more likely to complete suicide. This gender difference may be attributed to hormonal changes and to the female ruminative response style. Other risk factors for suicide attempt include lower self-esteem, alcohol misuse, marijuana use, delinquency, friend or family member with a history of suicide attempt or completion, physical or sexual abuse, and low connectedness with parents (Thompson & Light, 2011).

Use of alcohol and illicit drugs among adolescents varies by sex and age. Rates of binge alcohol use in 2011 were 1.1% among 12- or 13-year-olds, 5.7% among 14- or 15-year-olds, 15.0% among 16- or 17-year-olds, 31.2% among persons aged 18 to 20 years and peaked among those aged 21 to 25 years at 45.4% (Substance Abuse and Mental Health Services Administration [SAMSA], 2012). In a national sample of U.S. youth, 49.3% consumed at least one drink in the previous month. On average, adolescents aged 15 to 21 years consumed 14.3 drinks per month (Snyder, Milici, Slater, Sun, & Strizhakova, 2006). In 2011, 10.1% of youths aged 12 to 17 years were current illicit drug users. Marijuana was the most common illicit drug used by adolescents (7.9%), whereas psychotherapeutic drugs (2.8%), hallucinogens (0.9%), inhalants (0.9%), and cocaine (0.3%) were used by fewer teens. Use of illicit drugs was more common among older teens and young adults than among younger youth (SAMSA, 2012).

In 2011, the rate of current illicit drug use was higher among males aged 12 to 17 years than females aged 12 to 17 (10.8% vs. 9.3%). Males aged 12 to 17 years also were more likely than females to be current marijuana users (9.0% vs. 6.7%). However, females aged 12 to 17 years were more likely than males to be current nonmedical users of psychotherapeutic drugs (3.2% vs. 2.4%) and current nonmedical users of pain relievers (2.6% vs. 1.9%).

In 2011, 10.4% of male adolescents suffered from substance dependence or abuse compared with 5.7% of females. Among youths aged 12 to 17 years, there were 1.2 million persons (4.7%) who needed treatment for an illicit drug use problem and 978,000 who needed treatment for alcohol misuse (3.9%). Unfortunately, only 10% of individuals who needed help received treatment, leaving nearly 1 million teens with a substance abuse problem and another million who suffer from alcohol abuse without treatment (SAMSA, 2012).

Alcohol use, smoking, marijuana use, and truancy are co-occurring behaviors; teens who use marijuana are more likely to also use tobacco than are nonusers (Weden & Zabin, 2005). Regular or excessive alcohol consumption is positively associated with marijuana use (Boys et al., 2003; Rey, Martin, & Krabman, 2004). These behaviors are less likely to persist into adulthood if the onset occurs during adolescence, if the frequency of the behaviors is occasional, and if the behaviors occur within the peer group (Waylen & Wolke, 2004). In addition, parental involvement reduces tobacco use and binge drinking. Use of illicit drugs and cigarettes and binge alcohol consumption were lower among teens who reported that their parents always or sometimes engaged in monitoring behaviors than among youths whose parents seldom or never engaged in such behaviors (SAMSA, 2012).

Tobacco use, particularly cigarette smoking, also is common among adolescents. In 2011, about 10% of youths aged 12 to 17 years used tobacco (SAMSA, 2012) and approximately 2% used smokeless tobacco. In 2009, 5% of middle school students and 20% of high school students were cigarette smokers (Centers for Disease Control and Prevention [CDC], 2005). In 2011, 5% of males and females aged 12 to 17 years smoked their first cigarette (SAMSA, 2012). Each day approximately 4,000 children and adolescents younger than 18 years of age smoke their first tobacco cigarette. One-half of these teens become regular smokers (SAMSA, 2003). Teenagers perceive increased social status from cigarette smoking. Girls often smoke cigarettes to control their body weight. Adolescents also believe cigarette smoking is harmless during the first few years of smoking (Waylen & Wolke, 2004). Advertising increases risk of cigarette smoking by glamorizing smoking and by fostering brand recognition and desirability (CDC, 2005).

## Nutrient Requirements and Temporal Consumption Trends

Absolute nutrient requirements are increased in adolescence compared with childhood as a result of increased growth and body size. Adolescent boys have greater requirements for most nutrients compared with girls because of differences in growth and development. The exception is iron; postmenarcheal adolescent girls need more iron than do boys because of menstrual blood losses (Institute of Medicine [IOM] Food and Nutrition Board, 2001).

### Macronutrients

Average daily energy consumption assessed in National Health and Nutrition Examination Survey (NHANES) I (1971–1974) and III (1988–1994) has remained relatively constant (Briefel & Johnson, 2004; Troiano, Briefel, Carroll, & Bialostosky, 2000). Data from NHANES (2005–2006) reported that boys consumed 2,707 kilocalories (kcal) per day and girls 1,906 kcal/day (United States Department of Agriculture [USDA], Agricultural Research Service, 2008). The proportion of energy derived from fat and saturated fat decreased over time but remains above the recommendations in the *Dietary Guidelines* at approximately 33% for total fat and approximately 11% for saturated fat (USDA, Agricultural Research Service, 2008). In children older than 6 years of age, 10% to 18% of saturated fat and 5% to 10% of total fat was consumed in 2% fat and whole milk products.

### Minerals

#### Calcium

The Food and Nutrition Board of the IOM established the RDA for calcium based on maximal calcium retention using data from epidemiologic studies, randomized controlled trials, and balance studies (IOM Food and Nutrition Board, 2010). Calcium balance studies in adolescents determined that retention increased with dietary intake up to 1,300 mg/day. Recent longitudinal data of bone mineral deposition suggest that calcium requirements may be higher—1,500 mg for girls and 1,700 mg for boys—during peak calcium accretion (Whiting et al., 2004). Dairy foods supply 75% of the calcium in the American diet (Weaver, 2000). Some vegetables contain calcium, but the bioavailability is poor: 5 servings of broccoli and 15 servings of spinach provide as much absorbable calcium as 1 cup of milk (Weaver, 2000).

Adolescent boys aged 12 to 19 years consume, on average, the equivalent of 2.2 servings of dairy and 83% of the RDA for calcium (1,081 mg/day). Girls consume the equivalent of 1.7 servings of dairy and 61% of the RDA for calcium (793 mg/day) (Ervin, Wang, Wright, & Kennedy-Stephenson, 2004). There also are significant ethnic differences in calcium consumption; African American and Hispanic American teenagers have lower daily calcium intakes than do white teenagers (Storey, Forshee, & Anderson, 2004).

### CRITICAL Thinking

Milk consumption has declined 36% among teenage girls from the late 1970s to the mid-1990s. Teenage girls may avoid dairy products because they believe dairy products promote weight gain. Lactose intolerance is another reason that teenagers, especially nonwhites, do not consume dairy products. Consumption of soft drinks has been associated with reduced bone mineral deposition in adolescent girls with average calcium intakes less than 900 mg/day but not in boys, whose calcium intakes averaged more than 1,000 mg (Whiting et al., 2004). Noncaffeinated carbonated soft drinks do not increase urinary calcium losses (Heaney & Rafferty, 2001). Because soft drinks often replace milk, reduced calcium consumption likely explains the relationship between soft drinks and bone mineral. What effects will this have in the aging process?

#### Iron

Iron requirements increase during adolescence to meet the demands of growth and inevitable losses. Iron is lost from the gastrointestinal tract, skin, and urine and from menstrual blood in females. The requirements for absorbed iron for adolescent boys and girls for growth are estimated to be 1.47 and 1.15 mg/day, respectively (Fomon et al., 2003). The mean iron intake of adolescent boys aged 12 to 19 years is approximately 18 mg/day (Ervin et al., 2004). Thus, most boys probably meet the absorbed iron requirement. In contrast, girls average a daily intake of 13 mg (Ervin et al., 2004) and thus are at risk for iron deficiency.

#### Sodium

Average daily sodium consumption has increased by approximately 1,000 mg for adolescent boys and girls between NHANES I (1971–1974) and NHANES (1999–2000). The average daily sodium intake for teenage boys was about 4,000 mg and for girls, 3,000 mg (Ervin et al., 2004).

### Vitamins

#### Vitamin D

The Recommended Dietary Allowance (RDA) for vitamin D is 15 µg (600 IU) per day for individuals

aged 1 to 50 years (IOM Food and Nutrition Board, 2010). With sufficient exposure to ultraviolet light synthesis of vitamin D in the skin is adequate to meet the requirement. Skin vitamin D synthesis is reduced by use of sunscreen and by dark skin pigmentation. Recently, there have been increased reports of vitamin D insufficiency and deficiency, including reports of rickets among African American children and vitamin D deficiency in adolescents. Individuals with darkly pigmented skin have reduced ability to synthesize vitamin D and therefore are at greater risk for insufficiency when dietary intakes are marginal.

NHANES data (2001–2004) showed that more than 50% of children and adolescents in the United States are vitamin D insufficient or deficient. Alarmingly, among non-Hispanic black youth 59% of girls and 43% of boys were vitamin D deficient compared with 5% of non-Hispanic white girls and 3% of boys. Mexican American youth also had a very high prevalence of vitamin insufficiency (69% for girls and 79% for boys) and deficiency (20% for girls and 7% for boys). In addition to ethnic differences, obesity, time spent watching television or playing video games, and low milk consumption were associated with poor vitamin D status (Kumar, Muntner, Kaskel, Hailpern, & Malemed, 2009).

Dietary intake of vitamin D among adolescents varies by ethnicity; non-Hispanic American white teenagers had the highest intakes of vitamin D and African Americans the lowest (Moore, Murphy, & Holick, 2005). As a group, adolescent boys are more likely to have adequate vitamin D intakes than are girls and older males. However, there are significant ethnic differences; approximately 75% of non-Hispanic American white adolescents meet or exceed the recommendation, compared with 45% of non-Hispanic African Americans and 58% of Mexican Americans (Moore et al., 2005). Approximately one-half of adolescent girls meet or exceed the recommended intake for vitamin D; only 38% of non-Hispanic African Americans met the Adequate Intake (AI) compared with 59% of whites and 60% of Mexican Americans (Moore et al., 2005).

Fortified foods, mainly fortified milk, are the primary dietary sources of vitamin D. Because the prevalence of lactose intolerance is higher among African Americans (75%) than among Hispanic Americans (53%) and white Americans (6% to 22%), avoidance of dairy products may explain the reduced vitamin D intakes among African American adolescents (Jackson & Savaiano, 2001). In 2003, the U.S. Food and Drug Administration approved vitamin D fortification of calcium-fortified juices and juice drinks. African Americans and Mexican Americans who avoid dairy products may benefit from these fortified products; for example, 8 oz of fortified orange juice provides up to 2.5 µg (100 IU) of vitamin D.

In 2010, the recommended intakes for vitamin D were increased from 5 µg to 15 µg (IOM Food and Nutrition Board, 2010). Without ultraviolet synthesis of vitamin D, 20 to 25 µg (800 to 1,000 IU) of dietary vitamin D per day are needed to maintain serum vitamin D concentrations within the desired range (Dawson-Hughes, 2004). During peak pubertal growth, low serum concentrations of 25-hydroxyvitamin D increase serum **parathyroid hormone** and 1,25-dihydroxyvitamin D and, consequently, fractional calcium absorption (Abrams et al., 2005). In contrast to the relationship observed in adults, in adolescents 25-hydroxyvitamin D was not correlated with fractional calcium absorption. These findings suggest that adolescents adapt to suboptimal vitamin D status better than adults do. Possible mechanisms include enhanced responsiveness of calcium absorption to 1,25-dihydroxyvitamin D, increased conversion of 25-hydroxy to 1,25-dihydroxyvitamin D, or increased vitamin D–independent calcium absorption (Abrams et al., 2005).

**parathyroid hormone**
Peptide hormone secreted by the parathyroid gland; it increases blood calcium by acting on the bone, intestine, and kidneys.

### CRITICAL Thinking

Individuals who live at northern latitudes above the 35th parallel do not receive enough ultraviolet radiation to support adequate synthesis of vitamin D; between November and March vitamin D synthesis is dramatically reduced in the Northern Hemisphere (Moore et al., 2005). What effect does global warming have on this issue?

## Dietary Patterns

### Serving Size

Average serving sizes for foods eaten at home and away from home have increased during the past 30 years. The average serving sizes for foods frequently consumed by adolescents—salty snacks, french fries, ready-to-eat cereals, and soft drinks—also have significantly increased. The volume of soft drinks consumed per occasion increased more than 50% among female adolescents. Foods with low nutrient density, such as soft drinks, candy,

desserts, and salty snacks, and added sugar and fats account for 30% of daily energy intake in children aged 8 to 18 years (Briefel & Johnson, 2004). Energy from added sugar and solid fat accounts for nearly 1,000 kcal in the diets of adolescent boys and 760 kcal in the diets of girls (National Cancer Institute, 2010b). Consumption of these low-nutrient-density foods is associated with higher energy intakes and lower consumption of vitamin A, folate, calcium, magnesium, iron, and zinc.

### Food Groups

Adolescents do not consume the recommended number of servings of fruits, vegetables, and dairy products, and they consume excessive amounts of added sugar (Xie, Gilliland, Li, & Rockett, 2003), fat, and saturated fat (Briefel & Johnson, 2004). In a study of 18,000 adolescents who participated in the National Longitudinal Study of Adolescent Health, many did not eat the minimum recommended number of servings of vegetables (71%), fruits (55%), and dairy products (47%) (Videon & Manning, 2003). Teenagers who did not consume milk had inadequate intakes of vitamin A, folate, calcium, phosphorous, and magnesium (Briefel & Johnson, 2004).

### Skipping Breakfast

One-fifth of adolescents report skipping breakfast (Videon & Manning, 2003) and one study reported that adolescents eat breakfast, on average, 4 days per week (Niemeier, Raynor, Lloyd-Richardson, Rogers & Wing, 2006). Children who skip breakfast have lower daily intakes of vitamins A, E, D, and $B_6$; calcium; phosphorous; and magnesium compared with breakfast eaters (Nicklas, Bao, Webber, & Berenson, 1993) and frequency of breakfast skipping is associated with increased BMI (Niemeier et al., 2006). Eating breakfast is associated with increased school attendance and academic performance (Pollitt, 1995).

### Added Sugars

Adolescents have the highest intake of added sugars than any other age group; approximately 40% of added sugars are consumed in carbonated soft drinks (Briefel & Johnson, 2004). Soft drinks supply approximately 8% of total energy intake among adolescents. Overweight adolescents derive more of their daily energy intake from soft drinks than do normal-weight teens (Troiano et al., 2000). Teenagers consume more carbonated soft drinks than they do fruit juices, fruit ades, or milk. Consumption of soda and fruit drinks among adolescent girls doubled and milk consumption decreased between NHANES I (1971–1974) and NHANES III (1988–1994). Based on the U.S. Department of Agriculture (USDA) Continuing Survey of Food Intakes by Individuals for Adolescents age 12 to 17 years, consumption of sweetened breakfast cereals is positively associated with dietary intakes of calcium, folate, and iron and sweetened dairy products increase calcium intakes. However, consumption of other foods and beverages that are high in sugar was associated with poor diet quality, including reduced intakes of calcium, iron, and folate (Frary, Johnson, & Wang, 2004). Displacement of milk by high-sugar beverages reduces consumption of protein, calcium, and vitamins $B_2$, $B_{12}$, and D (St. Onge, Keller, & Heymsfield, 2003).

### Fast Food Consumption

Most adolescents frequently eat meals and snacks away from home. Fast food consumption is a likely contributor to the increase in adolescent obesity. In one study, 26% of all meals and snacks were consumed away from home, accounting for 32% of total energy (Lin, Guthrie, & Frazao, 1999). In another survey, 75% of adolescents reported eating at a fast food restaurant in the previous week (French, Story, Neumark-Sztainer, Fulkerson, & Hannan, 2003). In addition, fast food consumption appears to be increasing over time, and African American youth consume more fast food than white teens do (Niemeier et al., 2006). Teens who ate at a fast food restaurant at least three times in the past week had energy intakes about 40% higher than adolescents who did not eat fast food. In addition, fast food consumption was associated with increased BMI (Niemeier et al., 2006). Frequency of fast food consumption also is inversely related to daily servings of vegetables, fruit, and dairy products (French et al., 2001). Adolescents who described themselves as overweight were more likely to have poor dietary consumption patterns (Videon & Manning, 2003). Adolescent boys are more likely than girls to eat away from home (Briefel & Johnson, 2004). Teenagers who were employed ate fast food more frequently than those who did not work (French et al., 2001).

### Sociodemographic Moderators of Dietary Intake

Gender, ethnicity, parental income, and education affect diet quality in adolescents. Non-Hispanic American whites have the lowest intakes of fruit;

African Americans and Asian Americans have significantly higher consumption of vegetables (Xie et al., 2003). Degree of acculturation affects nutrient intake among Hispanic American youths. Adolescents whose families were less acculturated had lower intakes of energy, protein, sodium, and percentage of energy from fat and saturated fat and greater intakes of folate compared with acculturated families (Mazur, Marquis, & Jensen, 2003). Higher parental education level was associated with better diet quality, and the presence of a parent at the evening meal was positively associated with consumption of fruits, vegetables, and dairy products. Another study of 3,200 adolescents reported higher intakes of polyunsaturated fat, protein, calcium, and folate in teenagers from families with higher incomes and lower intakes of total fat, saturated fat, and cholesterol with higher parental education (Xie et al., 2003). Snacking is more common among families with higher incomes, and income modifies the selection of snack foods. Family income at or below 130% of the poverty line reported higher intakes of fried potatoes, potato chips, whole milk, and fruit drinks than those with incomes at or above 300% of the poverty line (Briefel & Johnson, 2004).

As children make the transition from dependence on their parents to autonomy, normative family development includes increased frequency and intensity of parent–child conflicts. During this time, peer influence on values and behavior becomes stronger as peers replace parents as the primary source of social support. Teenagers may use diet as a means of expressing their self-identity and autonomy. Peers may exert either positive or negative influences on food intake.

Advertising and marketing of foods and beverages influences the food preferences, purchase requests, purchase, and consumption of children and youth (IOM Food and Nutrition Board, 2006). Television is the primary source of advertising, but other forms of marketing such as radio, print, billboards, and the Internet also are used to influence purchases by adolescents. Most products marketed to children and teenagers are high in energy, sugars, fat, and salt and low in micronutrients. Children are more vulnerable to advertising than teenagers because they cannot differentiate between informative and commercial programming. There is strong evidence that advertising influences food preferences, purchase requests, and short-term food consumption of children 2 to 11 years of age. However, television advertising is positively correlated with body fat in children and adolescents (IOM Food and Nutrition Board, 2006). In addition, "screen media" have been implicated as playing a causative role in childhood and adolescent obesity as a result of the reduction in physical activity and food advertising (Hingle & Kunkel, 2012). Television and video viewing are associated with reduced consumption of fruits and vegetables in adolescents (Boynton-Jarrett et al., 2003).

### Influence of Advertising

Advertising influences the consumption of alcoholic beverages by adolescents. Exposure to alcohol advertising on television, radio, magazines, and billboards increased alcohol consumption in a dose-dependent manner in individuals aged 15 to 20 years. Each additional advertisement seen increased the number of drinks consumed by 1% (Snyder et al., 2006).

## School Food Environment

The recent increase in excessive weight and obesity in adolescents has brought the food environment in schools under increasing scrutiny. Most schools have "competitive" foods for sale in addition to the USDA National School Breakfast and Lunch Programs that provide free and reduced-cost meals to students from low-income families. The USDA meals programs are subject to federal regulations, including nutrient standards (USDA, 2012). Specifically, school lunches must provide one-third the RDA for protein, calcium, iron, vitamin A, and vitamin C (based on weekly averages), and intake of fat, saturated fat, and sodium must be consistent with the *Dietary Guidelines*. Despite these regulations, the meals offered by these programs have been reported to exceed the USDA's recommendations for fat (38% vs. 30% of total energy), saturated fat (15% vs. 10% of total energy), and sodium (~1,500 mg per meal vs. 2,400 mg/day).

Competitive foods include those that are sold à la carte, in vending machines, from school stores, or by school fundraisers. The only federal regulation regarding competitive foods is restriction of the sale of "foods of minimal nutritional value" (e.g., carbonated beverages, water ices, chewing gum, and some candy) in the lunch rooms during meals (French, 2005). Fruits, fruit juices, and vegetables are rarely offered for sale in school stores or in vending machines (St. Onge et al., 2003). Concern about the increasing incidence of excessive weight in children has caused some states and

local school districts to regulate competitive foods through legislation and policy.

Sales of à la carte foods and vending machine purchases are significant in middle and high schools, and many of the available selections are high in fat and/or added sugar (Kann, Grunbaum, McKenna, Wechsler, & Galuska, 2005). The School Health Profiles assessment of schools from 27 states and 11 large urban school districts found that nearly 90% of districts offered competitive foods. The most prevalent types of less nutritious foods were chocolate candy (65% of schools); other candy (68%); salty snacks, not low in fat (75%); and soft drinks, sports drinks, or fruit drinks (95%). Low-fat salty snacks (80%) and 100% fruit juice (84%) were the most common choices with higher nutritional value; fruits and vegetables were offered by 45% of schools surveyed (Kann et al., 2005). In a survey of public high schools in Pennsylvania, the top-selling items were pizza, hamburgers, or sandwiches; baked goods not low in fat; french-fried potatoes; salty snacks not low in fat; carbonated beverages, sports drinks, and fruit drinks; and bottled water (Probart et al., 2005).

Availability of competitive foods reduces diet quality (French & Stables, 2003). Interventions to the school food environment designed to promote consumption of fruits and vegetables significantly increase fruit intake with a negligible effect on vegetable consumption (French & Stables, 2003). Increased availability of fruit and fruit juices effectively increases intake. Reducing the cost of healthy food items offered in school snack bars and vending machines increases the purchases of these items. Alternatively, increasing the cost of higher fat "junk" foods reduces purchase and consumption of these foods (French, 2005). Other innovative strategies include school gardening programs, use of produce from local farmers markets in school salad bars, and free fruit and vegetables.

## Health Status of U.S. Adolescents

### Excessive Weight and Obesity

The prevalence of obesity (i.e., > 95th percentile for BMI on 2000 CDC growth charts) among adolescents has increased from 6.1% in NHANES I (1974–1976) to 15.5% in NHANES (1999–2000). The most recent data show that 19.3% of adolescent males and 16.8% of females are obese (Ogden, Lamb, Carroll, & Flegal, 2010). The increase in obesity is greatest in African American and Mexican American teens; the prevalence of excessive weight approaches 25% in these ethnic groups (Moore, 2004). Adolescents from lower-income households are twice as likely to be overweight or obese compared with those from higher-income families.

The increase in overweight and obesity in adolescents has been attributed to a dietary shift away from fruits and vegetables toward fats and simple carbohydrates and to decreased energy expenditure in physical activity. Among children aged 12 to 19 years, only 50% report regular vigorous physical activity and 25% do not get any vigorous exercise (Moore, 2004). Only 8% of American youth meet the public health guideline for physical activity of 60 minutes per day (Troiano et al., 2008). The time spent in sedentary activities also is increasing among adolescents. In 1997, parents reported that their children (aged 2 to 17 years) watched an average of 2.1 hours of television per day; by 2000, the average time spent watching television increased to 2.45 hours per day, and a total of 382 minutes were spent watching television, playing video games, using a computer, talking on the telephone, or reading (Ford, Mokdad, & Ajani, 2004).

Excess adiposity during adolescence increases the risk of adult obesity and chronic diseases. In the National Longitudinal Study of Adolescent Health of 9,795 adolescents, baseline prevalence of obesity was 12.5%, and only 1.6% of adolescents who were obese between ages 13 and 20 years were not obese 5 years later (Ford et al., 2004). Non-Hispanic African American females were more likely to become and remain obese than were non-Hispanic American white females; Asian women were less likely to become and remain obese (Ford et al., 2004). The risk of hypertension is increased 10-fold, dyslipidemias 3- to 8-fold, and diabetes mellitus 2-fold by excess adiposity. Surprisingly, only 39.6% of adolescents aged 12 to 15 years and 51.6% of older teens aged 16 to 19 years were ever informed of their overweight status by a physician or healthcare provider (Ogden, 2005).

### Metabolic Syndrome

The comorbidities of diabetes, hypertension, and hyperlipidemia in patients with cardiovascular disease have been described as the metabolic syndrome. Using the guidelines from NHANES III, the metabolic syndrome is defined as having three or more of the following risk factors: abdominal

obesity (i.e., waist circumference in or above the 90th percentile for age and gender), triglycerides at least 110 mg/dL, fasting glucose at least 110 mg/dL, systolic or diastolic blood pressure at least in the 90th percentile (age, height, gender specific), and no more than 40 mg/dL high-density lipoprotein for males and no more than 50 mg/dL for females.

Data from NHANES III of 1,366 adolescents aged 12 to 17 years found the prevalence of the metabolic syndrome to be 6.3% among boys and 4.1% among girls. Abdominal obesity, assessed by waist circumference, was identified in 17.9%, hypertriglyceridemia was present in 21.0%, 18.0% had low high-density lipoprotein, and 7.1% had hypertension. Furthermore, the prevalence of elevated C-reactive protein was nearly four times greater in adolescents with the metabolic syndrome compared with those without it (Ford, Ajani, Mokdad, & National Health and National Examination, 2005).

Using slightly different criteria for the metabolic syndrome (i.e., waist circumference >75th percentile and high-density lipoprotein <45 mg/dL for adolescent boys), the prevalence of the metabolic syndrome was 9.2% in 1,960 adolescents aged 12 to 19 years and was comparable among boys and girls (de Ferranti et al., 2004). However, a clinic-based sample of 251 children and adolescents reported an overall prevalence of 21% (Lee, Bacha, Gungor, & Arslanian, 2008). Risk of the metabolic syndrome is approximately 15 times higher in obese compared with lean youth (Tailor, Peeters, Norat, Vineis, & Romaguera, 2010).

There were small changes in risk factors for the metabolic syndrome in children and adolescents between NHANES III and NHANES (1999–2000): waist circumference increased by 2.0 cm in children aged 2 to 17 years, systolic blood pressure increased 2.2 mm Hg in children aged 8 to 17 years, and triglycerides decreased by 8.8 mg/dL and glucose by 2.5 mg/dL in teens aged 12 to 17 years. Total cholesterol, high-density lipoprotein and low-density lipoprotein cholesterol, and glycosylated hemoglobin were unchanged (Ford et al., 2004).

### Other Health Issues

Among overweight children, the metabolic syndrome was nearly three times as common, with a prevalence of 31.2%. Non-Hispanic African Americans (2.5%) were less likely to have the metabolic syndrome than were non-Hispanic American whites (10.9%) and Mexican Americans (12.9%) (de Ferranti et al., 2004).

### Iron Deficiency

Approximately 9% of adolescent girls aged 12 to 15 years and 16% of girls aged 16 to 19 years are iron deficient (Looker, 2002). Iron-deficiency anemia affects 2% of girls aged 12 to 19 years. The prevalence of iron deficiency among males 12 to 15 years is 5%. Iron deficiency causes reduced exercise capacity, immune function, and cognitive performance. Adolescent girls who were iron deficient scored lower in verbal learning and memory (Ford et al., 2004) and in math (Halterman, Kaczorowski, Aligne, Auinger, & Szilagyi, 2001) compared with those with normal iron status. Iron deficiency also may contribute to attention deficit hyperactivity disorder. Children with attention deficit hyperactivity disorder have significantly lower concentrations of ferritin than do control subjects (Konofal, Lecendreux, Arnulf, & Mouren, 2004).

### Tobacco Use and Nutritional Status

In 2011, approximately 10% of adolescents aged 12 to 17 years smoked. Children are starting to smoke at a younger age, and rates of regular tobacco use increase significantly at age 11 years (DuRant & Smith, 1999; Winkleby, Robinson, Sundquist, & Kraemer, 1999). Non-Hispanic American white adolescents are more likely to smoke cigarettes than are African American, Mexican American, and Native American teens (CDC, Division of Adolescent and School Health & Office on Smoking and Health, 1999). Most adolescents smoke to control their weight, and some substitute smoking a cigarette for eating (Dowdell & Santucci, 2004).

Adult smokers have significantly higher intakes of energy, total fat, saturated fat, cholesterol, and alcohol and lower intakes of polyunsaturated fat, fiber, vitamin C, vitamin E, and beta-carotene than nonsmokers (Dallongeville, Marécaux, Fruchart, & Amouyel, 1998). Similarly, adolescent smokers consume less fiber, vitamins, and minerals than do nonsmokers (Hampl & Betts, 1999). The more a teenager smokes, the less likely he or she is to exercise at least three times per week and consume at least one serving per day of vegetables or milk/dairy products (Wilson et al., 2005). Adolescents who smoke consume more alcohol than nonsmokers do (Crawley & While, 1996). For some teens who smoke, their poor diet may reflect the poor diet quality of their parents, who also smoke. Nonsmoking children of parents who smoke had lower intakes of folate and vitamins C and E than did children whose parents do not smoke

(Crawley & While, 1996). Among young women, passive and active smokers had lower intakes of folate and concentration of folate in serum compared with nonsmokers and values reported in 1999–2000 NHANES (Ortega et al., 2004).

Data from NHANES III show that environmental tobacco smoke exposure and active cigarette smoking increase the risk of the metabolic syndrome among adolescents. Teens exposed to environmental tobacco smoke were 4.7 times as likely to develop the metabolic syndrome compared with nonexposed adolescents; the odds ratio for active smokers was 6.1 (Weitzman et al., 2005). Among overweight adolescents, 19.6% of those exposed to environmental tobacco smoke and 23.6% of smokers had the metabolic syndrome compared with 5.6% of nonexposed teens (Weitzman et al., 2005). Unfortunately, 43% of children in the United States live in a household with at least one smoker. Alcohol and marijuana users eat more snack foods and less fruit, vegetables, and milk than nonusers (Farrow, Rees, & Worthington-Roberts, 1987).

### Adolescent Pregnancy

There are approximately 0.9 million adolescent pregnancies in the United States per year; more than 40% of adolescent girls have been pregnant at least once before 20 years of age (Kirby, Coyle, & Gould, 2001). About 51% of these pregnancies end in live births. Pregnant adolescents are more likely to have nutritional and medical complications than adult women are; the risk is greatest for the youngest teenagers. Teenagers are twice as likely to deliver a low-birth-weight infant and the rate of neonatal death is increased nearly threefold compared with the rate for adult women. Premature delivery is more common in teenagers. Inadequate and excessive maternal weight gain (Howie, Parker, & Schoendorf, 2003), pregnancy-induced hypertension, and anemia are common nutrition-related problems among pregnant adolescents (Klein, 2005). Low prepregnancy body weight and **gynecologic age**, parity, and poor nutritional status negatively affect pregnancy outcome (Klein, 2005). Smoking tobacco or marijuana, behaviors that are relatively common among adolescents, reduces birth weight (Fergusson & Woodward, 2000).

**gynecologic age** Years since menarche.

To optimize infant birth weight the IOM (1990) recommended that pregnant adolescents gain weight at the upper end of the recommended rates for each prepregnancy BMI category. However, the recommendation for greater weight gain in adolescents is controversial because it may increase the risk of maternal obesity (McAnarney & Stevens-Simon, 1993). Early and continuous weight gain is also important (American Dietetic Association, 1994; Kaiser & Allen, 2008). Inadequate weight gain in the first 24 weeks increases the risk of having a small for gestational age infant; low rates of weight gain late in gestation increase the risk of premature delivery (Hediger, Scholl, Belsky, Ances, & Salmon, 1989).

In addition to gestational weight gain, maternal growth is an important determinant of birth weight. Among teens who are still growing, gestational weight gain is positively associated with increased maternal subcutaneous adiposity and not with birth weight (Scholl, Hediger, Schall, Khoo, & Fischer, 1994). Growing adolescents give birth to infants who weigh 150 to 200 g less than the infants birthed by nongrowing teens or adult women. Reduced placental perfusion and transfer of micronutrients to the fetus explain, at least in part, the lower birth weight.

**Learning Point** An increase in leptin during gestation may mediate the increased maternal adiposity and fetal growth restriction in growing adolescents (Scholl, Stein, & Smith, 2000). Leptin also may mediate the increased postpartum weight retention observed in growing adolescents compared with nongrowing teens (Scholl et al., 2000).

Nutrition is one of the most important modifiable factors that alters pregnancy outcome. Adolescents may improve their diet quality during pregnancy, but total energy and iron intakes often are inadequate (American Dietetic Association, 1994). Suboptimal intakes of magnesium, zinc, folate, and vitamin $B_6$ also have been reported among pregnant adolescents (Pope, Skinner, & Carruth, 1992). Calcium, vitamin $B_6$, vitamin C, and folate supplements are recommended for teens at risk for poor dietary intakes (American Dietetic Association, 1994). Food cravings and aversions to previously preferred foods are common among adolescents. Cravings for sweet foods increase the consumption of sugar, and craving salty snacks is associated with increased consumption of sodium and fat (Pope et al., 1992).

### Body Dissatisfaction, Dieting, and Eating Disorders

Nearly 30% of high school students believe they are overweight; girls are more likely to believe they are overweight (35%) than boys (23%), who often

perceive themselves to be underweight. More than one-half of female students (62%) were trying to lose weight compared with 29% of males. Exercise is the most common weight control behavior (60% of all students), followed by dieting (44%), fasting (14%), diet pills (9%), and vomiting or laxative use (5%). All weight-control behaviors are more prevalent among female adolescents than among males (Grunbaum et al., 2002). A prospective, longitudinal study showed that, for girls, feeling overweight is associated with decreasing self-esteem and increased depressive symptoms and use of weight-control behaviors is also associated with increased depression (Vogt Yuan, 2010).

Eating disorders are psychiatric disorders with significant medical and psychosocial complications that affect about 5 million Americans, primarily adolescent girls and young women. Three eating disorders have been identified by the American Psychiatric Association: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified; the diagnostic criteria are outlined in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (American Psychiatric Association, 2000). The eating disorders share a factor that excessive importance is placed on body weight and shape that affects self-concept and self-esteem.

The estimated combined prevalence of anorexia nervosa and bulimia nervosa in the United States is 3% among young women and an additional 3% have eating disorder not otherwise specified, affecting about 7 million American women and 1 million men (Becker, Grinspoon, Klibanski, & Herzog, 1999). The incidence of anorexia nervosa is 19 females and 2 males per 100,000 per year compared with 29 females and 1 male for bulimia nervosa (Fairburn & Harrison, 2003). It is thought that the incidence of both anorexia nervosa and bulimia nervosa has increased in the recent past. It is very difficult to determine the frequency of eating disorders accurately in the general population (Agras et al., 2004).

Because of the reluctance to seek treatment, many eating disorders go undiagnosed in the community. In addition, there is bias in access to treatment, which in turn biases the reported prevalence of eating disorders among different ethnic and racial groups and between the genders. There is very little epidemiologic data regarding eating disorder not otherwise specified, although these atypical eating disorders seem to primarily affect adolescents and young adult women (Fairburn & Bohn, 2005).

## Issues to Debate

1. If girls experience peer pressure to eat like other teens, including boys, what might be the result?
2. Discuss how psychosocial development during adolescence affects health-related behaviors, including dietary patterns.
3. What are the sociodemographic factors affecting dietary patterns? How does this affect a pregnant teen?
4. What are the dietary implications to chronic disease incidence among adolescents?

## Case Study

### Adolescent Nutrition

Pamela S. Hinton, PhD

Anne is unhappy with her body shape and weight. As a child, Anne was "chubby" and was often teased for being overweight. After puberty, Anne was a normal weight for her height. Anne is 5 foot 8 inches tall and from her sophomore year in high school until she began her senior year she weighed 145 pounds. At the beginning of her senior year, she began to restrict her food intake by skipping breakfast and lunch. At the end of the first semester, Anne now weighs 110 pounds. She is not satisfied with her weight or the shape of her body and complains about feeling "fat." Anne has been amenorrheic for 4 months. Occasionally (about once a month), she becomes so hungry that she finds herself consuming large amounts of food and feels out of control while eating. Afterward, Anne is so consumed with guilt she does not eat anything for the next 24 hours and increases her exercise to "make up" for the extra calories.

#### Questions

1. What symptoms of an eating disorder does Anne exhibit?
2. What diagnosis would you give Anne?
3. What treatment recommendation would you make?
4. What psychosocial factors increase the risk of eating disorders in adolescents?

## References


Abrams, S. A., Griffin, I. J., Hawthorne, K. M., Gunn, S. K., Gundberg, C. M., & Carpenter, T. O. (2005). Relationships among vitamin D levels, parathyroid hormone, and calcium absorption in young adolescents. *Journal of Clinical Endocrinology and Metabolism, 90*, 5576–5581.

Agras, W. S., Brandt, H. A., Bulik, C. M., Dolan-Sewell, R., Fairburn, C. G., Halmi, K. A., … Wilfley, D. E. (2004). Report of the National Institutes of Health workshop on overcoming barriers to treatment research in anorexia nervosa. *International Journal of Eating Disorders, 35*, 509–521.

American Dietetic Association. (1994). Position of the American Dietetic Association: Nutrition care for pregnant adolescents. *Journal of the American Dietetic Association, 94*, 449–450.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Becker, A. E., Grinspoon, S. K., Klibanski, A., & Herzog, D. B. (1999). Eating disorders. *New England Journal of Medicine, 340*, 1092–1098.

Bonny, A. E., Ziegler, J., Harvey, R., Debanne, S. M., Secic, M., & Cromer, B. (2006). Weight gain in obese and non-obese adolescent girls initiating depot medroxyprogesterone, oral contraceptive pills, or no hormonal contraceptive method. *Archives of Pediatric and Adolescent Medicine, 160*, 40–45.

Boynton-Jarrett, R., Thomas, T. N., Peterson, K. E., Wiecha, J., Sobol, A. M., & Gortmaker, S. L. (2003). Impact of television viewing patterns on fruit and vegetable consumption among adolescents. *Pediatrics, 112*, 1321–1326.

Boys, A., Farrell, M., Taylor, C., Marsden, J., Goodman, R., Brugha, T., … Meltzer, H. (2003). Psychiatric morbidity and substance use in young people aged 13–15 years: Results from the Child and Adolescent Survey of Mental Health. *British Journal of Psychiatry, 182*, 509–517.

Briefel, R. R., & Johnson, C. L. (2004). Secular trends in dietary intake in the United States. *Annual Review of Nutrition, 24*, 401–431.

Bryant, R. J., Wastney, M. E., Martin, B. R., Wood, O., McCabe, G. P., Morshidi, M., … Weaver, C. M. (2003). Racial differences in bone turnover and calcium metabolism in adolescent females. *Journal of Clinical Endocrinology and Metabolism, 88*, 1043–1047.

Centers for Disease Control and Prevention. (2000). Growth Charts. Retrieved from www.cdc.gov/growthcharts/clinical_charts.htm

Centers for Disease Control and Prevention. (2005). Tobacco use, access, and exposure to tobacco in media among middle and high school students—United States, 2004. *Morbidity and Mortality Weekly Report, 54*, 297–301.

Centers for Disease Control and Prevention, Division of Adolescent and School Health & Office on Smoking and Health. (1999). Cigarette smoking among high school students—11 states, 1991–1997. *Journal of School Health, 69*(8), 303–306.

Crawley, H. F., & While, D. (1996). Parental smoking and the nutrient intake and food choice of British teenagers aged 16–17 years. *Journal of Epidemiology and Community Health, 50*, 306–312.

Dallongeville, J., Marécaux, N., Fruchart, J. C., & Amouyel, P. (1998). Cigarette smoking is associated with unhealthy patterns of nutrient intake: A meta-analysis. *Journal of Nutrition, 128*, 1450–1457.

Dawson-Hughes, B. (2004). Racial/ethnic considerations in making recommendations for vitamin D for adult and elderly men and women. *American Journal of Clinical Nutrition, 80*, 1763S–1766S.

de Ferranti, S. D., Gauvreau, K., Ludwig, D. S., Neufeld, E. J., Newburger, J. W., & Rifai, N. (2004). Prevalence of the metabolic syndrome in American adolescents: Findings from the Third National Health and Nutrition Examination Survey. *Circulation, 110*, 2494–2497.

Dowdell, E. B., & Santucci, M. E. (2004). Health risk behavior assessment: Nutrition, weight, and tobacco use in one urban seventh-grade class. *Public Health Nursing, 21*, 128–136.

DuRant, R. H., & Smith, J. A. (1999). Adolescent tobacco use and cessation. *Primary Care, 26*, 553–575.

Eastell, R. (2005). Role of oestrogen in the regulation of bone turnover at the menarche. *Journal of Endocrinology, 185*, 223–234.

Egan, S. K., Tao, S. S. H., Pennington, J. A. H., & Bolger, P. M. (2002). US Food and Drug Administration's Total Diet Study: Intake of nutritional and toxic elements, 1991–96. *Food Additives and Contaminants, 19*, 103–125.

Elias, C. F. (2012). Leptin action in pubertal development: Recent advances and unanswered questions. *Trends in Endocrinology and Metabolism*, *23*, 9–15.

Ervin, R. B., Wang, C.-Y., Wright, J. D., & Kennedy-Stephenson, J. (2004, April 27). Dietary intake of selected minerals for the United States population: 1999–2000. *Advanced Data*, 1–5.

Fairburn, C. G., & Bohn, K. (2005). Eating disorder NOS (EDNOS): An example of the troublesome "not otherwise specified" (NOS) category in *DSM-IV*. *Behavioral Research Therapy, 43*, 691–701.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *Lancet, 361*, 407–416.

Farrow, J. A., Rees, J. M., & Worthington-Roberts, B. S. (1987). Health, developmental, and nutritional status of adolescent alcohol and marijuana abusers. *Pediatrics, 79*, 218–223.

Federico, G., Baroncelli, G. I., Vanacore, T., Fiore, L., & Saggese, G. (2003). Pubertal changes in biochemical markers of growth. *Hormone Research, 60*, 46–51.

Fergusson, D. M., & Woodward, L. J. (2000). Educational, psychosocial, and sexual outcomes of girls with conduct problems in early adolescence. *Journal of Child Psychology and Psychiatry, 41*, 779–792.

Fomon, S. J., Drulis, J. M., Nelson, S. E., Serfass, R. E., Woodhead, J. C., & Ziegler, E. E. (2003). Inevitable iron loss by human adolescents, with calculations of the requirement for absorbed iron. *Journal of Nutrition, 133*, 167–172.

Ford, E. S., Ajani, U. A., Mokdad, A. H., & National Health and National Examination. (2005). The metabolic syndrome and concentrations of C-reactive protein among U.S. youth. *Diabetes Care, 28*, 878–881.

Ford, E. S., Mokdad, A. H., & Ajani, U. A. (2004). Trends in risk factors for cardiovascular disease among children and adolescents in the United States. *Pediatrics, 114*, 1534–1544.

Frary, C. D., Johnson, R. K., & Wang, M. Q. (2004). Children and adolescents' choices of foods and beverages high in added sugars are associated with intakes of key nutrients and food groups. *Journal of Adolescent Health, 34*, 56–63.

French, S. A. (2005). Public health strategies for dietary change: Schools and workplaces. *Journal of Nutrition, 135*, 910–912.

French, S. A., & Stables, G. (2003). Environmental interventions to promote vegetable and fruit consumption among youth in school settings. *Preventive Medicine, 37*, 593–610.

French, S. A., Story, M., Fulkerson, J. A., & Gerlack, A. F. (2003). Food environment in secondary schools: À la carte, vending machines, and food policies and practices. *American Journal of Public Health, 93*, 1161–1167.

French, S. A., Story, M., Neumark-Sztainer, D., Fulkerson, J. A., & Hannan, P. (2001). Fast food restaurant use among adolescents: Associations with nutrient intake, food choices

and behavioral and psychosocial variables. *International Journal of Obesity and Related Metabolic Disorders, 25*, 1823–1833.

Fujiwara, S., Kasagi, F., Yamada, M., & Kodama, K. (1997). Risk factors for hip fracture in a Japanese cohort. *Journal of Bone and Mineral Research, 12*, 998–1004.

Grunbaum, J. A., Kann, L., Kinchen, S. A., Williams, B., Ross, J. G., Lowry, R., & Kolbe, L. (2002). Youth risk behavior surveillance—United States, 2001. *Journal of School Health, 72*, 313–328.

Guo, S. S., Chunlea, W. C., Roche, A. F., & Siervogel, R. M. (1997). Age- and maturity-related changes in body composition during adolescence into adulthood: The Fels Longitudinal Study. *International Journal of Obesity and Related Metabolic Disorders, 21*, 1167–1175.

Halterman, J. S., Kaczorowski, J. M., Aligne, C. A., Auinger, P., & Szilagyi, P. G. (2001). Iron deficiency and cognitive achievement among school-aged children and adolescents in the United States. *Pediatrics, 107*, 1381–1386.

Hampl, J. S., & Betts, N. M. (1999). Cigarette use during adolescence: Effects on nutritional status. *Nutrition Reviews, 57*, 215–221.

Harris, K. M., Gordon-Larsen, P., Chantala, K., & Udry, J. R. (2006). Longitudinal trends in race/ethnic disparities in leading health indicators from adolescence to young adulthood. *Archives of Pediatric and Adolescent Medicine, 160*, 74–81.

Heaney, R. P., & Rafferty, K. (2001). Carbonated beverages and urinary calcium excretion. *American Journal of Clinical Nutrition, 74*, 343–347.

Hediger, M. L., Scholl, T. O., Belsky, D. H., Ances, I. G., & Salmon, R. W. (1989). Patterns of weight gain in adolescent pregnancy: Effects on birth weight and preterm delivery. *Obstetrics and Gynecology, 74*, 6–12.

Hingle, M., & Kunkel, D. (2012). Childhood obesity and the media. *Pediatric Clinics of North America*, *59*, 677–692, ix.

Howie, A., Parker, J., & Schoendorf, K. (2003). Excessive maternal weight gain patterns in adolescents. *Journal of the American Dietetic Association, 103*, 12, 1653–1657.

Institute of Medicine. (1990). *Nutrition during pregnancy. Part I. Weight gain. Part II. Supplements*. Washington, DC: The National Academies Press.

Institute of Medicine Food and Nutrition Board. (1997). *Dietary Reference Intakes for calcium, magnesium, phosphorous, vitamin D*. Washington, DC: National Academy of Sciences.

Institute of Medicine Food and Nutrition Board. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, nickel, silicon, vanadium, and zinc*. Washington, DC: The National Academies Press.

Institute of Medicine Food and Nutrition Board. (2006). *Food marketing to children and youth: Threat or opportunity?* Washington, DC: The National Academies Press.

Institute of Medicine Food and Nutrition Board. (2010). *Dietary Reference Intakes for calcium, vitamin D*. Washington, DC:The National Academies Press.

Jackson, K. A., & Savaiano, D. A. (2001). Lactose maldigestion, calcium intake and osteoporosis in African-, Asian-, and Hispanic-Americans. *Journal of the American College of Nutrition, 20*, 198S–207S.

Kaiser, L. & Allen, L. H. (2008). Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. *Journal of the American Dietetic Association*, *108*, 553–561.

Kann, L., Grunbaum, J., McKenna, M. L., Wechsler, H., & Galuska, D. A. (2005). Competitive foods and beverages available for purchase in secondary schools—selected sites, United States, 2004. *Morbidity and Mortality Weekly Report, 54*, 917–921.

Kawai, M., & Rosen, C. J. (2012). The insulin-like growth factor system in bone: Basic and clinical implications. *Endocrinology and Metabolism Clinics of North America*, *41*, 323–333, vi.

Kirby, D., Coyle, K., & Gould, J. B. (2001). Manifestations of poverty and birthrates among young teenagers in California zip code areas. *Family Planning Perspectives, 33*, 63–69.

Klein, J. D. (2005). Adolescent pregnancy: Current trends and issues. *Pediatrics, 116*, 281–286.

Konofal, E., Lecendreux, M., Arnulf, I., & Mouren, M. C. (2004). Iron deficiency in children with attention-deficit/hyperactivity disorder. *Archives of Pediatric and Adolescent Medicine, 158*, 1113–1115.

Kumar, J., Muntner, P., Kaskel, F. J., Hailpern, S. M., & Melamed, M. L. (2009). Prevalence and associations of 25-hydroxyvitamin D deficiency in US children: NHANES 2001–2004. *Pediatrics*, *124*, e362–370.

Lee, S., Bacha, F., Gungor, N., & Arslanian, S. (2008). Comparison of different definitions of pediatric metabolic syndrome: Relation to abdominal adiposity, insulin resistance, adiponectin, and inflammatory biomarkers. *Journal of Pediatrics*, *152*, 177–184.

Leung, K. C., Johannsson, G., Leong, G. M., & Ho, K. K. (2004). Estrogen regulation of growth hormone action. *Endocrine Reviews, 25*, 693–721.

Lin, B.-H., Guthrie, J., & Frazao, E. (1999). Nutrient contribution of food away from home. In Frazao, E. (Ed.), *America's eating habits: Changes and consequences* (pp. 213–242). Agriculture Information Bulletin No. 750. Washington, DC: U.S. Department of Agriculture, Economic Research Service, Food and Rural Economics Division.

Looker, A. C. (2002). Iron deficiency—United States, 1999–2000. *Mortality and Morbidity Weekly Report, 51*, 897–899.

Mazur, R. E., Marquis, G. S., & Jensen, H. H. (2003). Diet and food insufficiency among Hispanic-American youths: Acculturation and socioeconomic factors in the third National Health and Nutrition Examination Survey. *American Journal of Clinical Nutrition, 78*, 1120–1127.

McAnarney, E. R., & Stevens-Simon, C. (1993). First, do no harm. Low birth weight and adolescent obesity. *American Journal of Diseases of Children, 147*, 983–985.

McGinnis, J. M. (1993). Actual causes of death in the United States. *Journal of the American Medical Association, 270*, 2207–2212.

Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B. A., Marks, J. S., & Koplan, J. P. (2000). The continuing epidemic of obesity in the United States. *Journal of the American Medical Association, 284*, 1650–1651.

Molgaard, C., Thomsen, B. L., & Michaelsen, K. F. (1999). Whole body bone mineral accretion in healthy children and adolescents. *Archives of Disease in Childhood, 81*, 10–15.

Moore, C. E., Murphy, M. M., & Holick, M. F. (2005). Vitamin D intakes by children and adults in the United States differ among ethnic groups. *Journal of Nutrition, 135*, 2478–2485.

Moore, T. R. (2004). Adolescent and adult obesity in women: A tidal wave just beginning. *Clinical Obstetrics and Gynecology, 47*, 884–889.

National Cancer Institute. (2010a). Solid fats and added sugars (SoFAS). Risk Factor Monitoring and Methods. Retrieved from http://appliedresearch.cancer.gov/diet/usualintakes/energy/t8.html

National Cancer Institute. (2010b). Usual dietary intakes: Food intakes, US population, 2001–04. Retrieved from http://appliedresearch.cancer.gov/diet/usualintakes/pop

Nicklas, T. A., Bao, W., Webber, L. S., & Berenson, G. S. (1993). Breakfast consumption affects adequacy of total daily intake in children. *Journal of the American Dietetic Association, 93*, 886–891.

Niemeier, H. M., Raynor, H. A., Lloyd-Richardson, E. E., Rogers, M. L. & Wing, R. R. (2006). Fast food consumption and breakfast skipping: Predictors of weight gain from adolescence to adulthood in a nationally representative sample. *Journal of Adolescent Health*, *39*, 842–849.

Ogden, C. (2005). Children and teens told by doctors that they are overweight—United States, 1999–2002. *Mortality and Morbidity Weekly Report, 54*, 848–849.

Ogden, C. L., & Flegal, K. (2010). Changes in terminology for childhood overweight and obesity. *National Health Statistics Report*, *25*. Hyattsville, MD: National Center for Health Statistics.

Ogden, C. L., Lamb, M. M., Carroll, M. D., & Flegal, K. M. (2010, December). Obesity and socioeconomic status in children and adolescents: United States, 2005–2008. *NCHS Data Brief*, 1–8.

Ortega, R. M., Requejo, A. M., López-Sobaler, A. M., Navia, B., Mena, M. C., Basabe, B., & Andres, P. (2004). Smoking and passive smoking as conditioners of folate status in young women. *Journal of the American College of Nutrition, 23*, 365–371.

Pollitt, E. (1995). Does breakfast make a difference in school? *Journal of the American Dietetic Association, 95*, 1134–1139.

Pope, J. F., Skinner, J. D., & Carruth, B. R. (1992). Cravings and aversions of pregnant adolescents. *Journal of the American Dietetic Association, 92*, 1479–1482.

Probart, C., McDonnell, E., Weirich, J. E., Hartman, T., Bailey-Davis, L., & Prabhakher, V. (2005). Competitive foods available in Pennsylvania public high schools. *Journal of the American Dietetic Association, 105*, 1243–1249.

Rey, J. M., Martin, A., & Krabman, P. (2004). Is the party over? *Cannabis* and juvenile psychiatric disorder: The past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*, 1194–1205.

Riggs, B. L., Khosla, S., & Melton, L. J., 3rd. (2002). Sex steroids and the construction and conservation of the adult skeleton. *Endocrine Reviews, 23*, 279–302.

Rogol, A. D. (2010). Sex steroids, growth hormone, leptin and the pubertal growth spurt. *Endocrine Development*, *17*, 77–85.

Roy, D. K., O'Neill, T. W., Finn, J. D., Lunt, M., Silman, A. J., Felsenberg, D., … Reeve, J. (2003). Determinants of incident vertebral fracture in men and women: Results from the European Prospective Osteoporosis Study (EPOS). *Osteoporosis International, 14*, 19–26.

Scholl, T. O., Hediger, M. L., Schall, J. L., Khoo, C. S., & Fischer, R. L. (1994). Maternal growth during pregnancy and the competition for nutrients. *American Journal of Clinical Nutrition, 60*, 183–188.

Scholl, T. O., Stein, T. P., & Smith, W. K. (2000). Leptin and maternal growth during adolescent pregnancy. *American Journal of Clinical Nutrition, 72*, 1542–1547.

Schonau, E. (2004). The peak bone mass concept: Is it still relevant? *Pediatric Nephrology, 19*, 825–831.

Simm, P. J., & Werther, G. A. (2005). Child and adolescent growth disorders—an overview. *Australian Family Physician, 34*, 731–737.

Snyder, L. B., Milici, F. F., Slater, M., Sun, H., & Strizhakova, Y. (2006). Effects of alcohol advertising exposure on drinking among youth. *Archives of Pediatric and Adolescent Medicine, 160*, 18–24.

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Science, 9*, 69–74.

St. Onge, M. P., Keller, K. L., & Heymsfield, S. B. (2003). Changes in childhood food consumption patterns: A cause for concern in light of increasing body weights. *American Journal of Clinical Nutrition, 78*, 1068–1073.

Storey, M. L., Forshee, R. A., & Anderson, P. A. (2004). Associations of adequate intake of calcium with diet, beverage consumption, and demographic characteristics among children and adolescents. *Journal of the American College of Nutrition, 23*, 18–33.

Styne, D. M. (2003). The regulation of pubertal growth. *Hormone Research, 60*, 22–26.

Substance Abuse and Mental Health Services Administration. (2003). Results from the 2002 National Survey on Drug Use and Health: National findings. Retrieved from www.samhsa.gov/data/nhsda/2k2nsduh/results/2k2Results.htm

Substance Abuse and Mental Health Services Administration. (2012). *Results from the 2011 National Survey on Drug Use and Health: Summary of National Findings*. Rockville, MD: Author.

Tailor, A. M., Peeters, P. H., Norat, T., Vineis, P., & Romaguera, D. (2010). An update on the prevalence of the metabolic syndrome in children and adolescents. *International Journal of Pediatric Obesity*, *5*, 202–213.

Thompson, M. P., & Light, L. S. (2011). Examining gender differences in risk factors for suicide attempts made 1 and 7 years later in a nationally representative sample. *Journal of Adolescent Health*, *48*, 391–397.

Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. *Medicine and Science in Sports and Exercise*, *40*, 181–188.

Troiano, R. P., Briefel, R. R., Carroll, M. D., & Bialostosky, K. (2000). Energy and fat intakes of children and adolescents in the United States: Data from the National Health and Nutrition Examination Surveys. *American Journal of Clinical Nutrition, 72*, 1343S–1353S.

U.S. Department of Agriculture. (2012). National School Lunch Program [Fact Sheet]. Retrieved from www.fns.usda.gov/sites/default/files/NSLPFactSheet.pdf

U.S. Department of Agriculture, Agricultural Research Service. (2008). Table 2. Nutrient intakes from food: Mean amounts

and percentages of calories from protein, carbohydrate, fat, and alcohol, one day, 2005–2006. Retrieved from www.ars.usda.gov/SP2UserFiles/Place/12355000/pdf/0506/Table_2_NIF_05.pdf

Veldhuis, J. D., Roemmich, J. N., Richmond, E. J., Rogol, A. D., Lovejoy, J. C., Sheffield-Moore, M., … Bowers, C. Y. (2005). Endocrine control of body composition in infancy, childhood, and puberty. *Endocrine Reviews, 26*, 114–146.

Videon, T. M., & Manning, C. K. (2003). Influences on adolescent eating patterns: The importance of family meals. *Journal of Adolescent Health, 32*, 365–373.

Vogt Yuan, A. S. (2010). Body perceptions, weight control behavior, and changes in adolescents' psychological well-being over time: A longitudinal examination of gender. *Journal of Youth and Adolescence*, *39*, 927–939.

Wastney, M. E., Martin, B. R., Peacock, M., Smith, D., Jiang, X. Y., Jackman, L. A., & Weaver, C. M. (2000). Changes in calcium kinetics in adolescent girls induced by high calcium intake. *Journal of Clinical Endocrinology and Metabolism, 85*, 4470–4475.

Waylen, A., & Wolke, D. (2004). Sex 'n' drugs 'n' rock 'n' roll: The meaning and social consequences of pubertal timing. *European Journal of Endocrinology, 151*, S151–S159.

Weaver, C. M. (2000). The growing years and prevention of osteoporosis in later life. *Proceedings of the Nutrition Society, 59*, 303–306.

Weden, M. M., & Zabin, L. S. (2005). Gender and ethnic differences in the co-occurrence of adolescent risk behaviors. *Ethnic Health, 10*, 213–234.

Weitzman, M., Cook, S., Auinfer, P., Florin, T. A., Daniels, S., Nguyen, M., & Winickoff, J. P. (2005). Tobacco smoke exposure is associated with the metabolic syndrome in adolescents. *Circulation, 112*, 862–869.

Welt, C. K., Chan, J. L., Bullen, J., Murphy, R., Smith, P., DePaoli, A. M., … Mantzoros, C. S. (2004). Recombinant human leptin in women with hypothalamic amenorrhea. *New England Journal of Medicine, 351*, 987–997.

Whiting, S. J., Vatanparast, H., Baxter-Jones, A., Faulkner, R. A., Mirwald, R., & Bailey, D. A. (2004). Factors that affect bone mineral accrual in the adolescent growth spurt. *Journal of Nutrition, 134*, 696S–700S.

Wilson, D. B., Smith, B. N., Speizer, I. S., Bean, M. K., Mitchell, K. S., Uguy, L. S., & Fries, E. A. (2005). Differences in food intake and exercise by smoking status in adolescents. *Preventive Medicine, 40*, 872–879.

Winkleby, M. A., Robinson, T. N., Sundquist, J., & Kraemer, H. C. (1999). Ethnic variation in cardiovascular disease risk factors among children and young adults: Findings from the Third National Health and Nutrition Examination Survey, 1988–1994. *Journal of the American Medical Association, 281*, 1006–1013.

Wit, J. M., & Camacho-Hubner, C. (2011). Endocrine regulation of longitudinal bone growth. *Endocrine Development*, *21*, 30–41.

Xie, B., Gilliland, F. D., Li, Y. F., & Rockett, H. R. (2003). Effects of ethnicity, family income, and education on dietary intake among adolescents. *Preventive Medicine, 36*, 30–40.

# Special Section on Public Health Nutrition Programs for Children

*Rachel Colchamiro, MPH, RD, LDN, and Jan Kallio, MS, RD, LDN*



## CHAPTER OUTLINE



## Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Define pediatric needs for nutrition services.
2. Discuss federal nutrition programs.
3. Summarize nutrition surveillance systems in the United States.

Adequate nutrition during infancy, childhood, and adolescence is critical to promote optimal growth and development and is essential for realizing good health in adulthood. It is recognized that healthy dietary patterns established in the early childhood years tend to be carried into adulthood and set the stage for lifelong good health. Conversely, it is well known that poor health conditions and negative nutritional behaviors observed in childhood have a significant deleterious impact on the incidence of disease later in life.

Many families in the United States struggle to provide their children with the foods needed to achieve optimal growth and prevent disease. The federal government established numerous public health nutrition programs to provide access to resources and services to guide and assist these families in meeting the needs of their growing children. These public health nutrition programs evaluate and document nutritional status and need, provide food and financial support to prevent hunger, deliver nutrition-related services to those with special health and developmental needs, and offer nutrition education and counseling that support the development of healthy behaviors that can be carried into adulthood.

This special section identifies priority nutrition issues to be addressed in a public health setting and explores the federal public health nutrition programs serving the nutritional needs of infants, children, and adolescents. In addition, this special section presents federal nutrition monitoring systems that provide programs with data for planning and evaluation.

## Critical Need for Pediatric Public Health Nutrition Services

The U.S. Department of Health and Human Services, in partnership with several other key federal agencies, establishes 10-year, science-based objectives related to improving public health, an initiative known as Healthy People. Healthy People 2020 includes a number of goals targeted to the nutritional health of infants, children, and adolescents. These Healthy People 2020 objectives provide a framework for public health nutrition programs to understand national priority areas for health promotion and disease prevention. These objectives provide benchmarks and can be used to build administrative support for public health nutrition initiatives. Collecting and reviewing nutrition-related data elements in a community can justify and support the implementation of nutrition programs within public health settings. **Table 1** and **Table 2** feature the Healthy People 2020 objectives that relate to nutrition issues of infants, children, and adolescents.

These objectives outline the priority nutrition needs of infants, children, and adolescents, creating a critical need for public health nutrition programs to establish initiatives and programming to address these issues. A brief discussion of the importance of addressing these issues follows.

### Prematurity and Low Birth Weight

Prematurity, defined as less than 37 weeks of completed pregnancy, and subsequent low birth weight (a birth weight less than 5 lbs 8 oz) are the leading causes of death in the first month of life.

**TABLE 1** Maternal, Infant, and Child Health Objectives

| | |
|---|---|
| **MICH-1** | Reduce the rate of fetal and infant deaths |
| **MICH-8** | Reduce low birth weight (LBW) and very low birth weight (VLBW) |
| **MICH-9** | Reduce preterm births |
| **MICH-13** | Increase the proportion of mothers who achieve a recommended weight gain during their pregnancies |
| **MICH-21** | Increase the proportion of infants who are breastfed |
| **MICH-22** | Increase the proportion of employers that have worksite lactation support programs |
| **MICH-23** | Reduce the proportion of breastfed newborns who receive formula supplementation within the first 2 days of life |
| **MICH-24** | Increase the proportion of live births that occur in facilities that provide recommended care for lactating mothers and their babies |

Courtesy of U.S. Department of Health and Human Services. Office of Disease Prevention and Health promotion. *Healthy People 2020.* Washington, DC. Available at http://www.healthypeople.gov/2020/topicsobjectives2020/objectiveslist.aspx?topicid=26. Accessed November 12, 2013.

**TABLE 2** **Nutrition and Weight Status Objectives**

| | |
|---|---|
| **NWS-1** | Increase the number of States with nutrition standards for foods and beverages provided to preschool-aged children in child care |
| **NWS-2** | Increase the proportion of schools that offer nutritious foods and beverages outside of school meals |
| **NWS-5** | Increase the proportion of primary care physicians who regularly measure the body mass index of their patients |
| **NWS-6** | Increase the proportion of physician office visits that include counseling or education related to nutrition or weight |
| **NWS-10** | Reduce the proportion of children and adolescents who are considered obese |
| **NWS-11** | Prevent inappropriate weight gain in youth and adults |
| **NWS-12** | Eliminate very low food security among children |
| **NWS-13** | Reduce household food insecurity and in doing so reduce hunger |
| **NWS-14** | Increase the contribution of fruits to the diets of the population aged 2 years and older |
| **NWS-15** | Increase the variety and contribution of vegetables to the diets of the population aged 2 years and older |
| **NWS-16** | Increase the contribution of whole grains to the diets of the population aged 2 years and older |
| **NWS-17** | Reduce consumption of calories from solid fats and added sugars in the population aged 2 years and older |
| **NWS-18** | Reduce consumption of saturated fat in the population aged 2 years and older |
| **NWS-19** | Reduce consumption of sodium in the population aged 2 years and older |
| **NWS-20** | Increase consumption of calcium in the population aged 2 years and older |
| **NWS-21** | Reduce iron deficiency among young children and females of childbearing age |
| **NWS-22** | Reduce iron deficiency among pregnant females |

Courtesy of U.S. Department of Health and Human Services. Office of Disease Prevention and Health promotion. *Healthy People 2020*. Washington, DC. Available at http://www.healthypeople.gov/2020/topicsobjectives2020/objectiveslist.aspx?topicid=26. Accessed November 12, 2013.

Rates of prematurity have grown steadily over the past decade. Premature infants are at higher risk for general illness and disability, including developmental delays, chronic respiratory problems, and vision and hearing impairment, all of which have a critical impact on the nutritional needs and dietary intake of these high-risk infants.

In 2011, there were nearly 465,000 infants born premature in the United States, representing 11.7% of live births. Disparities in prematurity exist between ethnic groups. The highest rates of prematurity were experienced by non-Hispanic black infants (16.8%), followed by Native Americans (13.5%), Hispanic Americans (11.7%), non-Hispanic whites (10.5%), and Asians (10.4%) Although prematurity rates fell each year in the period between 2006 and 2011, the current rate of preterm birth is still higher than rates reported during the 1980s and most of the 1990s.

More than 320,000 infants were considered to have a low birth weight in 2011, accounting for slightly more than 8% of births. Whereas the low birth weight rate increased more than 20% from the mid-1980s through 2006, rates have improved gradually by approximately 2% between 2006 and 2011. Racial disparities in the prevalence of low birth weight continue to emerge, however; the most recent data show that 13.5% of non-Hispanic black infants were born at a low birth weight compared with 8.4% of Asian infants, 7.6% of Native American infants, 7.1% of non-Hispanic white infants, and 7.0% of Hispanic infants (National Center for Health Statistics, n.d.).

### Obesity

Pediatric obesity is defined as a body mass index (BMI) at or about the 95th percentile for age and sex. Nationally representative data from National Health and Nutrition Examination Survey (NHANES) conducted between 2009 and 2010 indicate that an estimated 16.9% of children and adolescents ages 2 to 19 years were obese. Between 1976–1980 and 2009–2010, obesity among preschool children aged 2–5 years increased from 5.0% to 12.1%; during the same time period it increased from 6.5% to 18.0% among those aged 6–11 years and from 5.0% to 18.4% among adolescents. Racial and ethnic disparities

in pediatric obesity rates are apparent; data from the 2007–2008 NHANES survey demonstrate that Hispanic boys aged 2 to 19 years were significantly more likely to be obese than were non-Hispanic white boys, and non-Hispanic black girls were significantly more likely to be obese than were non-Hispanic white girls (Fryar, Carroll, & Ogden, 2012; Ogden & Carroll, 2010).

This trend in pediatric obesity status is of great concern. As the prevalence of obesity in young children increases, so does the incidence of diseases that in the past were primarily associated with adults, such as heart disease and type 2 diabetes. Obesity in young children affects their ability to be physically active and participate in age-appropriate play. It also affects a child's social health and has been linked to difficulties with low self-esteem and depression. Excessive weight in childhood significantly increases the likelihood of adult obesity and the consequent reduction in quality of life and risk of serious chronic disease (Lakshman, Elks, Ong, 2012).

### Anemia

Iron deficiency is a common, yet preventable, nutritional condition that has serious lifelong health consequences. Although it is often thought that iron-deficiency anemia—the most severe form of iron deficiency—is necessary for adverse effects to be evident, it has been established that negative impacts on cognitive development in children and adolescents are evident without the full expression of anemia. Iron deficiency is associated with motor and mental development in children and affects the ability to learn by influencing attention span and memory; it also puts children at higher risk of lead poisoning (Baker, Greer, & the Committee on Nutrition, 2010).

Rates of iron deficiency remain higher than Healthy People 2020 goals. The prevalence of iron deficiency is greatest among certain categories of individuals; 15% of children aged 1 to 2 years were iron deficient in 2005–2008 compared to 5.3% of children aged 3 to 4 years and 10.4% of females aged 12 to 49 years. Non-Hispanic African American and Mexican American females experience iron deficiency at twice the rate of non-Hispanic American white females (National Center for Health Statistics, n.d.).

### Breastfeeding

Breastfeeding is one of the most important contributors to the optimal health and development of infants. All major medical and public health organizations in the United States recommend exclusive breastfeeding for about the first 6 months of life, followed by the addition of complementary foods for the second 6 months of life, as the preferred method of infant nutrition.

Human milk contains an ideal balance of nutrients that matches an infant's needs for growth and development. Because a mother's antibodies are passed to her infant through breast milk, breastfed infants are significantly less likely to suffer from a number of common infant illnesses—including ear infections, upper respiratory infections, and gastrointestinal infections—than are infants who are fed formula. Numerous studies have also shown links between breastfeeding and a reduction in risk for many chronic pediatric conditions such as diabetes, celiac disease, childhood cancers, and obesity. Some studies suggest that infants who are exclusively breastfed for the first year of life or longer may be protected from chronic diseases such as diabetes, lymphoma, leukemia, obesity, high cholesterol, and asthma (American Academy of Pediatrics [APA], 2012).

Despite the overwhelming evidence of the superiority of breastfeeding over formula feeding, breastfeeding rates in the United States lag behind the Healthy People 2020 goals. National breastfeeding initiation, duration, and exclusivity data are collected on a periodic basis as part of the Centers for Disease Control and Prevention (CDC) National Immunization Survey. The most recent data available from 2009 indicate that 76.9% of infants were breastfed at least once after delivery, compared with the 81.9% goal set by Healthy People 2020. Forty-seven percent of infants continue to breastfeed at 6 months, well below the goal of 60.6%, and 25.5% of infants are breastfeeding at their first birthdays, compared with the goal of 34.1%. The need for increased breastfeeding promotion and support initiatives in the United States becomes clearer when rates of exclusive breastfeeding are examined. Only 36% of infants are exclusively breastfeeding at 3 months, below the Healthy People 2020 goal of 46.2%. This rate drops to 16.3% at 6 months, below the Healthy People 2020 goal of 25.5% (Fryar et al., 2012).

### Hunger and Food Insecurity

Food insecurity and hunger continue to be problems of significance to infants, children, and adolescents. Food security is the ensured access to

enough food for an active, healthy life as well as access to enough food that is safe, nutritious, and acquired in socially acceptable ways. There are different levels of food insecurity, both with and without hunger. Hunger is the outcome of limited or uncertain access to food.

Both food insecurity and hunger have significant detrimental effects on the health and well-being of children because of the critical need of adequate energy and nutrients to promote growth and cognitive development. Children who experience inadequate food intake have poorer health status, are sick more often, are more likely to have ear infections, and are more likely to experience impaired growth. In addition, children who experience food insecurity have more missed days of school and learning difficulties and have more frequent hospitalizations. These children also experience higher rates of iron-deficiency anemia and higher risk of lead poisoning (Center on Hunger and Poverty, 2002; Murphy, Ettinger de Cuba, Cook, Cooper, & Weill, 2008). While a recent review of the evidence found no consistent statistically significant association between food insecurity and childhood obesity, the authors confirm that every study examined noted that food insecurity and obesity coexist and that food-insecure children continue to demonstrate high rates of overweight and obesity, despite having limited food resources (Eisenmann, Gundersen, Lohman, Grasky, & Stewart, 2011).

The United States Department of Agriculture's Economic Research Service utilizes an annual survey to monitor the extent and severity of food insecurity in the United States. In 2011, nearly 18 million households, 14.9% of all U.S. households, were food insecure. This figure represents approximately 50 million Americans, nearly 17 million of whom were children. Of these households, 6.8 million experienced very low food insecurity. Households with children reported being food insecure at almost twice the rate of those without children. Data show that in most households children themselves appear to be protected from very low food insecurity unless the hunger among adults in the household becomes severe. Only 1.1% of children in the United States were classified as having experienced very low food insecurity. Whereas rates of food insecurity have remained relatively stable since 2008, current levels represent a significant increase above food insecurity rates reported in the early 2000s (U.S. Department of Agriculture [USDA], 2013b).

Current public health nutrition programs that provide interventions and programs that promote achieving healthy dietary habits and target the prevention and treatment of these critical nutrition issues are warranted to reduce the incidence and prevalence of these conditions in infants, children, and adolescents.

## Federal Public Health Nutrition Programs

The federal government administers several public health nutrition programs that exclusively or primarily address the nutritional needs of infants, children, and adolescents. These programs work individually or in partnership with community programs to provide a food availability safety net and promote the development of sound nutrition patterns to promote lifelong nutritional health for its recipients.

Federal spending on food assistance programs is classified as either mandatory or discretionary. Mandatory programs—often referred to as entitlement programs—use eligibility guidelines that are written into law. All eligible individuals are entitled to the benefits offered. Discretionary programs are funded annually based on budgets determined in federal appropriations acts. Although the eligibility guidelines for discretionary programs may be broad, higher-need individuals may be prioritized to receive benefits in times of fiscal shortfall.

### Special Supplemental Nutrition Program for Women, Infants, and Children

The three primary goals of the Women, Infants, and Children (WIC) program are to provide supplemental nutrient-dense foods, to offer nutrition education, and to supply health and social service referrals to pregnant, postpartum, and breastfeeding women, infants, and children to 5 years of age who are at nutritional risk and meet specific income guidelines. (Refer to the USDA's WIC website at www.fns.usda.gov/wic for current eligibility criteria and information on the WIC program.) WIC is a discretionary program funded by the U.S. Department of Agriculture (USDA) and administered by state agencies and Indian Tribal Organizations; some states also provide funding to broaden their program's ability to serve participants.

Nutritional eligibility of WIC applicants is determined by a thorough health and dietary assessment completed during the certification process.

Nutrition staff use assessment outcomes to provide personalized nutrition education and counseling to meet the participant's needs and interests. Anticipatory guidance tailored to the stage of development of infants and children is also provided.

A key objective of WIC nutrition services is to promote and support breastfeeding as the ideal method of infant nutrition, unless contraindicated. WIC offers a range of breastfeeding education and support services, including peer counseling services. Many WIC programs offer breast pump loan programs to their participants. Breastfeeding mothers enrolled in WIC receive more food benefits and a longer eligibility period than women who formula-feed their infants.

WIC participants receive checks for monthly food benefits that are individually tailored to meet dietary preferences and needs. Some states now utilize Electronic Benefit Transfer (EBT) systems to issue benefits to their participants; the USDA has issued a mandate that all states must implement EBT by the year 2020. The WIC food packages provide supplemental foods designed to meet the special nutritional needs of low-income pregnant, breastfeeding, nonbreastfeeding postpartum women, infants, and children up to 5 years of age who are at nutritional risk. In December 2007, an interim rule revising the WIC food packages was published in the *Federal Register*. New food packages provided to WIC participants now align with the *Dietary Guidelines for Americans, 2010* and infant feeding practice guidelines of the American Academy of Pediatrics. The food packages promote and support the establishment of successful, long-term breastfeeding, provide WIC participants with a wider variety of foods including fruits and vegetables and whole grains, and provide WIC state agencies greater flexibility in prescribing food packages to accommodate the cultural food preferences of WIC participants. An interim rule allows the Food and Nutrition Service to obtain feedback on the revisions while allowing implementation to move forward. A final rule is in clearance at the time of this writing.

WIC participants may also receive coupons to purchase fresh fruits and vegetables during the summer months at authorized farmers markets through the Farmers Market Nutrition Program (FMNP). This program—administered through a federal and state partnership with USDA's Food and Nutrition Service—provides seasonal unprepared produce to WIC families and expands the awareness, use of, and sales at farmers markets. The federal food benefit for Farmers Market Nutrition Program recipients may be no less than $10 and no more than $30 per recipient per year. States may supplement the federal funds with state, local, or private dollars (USDA, 2013).

In 2011, the WIC program served approximately 9 million participants, of whom roughly one-half are children ages 1 to 5; one-fourth are pregnant, postpartum, or breastfeeding women; and one-fourth are infants younger than 12 months (USDA, n.d.b). The WIC program has significant potential impact on the health and nutritional status of the pediatric population because almost half of all infants and one-fourth of all children ages 1 to 4 years in the United States are program participants (Oliveira, Racine, Olmsted, & Ghelfi, 2002).

The WIC program is the most studied of all the federal nutrition programs in terms of its influence on health and nutrition status. The bulk of the research has focused on the impact of WIC participation during pregnancy on birth outcomes. A recent review of the literature examining the effects of the WIC program found that WIC participation is positively associated with gestational age and mean birth weight and negatively associated with the incidence of low and very low birth weight. This review also highlighted findings related to the positive impact of the WIC program on the intake of iron, fiber, fruits, and vegetables as well as on overall variety in foods consumed. Additionally, children participating in WIC have a greater utilization of both preventative and curative health services compared to nonparticipants (USDA, n.d.b).

In 2002, in a study conducted by the General Accounting Office, researchers calculated that the provision of WIC services saves more than $1 billion in costs for federal, state, local, and private payers (Oliveira et al., 2002). Although some analysts believe this figure overestimates the cost savings associated with WIC participation, the body of WIC research in general is strongly suggestive of WIC's positive impact on perinatal outcomes and that these positive effects lead to savings in healthcare costs. Because of a higher utilization of healthcare services by WIC families is suggested in the recent literature, higher medical expenditures may occur in the short term but lead to cost savings in the long term, assuming that underutilization of healthcare services by nonparticipants ultimately leads to a greater incidence of health problems later in life (USDA, n.d.b).

Children's HealthWatch is a pediatric research center that monitors the impact of economic conditions and public policy on the health and well-being of very young children. Research released in a 2009 report from the organization found that children who are eligible for WIC but who do not receive it because of access problems are more likely to be in poor health, at risk for developmental delays, food insecure, underweight, and short for their age (Children's HealthWatch, 2009).

### Supplemental Nutrition Assistance Program

The intention of the Supplemental Nutrition Assistance Program, referred to as SNAP, is to be the nation's first line of defense against hunger. The program provides low-income households with electronic benefit transfer cards to purchase eligible foods for a nutritionally adequate diet at authorized food stores. SNAP is a mandatory program funded by the USDA Food and Nutrition Service and is administered by state welfare, social service, or human service agencies. Local welfare, social service, or human service offices provide the actual services.

Eligibility and allotments for SNAP are based on household size, income, assets, and other factors. (For specific information on income eligibility criteria, refer to the USDA's Supplemental Nutrition Assistance Program website at www.fns.usda.gov/snap.) Legal immigrants who are children or disabled are eligible for SNAP, as well as legal immigrants who have legally resided in the United States for at least 5 years. Able-bodied, childless, unemployed adults may have time limits on their receipt of food stamp benefits.

States have the option of providing nutrition education to SNAP recipients as a part of their program services. The Healthy, Hunger-Free Kids (HHFK) Act of 2010 (Public Law 111-296) restructured the SNAP nutrition education program (SNAP-Ed) to the Nutrition Education and Obesity Prevention Grant Program to improve SNAP-Ed programmatic operations and effectiveness to meet the needs of the eligible low-income population. With emphasis on obesity prevention and utilizing the Socio-Ecological Framework, SNAP-Ed aims to foster more effective and comprehensive programming that is behaviorally focused and evidence-based delivered in multiple venues at multiple, complementary levels.

The goal of SNAP-Ed is to improve the likelihood that individuals and families will make healthy food choices within a limited budget and choose physically active lifestyles consistent with the current *Dietary Guidelines for Americans* and the USDA food guidance system. State agencies primarily contract with Cooperative Extension System offices, state health departments, and university academic centers to provide a wide range of SNAP-Ed services; however, state nutrition education networks, emergency food organizations, and other community-based organizations can also be SNAP-Ed-implementing agencies.

In 2011, the Supplemental Nutrition Assistance Program served approximately 45 million individuals, about one in seven Americans, who each received an average of about $134 per month (in FY2010) in benefits. The average monthly household benefit for each of the 11 million households enrolled in the program that year was approximately $213. Total costs for the year, including both the direct cost of program benefits and the administrative expenses, exceeded $31 billion (USDA, 2012a).

Most SNAP recipients—nearly 55%—are either children or elderly adults. Forty-seven percent of all participants are children, and 8% of recipients are age 60 years or older. Approximately 16% of all households include an elderly member and about 20% include a disabled member. About 30% of all households work and 8% receive cash assistance from Temporary Assistance for Needy Families (TANF). Approximately 21% receive Supplemental Security Income (SSI). A review found that 12% of program participants live above the poverty line (Poikolainen, 2005).

SNAP benefits are based on the "Thrifty Food Plan," the national governmental standard for a minimally nutritionally adequate diet at a low cost. The Supplemental Nutrition Assistance Program allocation formula assumes that households will be able to purchase sufficient foods to reach Thrifty Food Plan levels using their benefits in addition to 30% of any income they receive (USDA, Food and Nutrition Service, Office of Research and Analysis, 2012a).

Studies have found a positive correlation between Supplemental Nutrition Assistance Program participation and food security in the household in which a preschooler lives if the food stamp benefits last throughout the entire month (Perez-Escamilla et al., 2000). The Supplemental Nutrition Assistance Program has been shown to significantly attenuate the negative impact of food insecurity on a child's health status (Cook et al., 2004).

A recent study suggests that receiving SNAP benefits reduces the likelihood of being food insecure by roughly 30% and reduces the likelihood of being very food insecure by 20% (Ratcliffe & McKernan, 2010).

Researchers have found SNAP to be effective in reducing hunger among children of color. Compared with African American children whose food stamp benefits were not reduced in the previous year, African American infants and toddlers whose family benefits were reduced were 33% more likely to be food insecure and those whose benefits were sanctioned were 84% more likely to be food insecure. Those who experienced a reduction in food stamp benefits were also 38% more likely to be reported as being in fair or poor health. Hispanic American children whose family food stamp benefits were reduced were more than twice as likely to be food insecure as were those receiving their full benefits (Joint Center for Political and Economic Studies, 2006).

SNAP households spend about a quarter of their income to purchase food. Additionally, SNAP increases household food expenditures. For each additional dollar in SNAP benefits, an additional 14 to 47 cents of new spending on food occurs, more than what would occur if the same dollar value of benefits were provided as an unrestricted cash grant (Fox, Hamilton, & Lin, 2004). SNAP recipients have been shown to obtain more nutrients for every dollar they spend on food than do shoppers who do not utilize SNAP (www.frac.org). In particular, food stamp participation has been shown to significantly increase preschoolers' intake of iron, zinc, folate, and vitamin $B_6$ (Perez-Escamilla et al., 2000).

### National School Lunch Program

The National School Lunch Program (NSLP) provides nutritious low-cost lunch at full or reduced prices—or free—to children enrolled in school or residential child care institutions. Any child attending school may participate; free meals are available to children from families with incomes at or below 130% of poverty level; reduced-price meals are available to children from families with incomes between 131% and 185% of poverty level. The NSLP is a mandatory program funded by the USDA and administered by each state's Department of Education and local school districts. NSLP services are provided by all public schools; they are voluntary in private schools. The total cost of administering the NSLP in 2011 was $11.1 billion (USDA, Food and Nutrition Service, 2012a).

NSLP also includes reimbursement for snacks for students enrolled in after-school programs. These snacks are provided to children on the same income eligibility basis as school meals; however, programs may serve all their snacks for free if they operate in schools where at least 50% of students are eligible for free or reduced-price meals.

The Healthy, Hunger-Free Kids Act of 2010 directed the update of the meal pattern and nutrition standards for the NSLP to support the *Dietary Guidelines for Americans* beginning in the 2012 school year. The new pattern increases the availability of fruits, vegetables, and whole grains, requires only lower fat and nonfat milk, as well as sets specific calorie limits to ensure age-appropriate meals. In future years, a gradual reduction in the sodium content of meals will go into effect. Local school food authorities make the decisions of what specific foods to serve based on these guidelines.

In the FY2011 school year, more than 31.8 million children each day received their lunch through the NSLP in more than 100,000 schools and nonprofit private schools and residential child care institutions. This represents 58% of this nation's school children (Food Research and Action Center, 2010b). African American children are almost five times more likely to participate in the NSLP than are other children (Dunifon & Kowaleski-Jones, 2003).

An Economic Research Service (ERS)-sponsored study found that NSLP participants had lower intakes of sugar-sweetened beverages than did similar nonparticipants. NSLP participation did not affect the likelihood of being overweight (Gleason, Briefel, Wilson, & Dodd, 2009).

Participation in the NSLP appears to reduce hunger among families with children ages 5 to 18 years (Kabbani & Yazbeck, 2004). Researchers have identified seasonal differences in food security between spring and summer for school-aged children, thereby reducing the prevalence of hunger during the school year (Nord & Romig, 2003).

Many of the available evaluations of the NSLP document the relationship between participating in the program and increased nutrient intake, particularly noting increases in intakes at lunch of vitamin A, calcium, and zinc. A study published in 2003 found that NSLP participants had significantly higher intakes of dietary fiber and 11 key nutrients (vitamin A, vitamin $B_6$, vitamin $B_{12}$, thiamin,

riboflavin, folate, calcium, magnesium, phosphorus, iron, and zinc) and significantly lower intakes of added sugars than children not consuming school lunch (Gleason & Suitor, 2003). Another study noted NSLP participation was associated with a reduced intake of sugar-sweetened beverages in both elementary and secondary school children, and elementary participants consumed a lower percentage of their calories from low-nutrient, energy-dense foods and beverages than did nonparticipants (Gleason et al., 2009).

Studies show that participating in NSLP can lead to improvements in test scores of children and did not have a negative effect on a child's weight (Dunifon & Kowaleski-Jones, 2004; Hofferth & Curtin, 2005).

USDA research indicates that children who participate in the NSLP have improved nutritional intakes compared with those who bring lunch from home or otherwise do not participate (Food Research and Action Center, 2010b). Finally, a study noted a positive impact on behavior of children in families experiencing hunger (Dunifon & Kowaleski-Jones, 2002).

### School Breakfast Program

The School Breakfast Program provides nutritious low-cost breakfast at full or reduced prices or free to children in participating schools or institutions. The goal of the School Breakfast Program is to promote learning readiness and improve healthy eating behaviors. All children attending schools where the breakfast program operates may participate: Free breakfast is available to children from families with incomes at or below 130% of poverty level, and reduced-price breakfast is available to children from families with incomes between 131% and 185% of poverty level. The School Breakfast Program is a mandatory program funded by the USDA and administered by each state's Department of Education and local school districts. Services are provided by both public and private schools as well as residential child care facilities.

More than 12.1 million children in more than 89,000 schools and institutions participated in the School Breakfast Program on a typical day in FY 2011, of which 10.1 million receive their meals free or at a reduced price. Like school lunch, the School Breakfast Program meal standards were changed with the Healthy, Hunger-Free Kids Act of 2010; however, implementation will take place gradually beginning in the 2013 school year.

Research shows that children who have school breakfast eat more fruits, drink more milk, and consume less saturated fat than those who do not eat breakfast or have breakfast at home (Food Research and Action Center, 2010c). Children who have the School Breakfast Program available consume a better overall diet as measured by the Healthy Eating Index (Bhattacharya, Currie, & Haider, 2004).

Studies have concluded that students who eat school breakfast have increased intakes of food energy (Gleason et al., 2009). In addition, students experience increased math and reading scores and improve their speed and memory in cognitive tests. Research shows that children who eat breakfast at school—closer to class and test-taking time—perform better on standardized tests than those who skip breakfast or eat breakfast at home (Food Research Action Center, 2001).

### Summer Food Service for Children

The Summer Food Service Program provides free meals and/or snacks to children as a substitute for the National School Lunch and School Breakfast Programs during summer vacation. Children younger than 18 years of age who come to an approved site are served. People older than age 18 years who are enrolled in school programs for persons with disabilities may also participate in the Summer Food Service Program. The Summer Food Service Program is funded by the USDA and administered generally by each state's Department of Education, but a state health or social service department or a Food and Nutrition Service regional office may be designated.

Summer Food Service Program sites are hosted by local governments and agencies at such places as schools, parks, recreation centers, housing projects, migrant centers, houses of worship, and summer camps. Most Summer Food Service Program sites are considered "open sites," that is, they are open to all the children in the community provided they are located in an area in which at least 50% of the children are from households that would be eligible for free or reduced-price school meals.

In 2011, the Summer Food Service Program served more than 2.3 million children in more than 39,000 sites. These numbers represent that 14.6 children received summer nutrition for every 100 low-income students who received lunch in the 2010–2011 school year and that only one in seven children who needed summer food was reached

(Food Research and Action Center, 2010e). Improving participation in the Summer Food Service Program is key to ensuring that low-income school-aged children receive the nutrition they need during the summer months so they are better able to learn when they return to school.

Most studies evaluating the Summer Food Service Program focus on participation and service delivery. One study did report that the meals served by the Summer Food Service Program supplied at least 33% of the Recommended Dietary Allowance for most nutrients (USDA, Food and Nutrition Service, 1998). Another study reported that Summer Food Service Program meals provided the levels of key nutrients provided in school meals; however, breakfast was slightly lower in food energy and lunches were higher in fat (Gordon et al., 2009).

### Special Milk Program

The Special Milk Program provides milk to children in schools, summer camps, and child care institutions that have no federally supported meal program. Milk is free to children from families that would be income eligible for free meals through the NSLP. The Special Milk Program is funded by the USDA and administered by the each state's Department of Education. Program services are provided by public or nonprofit private schools of high school grade and under, eligible camps, and public or nonprofit private child care institutions not participating in other federally supported meal programs. In 2011, 3,848 schools and residential child care institutions, 782 summer camps, and 527 nonresidential child care institutions participated in the Special Milk Program, serving more than 6.6 million half pints of milk (USDA, Food and Nutrition Service, 2012c). This program is substantially reduced since its peak in the 1960s because of the expansion of the National School Lunch and School Breakfast Programs. The program, however, helps to supply important nutrients to growing children in the absence of the availability of other programs.

### Child and Adult Care Food Program

The Child and Adult Care Food Program (CACFP) provides cash reimbursements and commodity foods for meals served in child and adult day care centers, Head Start programs, family and group day care homes, homeless shelters, and approved after-school care programs. The program provides reimbursement for food and meal preparation costs, ongoing training in the nutritional needs of children, and on-site assistance in meeting the program's strong nutritional requirements.

CACFP is funded by the USDA and administered by each state's Department of Education or alternate state agency (state health or social service department). CACFP services are provided by public and nonprofit private licensed child and adult day care centers and homes, outside after-school-hours care centers, Head Start, other licensed/approved day care centers, and homeless shelters. Any child up to age 12 years or adult attending a participating adult day care facility is entitled to meals. In 2011, 300,000 million children and 120,000 adults received meals and snacks through CACFP.

Research has found that children in sites receiving CACFP funds consume meals that are nutritionally superior to those served to children in child care settings without CACFP. They have higher intakes of key nutrients and fewer servings of fats and sweets than children not receiving CACFP-funded meals. Studies have documented that participation in CACFP is one of the major factors influencing quality child care; in one review the vast majority of family child care homes considered to be providing quality child care had participated in CACFP (Food Research and Action Center, 2010a).

### Commodity Supplemental Food Program

The Commodity Supplemental Food Program (CSFP) provides commodity foods to low-income pregnant, postpartum, and breastfeeding women; infants; children to 6 years of age; and elderly adults. Pregnant, postpartum, and breastfeeding women; infants; and children (up to 6 years) with household income determined to be at or below 185% of poverty level are eligible. Elderly participants must be at least 60 years of age and be living at or below 130% of the poverty level. Some states have residency and nutrition risk criteria. Individuals cannot participate in both WIC and CSFP at the same time.

USDA uses CSFP funds to purchase commodity foods, which are then stored by state health, social service, education, or agriculture agencies. The foods are distributed to local public and nonprofit agencies for provision to eligible individuals. CSFP foods are not intended to constitute a complete diet but rather provide good sources of the nutrients typically lacking in the diets of the target population. Available foods are generally canned

or packaged and include fruits, vegetables, meats, infant formula, cereals, rice, pasta, beans, cheese, and peanut butter.

There is very limited research on CSFP. An evaluation completed by the USDA in 1982 reported positive findings related to pregnancy outcomes; however, for children the findings were inconclusive of positive health outcomes associated with program participation (Fox et al., 2004).

### The Emergency Food Assistance Program

The Emergency Food Assistance Program (TEFAP) provides commodity foods to low-income persons, food banks, food pantries, and soup kitchens. Each state sets criteria for household income eligibility. Households may participate in another federal, state, or local food health or welfare program for which eligibility is based on income and still receive foods funded by the TEFAP. Recipients of prepared meals funded by TEFAP, such as participants in congregate meal programs for elderly adults or homeless persons, are considered to be the most needy and do not need to prove income eligibility. Funded by the USDA, foods are purchased and sent to state agencies for distribution to food banks, food pantries, soup kitchens, and other public and private nonprofit organizations that distribute food to needy individuals and families.

## Team Nutrition

Although not a separate public health nutrition program, the USDA funds the Team Nutrition initiative in an effort to support its Child Nutrition Programs. Team Nutrition is an integrated, behavior-based, comprehensive plan for promoting nutritional health of school children. Team Nutrition's goal is to improve children's lifelong nutrition and physical activity behaviors using the principles of the *Dietary Guidelines for Americans* and MyPlate. Team Nutrition activities fall into one of three key strategies: (1) to provide training and technical assistance to Child Nutrition Program food service staff to assist them in serving meals that taste good and meet nutrition standards, (2) to provide multifaceted, integrated nutrition education for children and their parents through the school system and with community programs, and (3) to provide support for healthy eating and physical activity by facilitating communication between school administrators and other community partners. Through six communication channels—food service initiatives, classroom activities, schoolwide events, home activities, community programs and events, and media events—a comprehensive network exists to deliver consistent nutrition messages through multiple venues.

Team Nutrition activities benefit 50 million school children in more than 96,000 schools nationwide. Schools are encouraged to enroll themselves as "Team Nutrition schools" and to affirm their commitment to prioritize the nutritional health and education of their students. Team Nutrition training grants are available for state agencies to establish or enhance sustainable infrastructures for implementing Team Nutrition goals. State agencies recruit Team Nutrition schools and assist them with the training and support necessary to sustain the local implementation of the initiative. Team Nutrition also makes efforts to provide reinforcing nutrition education messages to children and their parents through partnerships with the WIC Program and Supplemental Nutrition Assistance Program (SNAP) (USDA, 2013a).

## Expanded Food and Nutrition Education Program

The Expanded Food and Nutrition Education Program (EFNEP), administered by the USDA, National Institute of Food and Agriculture, aims to provide low-income individuals with the skills and education necessary to engage in behaviors that lead to nutritious diets and to provide overall nutritional well-being for their families. EFNEP services are provided in all 50 states, the District of Columbia, and 6 U.S. territories. The Land-Grant University, Cooperative Extension System delivers EFNEP locally.

EFNEP designates a major portion of its resources to services targeted at adults, largely consisting of a series of 10–12 lessons over several months in food preparation, purchasing, and safety taught by paraprofessional staff and volunteers. Lessons are generally provided in a group format. In 2011, EFNEP reached approximately 134,000 adults and more than 378,000 other household members.

EFNEP has had success connecting school-aged youth with nutrition education. EFNEP services for youth are provided as an addition to the regular curriculum in a variety of settings, including after-school programs, 4-H clubs, camps,

and community centers. In 2011, 500,000 youth nationwide received an EFNEP educational contact. Data from EFNEP's evaluation and reporting system show that EFNEP education has an impact of youths' nutrition knowledge and behavior. After receiving EFNEP services 73% of youth ate a greater variety of foods, 70% increased their knowledge of basic human nutrition, 65% increased their ability to select low-cost nutritious foods, and 65% improved their food-handling and preparation practices (Montgomery & Willis, 2006).

## Other Federal Programs that Respond to the Nutritional Needs of Infants, Children, and Adolescents

The following programs are not primarily targeted to respond to the nutritional health of infants and children. However, their services are comprehensive and include nutritional components that contribute to the nutritional health and well-being of program participants. Evaluations of the programs' services indicate a range of positive outcomes related to program participation, but generally nutritional outcomes are not specifically identified.

### Head Start and Early Head Start

The Head Start Program provides comprehensive medical, educational, nutrition, social, and dental services; referrals to social services; and other related assessment and early intervention services in a preschool setting to low-income children between the ages of 3 and 5 years and their families. Early Head Start offers early education both in and out of the home, parenting education, comprehensive health and mental health services, nutrition education, and family support services to women before, during, and after pregnancy and to young children up to age 3 years. Both Head Start and Early Head Start provide nutritious meals and snacks to children in their care.

Low-income families receiving public assistance (i.e., Temporary Assistance for Needy Families or Supplemental Security Income) or who have a total annual income less than 100% of the federal poverty level are eligible to receive Head Start services, as are children in foster care. Head Start reserves at least 10% of total enrollment for children with physical disabilities. Head Start is funded by the Federal Department of Health and Human Services Administration for Children and Families and is administered by U.S. Department of Health and Human Services regional offices. Services are provided by local public agencies, private nonprofit and for-profit organizations, American Indian tribes, and school systems.

More than 1 million children and pregnant women were enrolled in Head Start in 2011 through 1,600 local programs.

### Early Intervention

The early intervention program for infants and toddlers with disabilities is funded under Part C of the Individuals with Disabilities Education Act. Federal grants are provided to states to offer children younger than the age of 3 years and their families a comprehensive multidisciplinary system of services. In most states, the Department of Health and Human Services or Department of Education administers the program. Early intervention services include audiology, health and medical services, nutrition services, occupational therapy, physical therapy, psychological and social work services, speech and language services, and vision care.

Program eligibility is based on state-determined evaluation and assessment systems that identify children who have or who are at risk for a developmental delay. All states must ensure that all children birth to age 3 years who are eligible for program services are identified, located, and evaluated for need at no cost to parents. Once need is determined, services are either provided free of charge to families or fees are determined on a sliding scale basis. Health insurance plans often cover the cost of services (American Academy of Pediatrics, n.d.).

## Nutrition Surveillance Systems in the United States

Policy makers, program administrators, and program evaluators rely on the comprehensive collection of health statistics to set public health priorities, strategically design public health and nutrition programs, and determine the impact of program components on the health status of those served.

The Youth Risk Behavior Surveillance System (YRBSS), administered by the Centers for Disease Control and Prevention, monitors six categories of high-risk behaviors among 9th- to 12th-grade youth (i.e., behaviors that contribute to unintentional injuries and violence, tobacco use, alcohol and other drug use, sexual behaviors that contribute to unintended pregnancy and sexually

transmitted diseases, unhealthy dietary behaviors, and physical inactivity) as well as indicators of weight status. YRBSS data represent a national school-based survey conducted by the CDC as well as state and local school-based surveys conducted by education and health agencies. YRBSS data are used to measure progress toward achieving national health objectives and to monitor leading health indicators for youth. In addition, education and health officials at national, state, and local levels use Youth Risk Behavior Surveillance System data to improve policies and programs that reduce high-risk, health-related behaviors among young people (CDC, 2011).

The CDC also conducts the National Health and Nutrition Examination Survey (NHANES) on an ongoing basis. The survey assesses the health and nutritional status of all age segments of the population and monitors changes over time. A major objective of the survey's nutrition component is to provide data for nutrition monitoring purposes, including tracking nutrition, identifying risk factors related to food insecurity, and estimating the prevalence of compromised nutritional status. A second major objective is to provide information for studying the relationships among diet, nutritional status, and health. A dietary 24-hour recall is used to obtain dietary data. The survey is unique because it combines data from both interviews and physical examinations.

An additional source of surveillance nutrition data for young children is the biannual WIC Participant and Program Characteristics (PC) report, which summarizes the national demographic characteristics of participants in the WIC program. The report also includes information on participant income, growth data, and nutrition risk characteristics, as well as breastfeeding initiation rates for WIC infants.

## Summary

It is well established that achieving optimal nutritional status during infancy, childhood, and adolescence is critical to proper growth and development and for setting the stage for good health in adulthood. Federal nutrition programs provide a range of services that strive to target the nutritional needs of this population. These programs provide a food availability safety net and promote sound nutritional patterns for program participants and their families. These programs have demonstrated long-term success in delivering services that improve the nutritional well-being of infants, children, and adolescents. With the Healthy People 2020 goals as nutritional benchmarks and the available nutrition monitoring systems, these federal nutrition programs can enhance and adapt their services to meet the nutritional needs of infants, children, and adolescents in this population.

## Case Study

### Navigating Nutrition Resources for Families

Rachel Colchamiro, MPH, RD, LDN, and Jan Kallio, MS, RD, LDN

Julie and Sam Patterson have a 3-year-old daughter and a 7-year-old son. Julie is 3 months pregnant. About a year ago, Sam lost his job and the family eventually decided to move from out of state to live with Julie's parents who have an in-law apartment on their property. Although they can provide them housing, Julie's parents also have limited resources and are not able to offer much else to support the family. Julie has found a part-time job in the evenings; Sam is still looking for work. To date, the family has been living off their savings and has not yet enrolled in any assistance programs, but their resources are now quite limited. Julie recently came to the local Community Health Center for an appointment with her new obstetrician. As part of her visit, she met with a case manager who provided Julie with several referrals to help ensure that her family had adequate access to food.

**Questions**

1. What resources could be shared with Julie?
2. What eligibility criteria would need to be met for her family to participate?

## References


American Academy of Pediatrics. (n.d.).*The Medical Home and Early Intervention programs*. Retrieved from www.medicalhomeinfo.org/downloads/pdfs/eibrochuref.pdf

American Academy of Pediatrics, Section on Breastfeeding. (2012). Breastfeeding and the use of human milk. Retrieved from http://pediatrics.aappublications.org/content/early/2012/02/22/peds.2011-3552

Baker, R., Greer, F., & the Committee on Nutrition. (2010). Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children (0–3 years of age). *Pediatrics*, *126*, 1040.

Bhattacharya, J., Currie, J., & Haider, S. J. (2004). *Evaluating the impact of school nutrition programs final report*. Washington, DC: U.S. Department of Agriculture, Economic Research Service.

Bitler, M., & Currie, J. (2004). *Medicaid at birth, WIC take up, and children's outcomes*. Discussion Paper no. 1286-04. Institute for Research on Poverty. Retrieved from

www.rand.org/content/dam/rand/pubs/working_papers/2005/RAND_WR172.pdf
Center on Hunger and Poverty. (2002). *Consequences of hunger and food insecurity for children: Evidence from recent scientific studies*. Retrieved from www.hungernwnc.org/about-hunger/Consequences%20of%20hunger-children.pdf
Centers for Disease Control and Prevention. (2013). Breastfeeding: Data and statistics: Breastfeeding Among U.S. Children Born 2000–2010, CDC National Immunization Survey. Retrieved from www.cdc.gov/breastfeeding/data/nis_data
Centers for Disease Control and Prevention. (2011). Youth Risk Behavior Surveillance System. Dietary Behaviors. Retrieved from http://apps.nccd.cdc.gov/youthonline/App/QuestionsOrLocations.aspx?CategoryId=5
Children's HealthWatch. (2009, May). *Feeding our future*. Retrieved from www.childrenshealthwatch.org/upload/resource/Feeding_our_future.pdf
Coleman-Jensen, A., Nord, M., Andrews, M., & Carlson, S. (2012). *Household food security in the United States in 2011*. ERR-141. Washington, DC: U.S. Department of Agriculture, Economic Research Service. Retrieved from www.ers.usda.gov/media/884525/err141.pdf
Cook, J. T., Frank, D. A., Berkowitz, C., Black, M. M., Casey, P. H., Cutts, D. B., … Nord, M. (2004). Food insecurity is associated with adverse health outcomes among human infants and toddlers. *Journal of Nutrition, 134*, 1432–1438.
Dalenius, K., Borland, E., Smith, B., Polhamus, B., & Grummer-Strawn, L. (2012). Pediatric Nutrition Surveillance 2010 report. Atlanta, GA: U.S. Department of Health and Human Services and Centers for Disease Control and Prevention.
Dunifon, R., & Kowaleski-Jones, L. (2002). *Associations between participation in the National School Lunch Program, food insecurity, and child well-being*. Discussion Paper No. 1249-02. Madison, WI: Institute for Research on Poverty.
Dunifon, R., & Kowaleski-Jones, L. (2003). The influences of participation in the National School Lunch Program and food insecurity on child well-being. *Social Service Review, 77*(1), 72–92.
Dunifon, R., & Kowaleski-Jones, L. (2004). *Exploring the influence of the National School Lunch Program on children*. Discussion Paper No. 1277-04. Madison, WI: Institute for Research on Poverty.
Eisenmann, J., Gundersen, C., Lohman, B., Grasky, S., & Stewart, S. (2011). Is food insecurity related to overweight and obesity in children and adolescents? A summary of studies, 1995–2009. *Obesity Reviews*, *12*, e73–e83.
Food Research and Action Center. (2001). *Breakfast for learning*. Retrieved from http://frac.org/wp-content/uploads/2009/09/breakfastforlearning.pdf
Food Research and Action Center. (2010a). Federal food/nutrition programs: Child and Adult Care Food Program. Retrieved from http://frac.org/federal-foodnutrition-programs/child-and-adult-care-program
Food Research and Action Center. (2010b). Federal food/nutrition programs: National School Lunch Program. Retrieved from http://frac.org/federal-foodnutrition-programs/national-school-lunch-program
Food Research and Action Center. (2010c). Federal food/nutrition programs: School Breakfast Program. Retrieved from http://frac.org/federal-foodnutrition-programs/school-breakfast-program
Food Research and Action Center. (2010d). Federal food/nutrition programs: SNAP/food stamps. Retrieved from www.frac.org/html/federal_food_programs/programs/fsp.html
Food Research and Action Center. (2010e). Federal food/nutrition programs: Summer nutrition programs. Retrieved from http://frac.org/federal-foodnutrition-programs/summer-programs
Fox, M., Hamilton, W., & Lin, B. (2004). *Effects of food assistance and nutrition programs on nutrition and health*. Food Assistance and Nutrition Report No. FANRR-19-3. Washington, DC: U.S. Department of Agriculture, Food and Rural Economic Division, Economic Research Service.
Fryar, C. D., Carroll, M. D., & Ogden, C. L. (2012). Prevalence of obesity among children and adolescents: United States, trends 1963–1965 through 2009–2010. Health E-Stat. Retrieved from www.cdc.gov/nchs/data/hestat/obesity_child_09_10/obesity_child_09_10.pdf
Gleason, P., Briefel, R., Wilson, A., & Dodd, A. H. (2009). *School meal program participation and its association with dietary patterns and childhood obesity*. Food and Nutrition Assistance Research Program Report No. 55. Washington, DC: U.S. Department of Agriculture, Economic Research Service.
Gleason, P. M., & Suitor, C. W. (2003). Eating at school: How the National School Lunch Program affects children's diets. *American Journal of Agricultural Economics, 85*, 1047–1061.
**Gordon,** A., Cohen, R., Crepinsek, M., Fox, M., Hall, J., & Zeidman, E. (2009). The Third School Nutrition Dietary Assessment Study: background and study design. *Journal of the American Dietetic Association, 109*, S20–S30.
Hamm, K., & Ewen, D. (2006). *From the beginning: Early Head Start children, families, staff, and programs in 2004*. Washington, DC: Center for Law and Social Policy.
Hanes, S., Vermeersch, J., & Gale, S. (1984). The national evaluation of school nutrition programs: Program impact on dietary intake. *American Journal of Clinical Nutrition, 40*, 390–413.
HealthyPeople.gov. (2013). Healthy People 2020. Retrieved from www.healthypeople.gov/2020/default.aspx
Hofferth, S. L., & Curtin, S. (2005). Poverty, food programs and obesity. *Journal of Policy Analysis and Management, 24*, 703–726.
Joint Center for Political and Economic Studies Health Policy Institute. (2006). *Protecting the health and nutrition of young children of color: The impact of nutrition assistance and income support programs*. Washington, DC: Author.
Kabbani, N. S., & Yazbeck, M. (2004). *The role of food assistance programs and employment circumstances in helping households with children avoid hunger*. Discussion Paper No. 1280-04. Madison, WI: Institute for Research on Poverty.
Lakshman, R., Elks, C., Ong, K. (2012). Obesity. *Circulation, 126*, 1770–1779.
Montgomery, S., & Willis, W. (2006). *Fiscal year 2005 impact and review of the Expanded Food and Nutrition Education*

*Program*. Retrieved from www.csrees.usda.gov/nea/food/efnep/pdf/2005_impact.pdf

Murphy, C., Ettinger de Cuba, S., Cook, J., Cooper, R., & Weill, J. (2008). *Reading, writing and hungry: The consequences of food insecurity on children, and on our nation's economic success*. Food Research and Action Center. Retrieved from http://frac.org/newsite/wp-content/uploads/2010/03/reading_writing_hungry_report.pdf

National Center for Health Statistics. (n.d.). Health indicators warehouse. Retrieved from www.healthindicators.gov

Nord, M., & Romig, K. (2003). *Hunger in the summer: Seasonal food insecurity and the National School Lunch and Summer Food Service Programs*. Paper presented at the Association for Public Policy Analysis and Management Annual Research Conference, Washington, DC.

Ogden, C., & Carroll, M. (2010, June). Prevalence of obesity among children and adolescents: United States, trends 1963–1965 through 2007–2008. Health E-Stat. Retrieved from www.cdc.gov/nchs/data/hestat/obesity_child_07_08/obesity_child_07_08.pdf

Oliveira, V. (2003). *Food assistance research brief—WIC and breastfeeding rates*. Food Assistance and Nutrition Report No. FANRR-34-2. Washington, DC: U.S. Department of Agriculture, Economic Research Service.

Oliveira, V., Racine, E., Olmsted, J., & Ghelfi, L. (2002). *The WIC program: Background, trends, and issues*. Food Assistance and Nutrition Report No. FANRR27. Washington, DC: U.S. Department of Agriculture, Food and Rural Economic Division, Economic Research Service.

Perez-Escamilla, R., Ferris, A. M., Drake, L., Haldeman, L., Peranick, J., Campbell, M., … Bernstein, B. (2000). Food stamps are associated with food security and dietary intake of inner-city preschoolers from Hartford, Connecticut. *Journal of Nutrition, 130*, 2711–2717.

Poikolainen, A. (2005). *Characteristics of food stamp households: Fiscal year 2004*. Report No. FSP-05-CHAR. Alexandria, VA: U.S. Department of Agriculture, Food and Nutrition Service, Office of Analysis, Nutrition and Evaluation.

Ratcliffe, C., & McKernan, S. (2010). *How much does SNAP reduce food insecurity?* Washington, DC: Urban Institute. Retrieved from www.urban.org/UploadedPDF/412065_reduce_food_insecurity.pdf

U.S. Department of Agriculture. (n.d.a). Child and Adult Care Food Program (CACFP). Retrieved from www.fns.usda.gov/cnd/care

U.S. Department of Agriculture. (n.d.b). Women, Infants and Children (WIC). Retrieved from www.fns.usda.gov/wic

U.S. Department of Agriculture. (2013, July). WIC Farmers' Market Nutrition Program. Retrieved from www.fns.usda.gov/WIC/WIC-FMNP-Fact-Sheet.pdf

U.S. Department of Agriculture, Food and Nutrition Service. (1998). *Evaluation of the Summer Food Service Program, final report*. Washington, DC: Author.

U.S. Department of Agriculture, Food and Nutrition Service. (2012a). *National School Lunch Program*. Retrieved from www.fns.usda.gov/cnd/Lunch/AboutLunch/NSLPFactSheet.pdf

U.S. Department of Agriculture, Food and Nutrition Service. (2012b). *The School Breakfast Program*. Retrieved from www.fns.usda.gov/cnd/Breakfast/AboutBFast/SBPFactSheet.pdf

U.S. Department of Agriculture, Food and Nutrition Service. (2012c). Special Milk Program. Retrieved from www.fns.usda.gov/cnd/milk/AboutMilk/SMPFactSheet.pdf

U.S. Department of Agriculture, Food and Nutrition Service. (2013a). About Team Nutrition. Retrieved from www.fns.usda.gov/tn/about.html

U.S. Department of Agriculture, Food and Nutrition Service. (2013b). Program data. Retrieved from www.fns.usda.gov/pd/snapmain.htm

U.S. Department of Agriculture, Food and Nutrition Service, Office of Research and Analysis. (2012a). *Building a healthy America: A profile of the Supplemental Nutrition Assistance Program*. Retrieved from www.fns.usda.gov/ora/MENU/Published/snap/FILES/Other/BuildingHealthyAmerica.pdf

U.S. Department of Agriculture, Food and Nutrition Service, Office of Research and Analysis (2012b). *Effects of the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC): A review of recent research*. Special Nutrition Programs Report No. WIC-12-WM. Alexandria, VA: Author. Retrieved from www.fns.usda.gov/Ora/menu/Published/WIC/FILES/WICMedicaidLitRev.pdf

U.S. Department of Agriculture, National Institute of Food and Agriculture. (2013). Nutrition: Expanded Food and Nutrition Education Program (EFNEP). Retrieved from www.csrees.usda.gov/nea/food/efnep/efnep.html

# CHAPTER 5

# Special Topics in Prenatal and Infant Nutrition: Genetics and Inborn Errors of Metabolism and Failure to Thrive

*Laura Harkness, PhD, RD, Sara Snow, MS, RD, Claire Blais, RD, CDE, LDN, Jessica Burfield, RD, LDN, Jennifer Blair, RD, CSP, LDN, Christie Hussa, MBA, RD, LDN, and Rebecca Randall, MS, RD, LDN*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Define and describe the biochemistry of some of the more prevalent inborn errors of metabolism.
2. Describe the prevalence, diagnosis, clinical symptoms, and long-term complications of inborn errors of metabolism.
3. Discuss medical nutrition therapy for inborn errors of metabolism.
4. Identify and classify failure to thrive.
5. Evaluate the diet of the patient with failure to thrive and describe techniques to treat failure to thrive in the outpatient and inpatient settings.

# *Genetics and Inborn Errors of Metabolism*

*Laura Harkness, PhD, RD, Sara Snow, MS, RD, Jessica Burfield, RD, LDN, Jennifer Blair, RD, CSP, LDN, Christie Hussa, MBA, RD, LDN, and Rebecca Randall, MS, RD, LDN*

## Newborn Screening

Newborn screening (NBS) is a test that is performed on newborn babies within the first few days of life to identify infants at an increased risk for specific genetic disorders so treatment can begin as soon as possible. The aim of newborn screening is to prevent children from irreversible, and sometimes deadly, complications of untreated inherited disease. "Newborn screening is one of the best established and most important programs in preventive medicine" (Sarafoglou, Hoffman, & Roth, 2009). It is a measure for detecting newborn infants afflicted with metabolic and endocrine disorders. Most cases with **inborn errors of metabolism** result in serious consequences to the affected individual. These may include mild to severe irreversible mental retardation, lifelong disability, physical handicaps, coma, and early death (Couce et al., 2011; Loukas et al., 2010). Early identification of these conditions improves outcomes for affected individuals who receive aggressive and early treatment.

**inborn errors of metabolism** Traits arising from a variation in the structure of enzymes or protein molecules.

**phenylketonuria** A form of hyperphenylalaninemia caused by the complete or near complete deficiency of the liver enzyme phenylalanine hydroxylase that results in an accumulation of phenylalanine in body fluids and the central nervous system.

Newborn screening began in the United States in the 1960s when Dr. Robert Guthrie, an American bacteriologist and physician, developed a method to screen newborns for **phenylketonuria** (PKU). Prior to his screening, PKU was typically not diagnosed before 6 months of age, after developmental delay or other neurologic symptoms became apparent. At this point, treatment could improve the patient's symptoms, but not reverse the neurological damage (Schulze, Mayatepek, & Hoffmann, 2002). Guthrie's invention of screening for PKU using a bacterial inhibition assay (BIA) performed on a filter paper of dried blood enabled the presymptomatic identification of PKU. Therefore, treatment could be initiated prior to the onset of the irreversible damage. Once Guthrie's BIA was well established, newborn screening began in several areas in the United States and rapidly spread around the world (Guthrie, 1961, 1996; Sarafoglou et al., 2009). Additional disorders (e.g., congenital hypothyroidism, galactosemia, and sickle cell anemia) were subsequently added to the NBS panel.

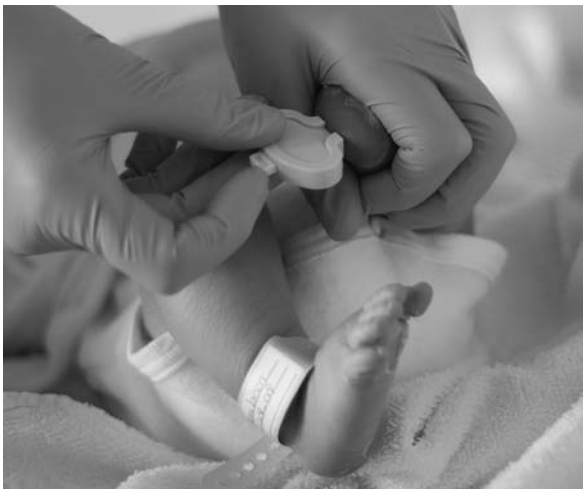
The BIA was modified to detect other disease makers, and over time, more sophisticated technologies were developed to determine other disease metabolites and enzyme activities. Millington, Kodo, Norwood, and Roe (1990) introduced tandem mass spectrometry (MS/MS) for detecting diseases. MS/MS can determine more than 30 analytes in blood samples saturated on paper, including amino acids and acylcarnitines. This allows for the detection of inborn errors of metabolism, such as aminoacidopathies, galactosemia, organic acidurias, and fatty acid oxidation disorders (Couce et al., 2011; Millington et al., 1990; Sarafoglou et al., 2009).

Today, newborn screen testing is performed on a blood sample obtained with a heel prick of an infant in the first 1 to 4 days of life. The blood obtained from the heel prick is spotted on to filter paper and sent to a screening lab. The lab tests

the dried blood spots of the newborn screening panel. The presence of disease can most often be ruled out on the initial screening. If a positive test result is found, most screening programs request a second blood spot for repeat testing. If an initial or repeat newborn screening result is positive, further diagnostic testing with a pediatric specialist, or geneticist, is recommended to confirm the results (National Newborn Screening and Global Resource Center, 2012, Sarafoglou et al., 2009).

Currently there is a panel of 29 metabolic and genetic diseases detected by the newborn screen. The screened diseases vary by state and do not always include the full panel of conditions but commonly include phenylketonuria, galactosemia, congenital hypothyroidism, and sickle cell disease (National Newborn Screening and Global Resource Center, 2012). Careful considerations are raised in regard to the impact incurred for testing affected versus nonaffected individuals when deciding which diseases are included in a newborn screening program. Recommendations and screening policies vary depending on the region's economic, political, and medical factors and their public health organization (Sarafoglou et al., 2009).

In 2006, the American College of Medical Genetics (ACMG) recommended screening of 29 diseases by all newborn screening programs. An additional 25 conditions were listed that could be identified in the course for screening core panel conditions. It is the responsibility of each state's newborn screening program to establish a diagnosis and communicate the result to the healthcare provider and family (Schulze, Mayatepek, & Hoffmann, 2002). Advances in the technology used in newborn screening are constantly occurring and affect the management of patients detected on newborn screening. Dietary manipulation is a treatment modality for approximately half of the disorders included on the full newborn screening panel. Typically, inborn errors of metabolism are categorized by the macronutrient substrate(s) primarily affected. This chapter highlights key inborn errors of metabolism within each macronutrient category as follows: protein, fats, and carbohydrates.



© Hemera/Thinkstock

## Disorders of Amino Acid Metabolism

### Phenylketonuria

**autosomal recessive** Both parents must be carriers of a gene on one of the autosomal (nonsex) chromosomes for a child to inherit the disease. If both parents are carriers, there is a 25% chance that a child will inherit the disease and a 50% chance the child will be a carrier.

Phenylketonuria (PKU) is an **autosomal recessive** disorder of the aromatic amino acid phenylalanine (phe) (Acosta, 2010; Sarafoglou et al., 2009). PKU is caused by a deficiency in the enzyme phenylalanine hydroxylase (PAH), which requires the coenzyme tetrahydrobiopterin (BH4) (Acosta, 2010) and involves the degradation of phe to tyrosine (tyr) (see **Figure 5.1**). Deficiency of PAH causes an accumulation of phe and phe metabolites in the serum, cerebrospinal fluid, and urine as well as a deficiency of tyr and its downstream products (melanin, thyroxine, and catecholamine neurotransmitters) (Acosta, 2010). Approximately 25% of phe in the body is used for protein synthesis, while 75% is converted to tyr and used for the synthesis of protein, catecholamines, melanin pigment, and thyroid hormones (Acosta, 2010). The incidence of PKU is 1 in 10,000 worldwide, but there is a great ethnic variability with reported rates of 1:2,600 in Turkey, 1:4,500 in Ireland, and 1:200,000 in Finland (Sarafoglou et al., 2009).

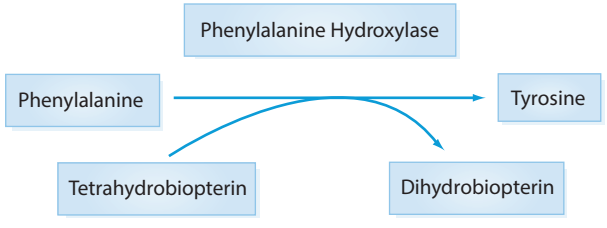



**FIGURE 5.1** Phenylalanine conversion to tyrosine

PKU is detected with an elevated plasma phe level on the newborn screen; confirmatory testing of a sustained elevated phe level verifies the diagnosis. Without screening, PKU could be detected by a musty or mousy odor of an affected person, a result of the buildup of the ketone phenylacetate. Untreated classical PKU patients typically have mental retardation, with microcephaly, spastic reflexes, eczema, and autistic behavior (Acosta, 2010). Diet therapy has essentially eliminated the incidence of the severe consequences of untreated PKU (Burton et al., 2011). More than 400 mutations of the PAH gene have been identified, leading to variable degrees of deficiency in PAH activity and a wide spectrum of clinical severity and dietary phe tolerance (Matalon et al., 2004). PKU type is determined according to the phe level at diagnosis (Scriver, Waters, & Sarkissian, 2000). Approximately 50% of cases are categorized as classical PKU (with confirmatory phe levels of > 1,200 umol/L), 30% of cases are mild PKU (with confirmatory phe levels of 600–1,200 umol/L), and 20% of cases are non-PKU hyperphenylalaninemia (HPA) with confirmatory phe levels between 120 and 599 umol/L (Acosta, 2010). In addition, maternal PKU patients with phe levels out of treatment range have poor reproductive outcomes, such as offspring having low birth weight, microcephaly, congenital anomalies, and mental retardation (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009).

### Nutrition Management of Phenylketonuria

The goal of PKU management is to maintain target blood phe levels, which vary depending on age group, and a phe-restricted diet for life (National Institutes of Health Consensus Development Panel, 2001). Patients with PKU manage their phe levels by limiting the offending amino acid phe and supplementing the conditionally essential amino acid tyr to prevent deficiency. Typically, a PKU patient is on a low-phe diet, where all high-protein foods are eliminated and the protein or phe in foods is counted daily. The diet is usually supplemented with medical food, including medical protein sources (phe-free amino acid formula) and specialty low-protein foods.

Total protein needs (intact protein and medical food) for PKU patients are higher than for the healthy population; there are guidelines for total protein recommendations by age group for the PKU population. Protein needs are higher because amino-acid-based medical foods are rapidly oxidized to ketoacids and there is less efficient utilization of amino-acid-based products compared with intact protein by the body. Medical foods are the primary source of protein for patients with classical PKU. These medical foods also contain calories, fat, carbohydrates, and micronutrients (Acosta, 2010). Energy requirements are somewhat greater in infants as a result of the use of free amino acids as the primary source of protein; energy needs after infancy are similar to healthy population recommendations. Most medical foods are supplemented with vitamins and minerals; micronutrient needs may be higher in this population (Acosta, 2010).

At diagnosis, the goal is the rapid reduction of plasma phe levels to the treatment range as well as the prevention of catabolism. Nutrition therapy is initiated as soon as the diagnosis is confirmed. Phe levels should be maintained within treatment range using prescribed amounts of medical food and intact protein. During times of good health, the goal is to achieve phe and tyr levels within treatment range goals, while also promoting normal growth and development. During illness, phe levels can become elevated, and the goal is to return plasma phe levels to the treatment range as soon as possible. PKU patients are prescribed a specific amount of phe or protein through intact protein (i.e., regular foods and beverages) (Acosta, 2010); medical foods meet the remainder of the protein goals. It is also recommended that patients monitor phe and tyr levels regularly and maintain stable concentrations within the treatment range (Acosta, 2010).

The long-term goals and outcomes of proper nutrition management of PKU include normal growth, lean body mass, and percentage of body fat (Acosta, 2010). There have been reports of neurocognitive deficits in the PKU population; these deficits vary based on timing of diagnosis, PKU type, and adherence to treatment (Anastasoaie, Kurzius, Forbes, & Waisbren, 2008). PKU patients have also demonstrated increased incidence of attention deficit disorder, impulsivity, hyperactivity, mental illness, pancreatitis, and recurrent infections (Acosta, 2010). Early diagnosis and medical management can result in normal intellectual and behavioral outcomes (Acosta, 2010).

There are several new approaches to therapy for PKU. Large neutral amino acids (LNAA), including methionine, tryptophan, tyrosine, valine, isoleucine, leucine, threonine, and histidine, can be administered to patients with PKU; LNAA supplementation increases plasma levels of LNAA, which compete with phe at the blood–brain barrier and

reduce the uptake of phe into the brain (*Dietitian's Guide*, 2010). Phenylalanine levels can be significantly reduced in a subset of PKU patients by the administration of the cofactor for the PAH enzyme, tetrahydrobiopterin (BH4). The synthetic form of BH4, sapropterin dihydrochloride (Kuvan) has allowed for a reduction of blood phe of at least 20% or a more liberalized diet restriction for patients (Belanger-Quntana, Burlina, Harding, & Muntau, 2011; Singh & Quirk, 2011). Finally, glycomacropeptide (GMP) is an ideal protein source for the nutritional management of PKU because the pure protein contains no phenylalanine. GMP is a naturally occurring intact protein that is produced during cheese-making and is one of several proteins that make up whey protein. GMP is now being used as a medical food for the treatment of PKU (Van Calcar et al., 2009).

### Tyrosinemia

**tyrosinemias** A group of inherited inborn errors of metabolism characterized by disordered tyrosine metabolism.

**Tyrosinemia** is an autosomal recessive disorder of the metabolism of the aromatic amino acid tyrosine (tyr) (Acosta, 2010; Sarafoglou et al., 2009). Tyrosine is a nonessential amino acid that derives from the breakdown of food, phe hydroxylation, and body protein catabolism. Tyrosine is used for the synthesis of protein, catecholamines, thyroid hormones, melanin pigments, and for energy. Increased plasma tyr levels occur in three disorders of tyr metabolism: fumarylacetoacidic acid hydrolase (FAH) (type 1), tyrosine aminotransferase (TAT) deficiency (type 2), and p-hydroxyphenylpyruvic acid dioxygenase (p-HPPAD) (type 3) (Acosta, 2010; Sarafoglou et al., 2009). The TAT enzyme requires pyridoxine as a coenzyme. It is important to note that there are also secondary causes of raised tyr levels including acute liver failure and transient neonatal tyrosinemia (Sarafoglou et al., 2009). Of the tyrosinemias, type 1 occurs most frequently, with 1:100,000 in Sweden, 1:17,000 in Quebec, and lower incidence elsewhere. There have been 50 reported cases of type 2; type 3 is even rarer (Sarafoglou et al., 2009).

For type 1, plasma tyr levels may not be elevated; diagnosis is confirmed through the detection of a toxic metabolite, succinylacetone, in the urine (Acosta, 2010; Sarafoglou et al., 2009). Patients with type 1 may present with the following symptoms: a cabbage-like odor, generalized renal tubular impairment with pyrophosphatemic rickets, progressive liver failure resulting in cirrhosis with hepatic cancer, hypertension, acute porphyric episodes, and peripheral nerve deficiencies (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009). Type 2 is characterized by elevated concentrations of blood and urine tyr as well as other metabolites. Patients with type 2 may present with corneal erosions, hyperkeratotic lesions on the soles and palms, and cognitive or behavioral problems (Sarafoglou et al., 2009). Type 3 presents with elevated plasma tyr levels and other metabolites. Patients with type 3 have a variable presentation, some patients have neurological findings and learning difficulties (Sarafoglou et al., 2009). Neither type 2 or type 3 present with succinylacetone in the urine.

#### Nutrition Management of Tyrosinemia

The nutrition goals for patients with tyrosinemia are to provide a biochemical environment that supports normal growth and development of intellectual potential. Diet therapy is recommended for tyrosinemia; a diet prescription includes a restricted intake of intact protein (to limit the phe and tyr) and inclusion of medical foods free of phe and tyr, but containing amino-acid-based protein, calories, fat, carbohydrates, and micronutrients (Acosta, 2010). Nutrition guidelines are based on age, energy and protein intake, and plasma phe and tyr levels (Acosta, 2010). Total protein needs from intact protein and amino-acid-based protein are higher than in the healthy population because amino-acid-based medical foods are rapidly oxidized to ketoacids (Acosta, 2010). Energy requirements are determined with the goal of supporting normal growth and preventing catabolism (Acosta, 2010). Most medical foods are supplemented with vitamins and minerals; however, micronutrient needs may be higher in this population (Acosta, 2010).

During illness and at diagnosis, the goal is the rapid reduction of plasma tyr levels to the treatment range as well as the prevention of catabolism. After diagnosis is confirmed, nutrition support should be initiated (Acosta & Yannicelli, 2001). The plasma levels of phe and tyr should be monitored regularly and maintained within the treatment range using prescribed amounts of medical food and intact protein (Acosta & Yannicelli, 2001). During times of good health, the goal is to achieve phe and tyr levels within the treatment range goals while promoting growth and development; if levels of either amino acid are low, supplementation may be warranted. Tyrosinemia patients are prescribed a specific amount of dietary phe and tyr,

or protein through intact protein (Acosta, 2010); medical foods meet the remainder of the protein goals. During illness, a phe- and tyr-free diet that is high in calories may be recommended (Acosta, 2010; Sarafoglou et al., 2009).

The long-term management for tyrosinemia depends on the type. Type 1 is now treated with a medication, nitisinone (also called NTBC, 2-(2-nitro-4-trifluoromethyl-benzoyl)-1, 3-cyclohexanedione, or orfadin), which prevents the synthesis of succinylacetone (Acosta, 2010); treatment with this medication eliminates the acute hepatic and porphyric crises. Nutrition management in conjunction with nitisinone are essential in the treatment of patients with type 1 (Acosta, 2010). Nitisinone does not completely eliminate the risk of developing hepatic cancer (Acosta, 2010); the risk mainly depends on the age at which the medication is started (Sarafoglou et al., 2009). Treatment with diet therapy and nitisinone has greatly improved the survival of patients and reduced the need for liver transplantation (Acosta, 2010). Prognosis is excellent for patients with types 2 and 3 tyrosinemia with nutrition management alone (Acosta, 2010).

## Disorders of Branched-Chain Amino Acid Metabolism

### Maple Syrup Urine Disease

**Maple syrup urine disease (MSUD)** is an autosomal recessive organic acid disorder of the branched-chain amino acids (BCAAs) (Acosta, 2010; Acosta & Yannicelli, 2001; Hansen & Horslen, 2008; Sarafoglou et al., 2009). The BCAAs, which are leucine, isoleucine, and valine, compose 40% of all essential amino acids in the human body. Their function is to assist with protein synthesis, signal for cell growth, utilize glucose, and metabolize lipids (Acosta, 2010; Zhou, Lu, Gao, Wang, & Sun, 2012) (see **Figure 5.2**). MSUD is caused by a deficiency in the branch-chain alpha ketoacid dehydrogenase enzyme (BCKAD), which causes an accumulation of BCAAs in serum, cerebrospinal fluid, and urine (Cakir, Teksam, Kosehan, Akin, & Koktener, 2011; Sarafoglou et al., 2009;

**maple syrup urine disease (MSUD)** An autosomal recessive disorder characterized by a defect in the metabolism of the branched-chain amino acids isoleucine, leucine, and valine caused by a deficiency of branched-chain α-ketoacid dehydrogenase, resulting in the accumulation of the branched-chain amino acids in the plasma.

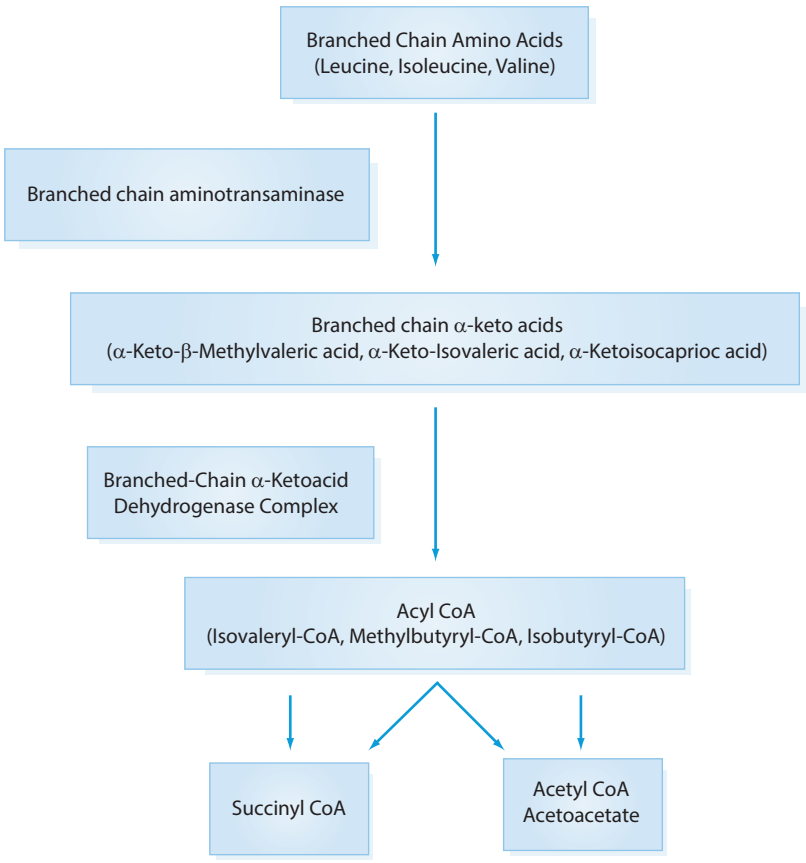



**FIGURE 5.2** Branched-chain amino acid metabolism

Zhou et al., 2012). Coenzymes required for catabolism of BCAAs include thiamine pyrophosphate, lipoic acid, coenzyme A (CoA), and nicotinamide adenine dinucleotide (NAD+) (Acosta, 2010). The incidence of MSUD is 1 in 150,000–215,000 in the general population; however, it is much more common in the Mennonite population at 1 in 200–760 (Acosta & Yannicelli, 2001; Raghuveer, Garg, & Graff, 2006; Sarafoglou et al., 2009; Walsh & Scott, 2010).

MSUD is detected with elevated plasma leucine levels on a newborn screening test. The disorder is named for the maple syrup smell in urine and ear wax from the buildup of ketoacids. There are several MSUD phenotypes, the most common (80% of all cases) and severe is classical MSUD, with only 0–2% BCKAD activity (Acosta, 2010). The intermittent phenotype presents with symptoms later in life and has 5–20% BCKAD activity (Acosta, 2010). There have also been reports of two other phenotypes, thiamine-responsive MSUD and dihydrolipoyl dehydrogenase (E3)-deficient phenotype. The latter is characterized by lactic acidosis and deficiencies in three dehydrogenase complexes (Acosta, 2010; Delis, Michelakakis, Katsarou, & Bartsocas, 2001).

Although MSUD is detected on NBS, often individuals present with signs and symptoms prior to NBS results. The initial symptoms present between 1 and 5 days of life and include poor feeding and sucking, vomiting, lethargy, muscle fatigue, epigastric pain, and irregular respiration (Acosta & Yannicelli, 2001; Chuang & Shih, 2001; Elsas & Acosta, 1999; Holmes Morton, Strauss, Robinson, Puffenberger, & Kelley, 2002; Sarafoglou et al., 2009; Scaini et al., 2012). If left untreated, within 10 days these symptoms progress to rigidity with alternating flaccidity, progressive loss of moro reflux, acute neurological dysfunction, seizures, apnea, cerebral edema, decreased cognitive ability, anorexia, sleep disturbances, hallucinations, dystonia, ataxia, stupor, coma, and eventually death (Acosta & Yannicelli, 2001; Chuang & Shih, 2001; Elsas & Acosta, 1999; Holmes Morton et al., 2002; Sarafoglou et al., 2009; Scaini et al., 2012; Walsh & Scott, 2010). If MSUD is not treated, the long-term symptoms and progression of disease when left untreated include severe neurological injury, retarded physical and mental development and disabilities, optic atrophy, nystagmus, bilateral ptosis, strabismus, and cortical blindness (Acosta, 2010; Acosta & Yannicelli, 2001; Hansen & Horslen, 2008).

### Nutrition Management of Maple Syrup Urine Disease

The mainstay of nutrition treatment of MSUD is to restrict BCAAs and, therefore, all intact protein. Set BCAA requirements for individuals with MSUD by age group are used as guidelines, along with plasma amino acid laboratory values to prescribe adequate BCAAs while maintaining metabolic control. On average, individuals tolerate 50–65% of total protein from intact protein (Acosta, 2010). The remaining protein needs should be met with BCAA-free medical food. These medical foods contain calories, fat, carbohydrates, and micronutrients and generally come in the form of formula (Acosta, 2012; Hansen & Horslen, 2008; Holmes Morton et al., 2002; Sarafoglou et al., 2009). Total protein intake (intact protein and BCAA-free protein) is recommended to be higher than in the healthy population because the body uses amino-acid-based products less efficiently than it does intact protein.

During times of health (i.e., no stress), it is necessary for patients to receive a consistent amount of intact protein. Intact protein restriction often overrestricts isoleucine and valine because they are less concentrated in protein than leucine. For this reason, supplemental isoleucine and valine are needed to prevent deficiencies and for optimal protein synthesis (Acosta, 2010; Acosta & Yannicelli, 2001; Holmes Morton et al., 2002). Energy requirements are similar to the recommendations for healthy people; however, fluid requirements are higher, to facilitate optimal excretion of toxic metabolites (Acosta, 2010; Behrman, Kliegman, & Jenson, 2000). Because micronutrient needs may be higher in this population, BCAA-free medical foods may need to be supplemented with additional micronutrients (Acosta, 2010; Barschak, Sitta, & Deon, 2007; Gropper et al., 1993).

Acute metabolic decompensation is serious and life-threatening for individuals with MSUD and hospitalization is recommended (Acosta, 2010; Holmes Morton, 2002; Sarafoglou et al.,

### CRITICAL Thinking

Endogenous catabolism of protein and amino acids is a problem in inborn errors of metabolism. It is thought that the endogenous catabolism leads to worsening symptoms and disease progression. Frequently, endogenous catabolism of protein is caused by restrictive diets. It is suggested that therapy should be directed toward control of endogenous protein turnover rather than the restriction of dietary protein in treatment of inborn errors of protein metabolism.

2009). The treatment goal during times of stress is to rapidly reduce toxic molecules, to decrease leucine levels in blood, to prevent catabolism, and to prevent cerebral edema. During initiation of treatment in the neonatal period, clinicians should not wait for confirmation of diagnosis to implement nutrition support (Acosta & Yannicelli, 2001). Nutrition support consists of high-energy BCAA-free medical food and adequate fluid. The route of feeding is determined by clinical status; oral feeds are the first choice, then nasogastric feeds when individuals are unable to take 100% of needs orally, and as a last option parental nutrition should be initiated. It is recommended to introduce BCAA-free medical food as soon as it is tolerated and to start isoleucine and valine supplements immediately because leucine levels will not decrease if these amino acids are deficient (Acosta & Yannicelli, 2001). Once plasma leucine levels reach the upper limit of treatment range, intact protein can be added to the diet and the amount will be adjusted based on plasma levels (Acosta & Yannicelli, 2001). During times of stress and illness (other than the initial diagnosis period), a "sick day diet" (i.e., 100% leucine-free, high in calories and fluids) should be prescribed to prevent catabolism; in addition, supplementation of isoleucine and valine should continue (Acosta, 2010; Holmes Morton et al., 2002).

The long-term goals and outcomes of MSUD treatment include normal growth and body composition, which can be achieved with proper nutrition management, calories, protein, and BCAAs (Acosta, 2010; Henstenburg, Mazur, Kaplan, & Stallings, 1990). It is imperative to prevent catabolism, maintain usual growth, and closely monitor BCAAs during times of illness (Acosta, 2010; Holmes Morton et al., 2002; Strauss et al., 2006). An additional outcome of importance is neurocognitive function, which varies based on the timeliness of diagnosis, genotype, and adherence to treatment (Kaplan, Ficicioglu, Mazur, Palmieri, & Berry, 2006; Shellmar et al., 2011; Walsh & Scott, 2010). With poor metabolic control, there is an increase in attention deficit disorder, impulsivity, hyperactivity and mental illness, pancreatitis, and recurrent infections (Acosta, 2010; Strauss et al., 2006). Early diagnosis and aggressive medical management can result in normal intellectual outcomes (Acosta, 2010; Sarafoglou et al., 2009).

### Disorders of Leucine Catabolism

**Isovaleric academia**, or isovaleryl-CoA dehydrogenase deficiency (IVA), and 3-methylcrotonyl-CoA carboxylase deficiency (3-MCC) are autosomal recessive organic acidemia disorders of leucine catabolism (Acosta, 2010; Sarafoglou et al., 2009). Leucine, the amino acid involved, is important for protein synthesis. IVA is caused by a deficiency of isovaleryl-CoA dehydrogenase (IVD) that leads to a buildup of isovaleryl-CoA, forming isovalerylglycine, with help from glycine-N-acylase. The detoxification pathway for IVA is isovaleryl CoA with an added amino group of glycine (Acosta, 2010; Sarafoglou et al., 2009). The toxic metabolite (isovaleric acid) elevates during stress because the capacity for an alternative pathway is taxed (Acosta, 2010). 3-MCC is caused by a deficiency in B-methylcrotonyl CoA carboxylase enzyme, which is biotin dependent, resulting in excretion of metabolites in the urine and leading to secondary carnitine deficiency (Acosta, 2010). The incidence of IVA in the United States is 1 in 250,000, and 3-MCC, the most common organic aciduria, has an incidence of 1 in 36,000–50,000 newborns worldwide (Acosta, 2010).

**isovaleric acidemia**
A disorder of leucine metabolism caused by a deficiency of isovaleryl-CoA characterized by the excessive production of isovaleric acid upon ingestion of protein or during infectious episodes and resulting in severe metabolic acidosis.

IVA is detected with elevated plasma isovalerylcarnitine (C5), and 3-MCC is detected by elevated B-hydroxyisovalerylcarnitine (C50H) on NBS (Acosta, 2010). IVA can also be detected by a "sweaty foot" odor presentation caused by an accumulation of isovaleric acid (Sarafoglou et al., 2009). There are three IVA phenotypes. The most common (50% of cases) is the acute and most severe neonatal onset, in which individuals present in metabolic crisis within the first few days of life (Acosta, 2010; Sarafoglou et al., 2009). This will become the chronic intermittent type after the first few years of life (Acosta, 2010). The chronic intermittent phenotype of IVA presents in childhood with recurrent metabolic crises from stressors (high protein intake, infections, and catabolism); however, episodes become less frequent and less severe as a child ages (Sarafoglou et al., 2009). The mild or asymptomatic phenotype of IVA was recently identified; however, not much is known except that these individuals appear to be asymptomatic and do not require treatment (Acosta, 2010; Sarafoglou et al., 2009).

The clinical symptoms of both IVA and 3-MCC can vary based on phenotype. For example, approximately 27% of individuals with 3-MCC are asymptomatic (Acosta, 2010; Staddler, Polanetz, & Maier, 2006). The neonatal onset of IVA and 3-MCC are quite similar in presentation with symptoms of poor feeding, vomiting, and diarrhea. Without immediate medical intervention, these symptoms progress to tachypnea, lethargy, hypothermia, acidosis, ketosis, seizures, hyperammonemia, progressive alteration in consciousness, cerebral edema, hemorrhage, deep coma, and possibly death (Acosta, 2010; Coude et al., 1982; Loots, Mienie, & Erasmus, 2007; Sarafoglou et al., 2009).

### Nutrition Management of IVA and 3-MCC

The nutrition requirements for IVA include a restriction of total intact protein (to restrict leucine intake) and supplementation of medical food and other supplements. Intake recommendations are based on age. Energy and protein intake in IVA should be adjusted based on presence of isovaleric acid in urine and growth (Acosta, 2010). Supplementation with leucine-free medical food is recommended so total protein (intact and leucine-free) is more than the Recommended Dietary Allowance (RDA) for healthy individuals to meet protein requirements (Acosta, 2010; Sarafoglou et al., 2009). Also, supplementation of glycine and L-carnitine is recommended to optimize detoxification, to maximize excretion of isovaleryl-CoA metabolites, and to reduce the severity and frequency of metabolic crisis (Acosta, 2010; Sarafoglou et al., 2009).

Individuals with IVA have shown higher intact protein tolerance when supplemented with glycine and carnitine (Acosta, 2010). There is currently no consensus in the medical community as to whether to restrict intact protein in individuals affected by 3-MCC. If a clinic does restrict intact protein, general guidelines are 50% from leucine-free medical food and the remainder from intact protein (Acosta, 2010; Arnold et al., 2008). Individuals with 3-MCC should get at least the RDA for age for overall protein. L-carnitine should also be supplemented in 3-MCC patients, regardless of whether there is a protein restriction, to prevent secondary carnitine deficiency (Acosta, 2010; Arnold et al., 2008). For both IVA and 3-MCC, energy needs are similar to those of the healthy population and should be adequate for growth and development (Acosta, 2010; Arnold et al., 2008).

The most critical times for management of IVA and 3-MCC are during initiation of nutrition intervention and during times of stress or illness. For individuals with acute neonatal onset, nutrition support should be initiated immediately, sometimes before confirmation of diagnosis (Acosta & Yannicelli, 2001). Initiation should begin with high-energy, leucine-free feeds and adequate fluid. Based on the clinical status of the neonate, feeds should be oral; however, if an individual is unable to consume 100% of nutrient needs orally, enteral or parental nutrition may be necessary (Acosta & Yannicelli, 2001). L-carnitine and glycine (for IVA) supplements should be added to feeds immediately. Once plasma leucine levels reach the upper limit of the treatment range (> 100 µmol/L or > 1.31 mg/dL), intact protein can be added and adjusted based on plasma levels (Acosta & Yannicelli, 2001). During times of stress, individuals with IVA require completely leucine-free diets that are high in energy and high in leucine-free protein as well as increased glycine and L-carnitine supplementation (Acosta, 2010; Sarafoglou et al., 2009). During times of stress, individuals with 3-MCC require strict "sick day" precautions, consisting of an increase in leucine-free energy, L-carnitine supplementation, and a decrease in intact protein as well as adequate fluids. Individuals affected by 3-MCC should never fast (Acosta, 2010).

Long-term nutrition management for both IVA and 3-MCC should consist of strict sick day precautions and avoidance of catabolism and fasting. Continued glycine (for IVA) and L-carnitine supplementation as well as leucine-restricted diets (for IVA and sometimes for 3-MCC) that are supplemented with leucine-free medical foods to meet macronutrient and micronutrient needs are recommended (Acosta, 2010; Sarafoglou et al., 2009). The long-term outcomes for well-treated IVA is normal growth and development; however, the clinical outcome may vary based on the timing of diagnosis (Castorina, Rigante, Antuzzi, Cannizzaro, & Ricci, 2008). Normal cognitive outcomes and psychomotor development can be achieved; however, demonstration of mild to severe mental retardation and neurologic impairment is dependent on initial presentation and treatment. Since the advent of NBS, mortality from IVA has

notably decreased (Acosta, 2010; Sarafoglou et al., 2009). Long-term outcomes for 3-MCC are not well known. It appears that early detection and appropriate treatment reduces morbidity and mortality. Unfortunately, there continue to be reported cases of cerebral edema, severe neurologic dysfunction, and death in untreated patients (Acosta, 2010).

## Disorders of Other Amino Acid Metabolism

### Propionic Acidemia and Methylmalonic Acidemia

Organic acidurias (OAs) are a group of disorders characterized by an increased excretion of organic acids in the urine. Organic acids occur as intermediates in a host of metabolic pathways including catabolism of amino acids, oxidation of fatty acids, the TCA cycle, and cholesterol synthesis. OAs are caused by a deficiency of an enzyme involved in the breakdown of amino acids and can result in the accumulation of precursors or metabolites that are toxic. The clinical manifestations of these disorders are the result of damage from toxic metabolites to different organs including but not limited to the brain, liver, kidney, pancreas, and retina. The range of clinical and biochemical manifestations of OAs is extensive (Sarafoglou et al., 2009). This section will focus on propionic aciduria (PROP) and methylmalonic aciduria (MMA), which are classified as "intoxication type" OAs (Sarafoglou et al., 2009). These diseases may also be referred to as organic acidurias, organic acidemias, organic acid disorders, or organoacidopathies (Sarafoglou et al., 2009).

Both PROP and MMA are autosomal recessive organic acidurias that involve the defective metabolism of isoleucine (ile), valine (val), methionine (met), threonine (thr), and odd-chain fatty acids (Acosta, 2010). Under normal conditions, ile, val, met, thr, and the odd-chain fatty acids are metabolized to propionyl-CoA and methylmalonyl-CoA for entry into the Krebs cycle via succinyl-CoA. MMA and PROP are caused by deficiencies in the enzymes methylmalonyl-CoA mutase and propionyl-CoA carboxylase, respectively. Patients could also have a subgroup of MMA, a cobalamin defect that is caused by the impaired synthesis of adenosylcobalamin, a coenzyme necessary for the function of methylmalonyl-CoA mutase. Without sufficient enzyme activity, there is an accumulation of organic acids that leads to a buildup of toxic metabolites (Sarafoglou et al., 2009). The incidence of MMA and PROP varies between 1 in 20,000 to 1 in 200,000 worldwide, with great variance by region (Acosta, 2010; Sarafoglou et al., 2009).

MMA and PROP are detected with an elevated plasma propionylcarnitine (C3) on the acylcarnitine profile. Confirmatory testing can verify elevations of C3, as well as other metabolites, and reduce enzyme fibroblast activity. Without screening, severe cases of MMA and PROP present with life-threatening illness during the neonatal period, with hypoglycemia, hyperammonemia, ketosis, and metabolic acidosis (Acosta, 2010; Sarafoglou et al., 2009). Other clinical symptoms include vomiting, lethargy, poor feeding, and coma. A genotype/phenotype correlation has been established, and disease outcome depends on residual enzyme activity or genotype (Acosta, 2010; Sarafoglou et al., 2009). The symptoms of the diseases vary depending on the severity of the disease. MMA or PROP can be a catastrophic disease in the neonatal period or could present later in life. Metabolic decompensations in infancy or early childhood are similar to those in the neonatal period. These decompensations can be triggered by infection, extended fasting, or exposure to excess protein load (Acosta, 2010). Later symptoms can include progressive psychomotor retardation, developmental delay, cognitive impairment, seizures, movement disorders, osteoporosis, pancreatitis, and cardiomyopathy. Those with MMA may also develop renal disease (Acosta, 2010; Sarafoglou et al., 2009).

### Nutrition Management of Propionic Acidemia and Methylmalonic Aciduria

The nutrition management goal for patients with MMA and PA is to reduce toxic tissue concentrations of organic compounds while promoting anabolism, normal growth, and nutrition status (Acosta, 2010). Total protein needs are higher than in the healthy population because amino-acid-based medical foods are rapidly oxidized to ketoacids (Acosta, 2010). Patients with MMA and renal disease may require lower total protein to help preserve renal function (Acosta, 2010). The goal is to restrict the essential amino acids (ile, met, thr, and val) without preventing deficiencies and to restrict dietary sources of odd-chain fats (Acosta, 2010; Acosta & Yannicelli, 2001).

Medical foods (free of ile, met, thr, and val) and limited intact protein intake are cornerstones of nutrition management (Acosta, 2010). Typically, intact protein provides half of the total prescribed

protein, with amino-acid-based medical foods providing the remainder (Acosta, 2010). Intact protein intake depends on tolerance and age-specific requirements (Acosta, 2010; Sarafoglou et al., 2009). Energy needs have been reported to be lower than the RDA and may be the result of decreased lean body mass (Acosta, 2010); however, energy intakes at or above the RDA are indicated for infants and young children when metabolic crises are common to prevent or overcome catabolism. Fasting must be avoided because it leads to increased production of odd-chain fatty acids; oxidation of these fatty acids contributes to proprionate production and subsequent toxic metabolites for PROP and MMA patients (Acosta, 2010). It is also important to maintain adequate hydration to prevent dehydration and promote excretion of toxic metabolites (Acosta, 2010). For micronutrients, the goal is to meet more than 100% of the Dietary Reference Intakes for age (particularly thiamine; vitamins C, E, and D; and calcium) (Acosta, 2010).

Aggressive initiation of nutrition management during illness is imperative. The goal is to eliminate all sources of protein that contain the offending amino acids to help reduce toxic metabolites, give intravenous glucose with electrolytes to maintain hydration needs, and provide additional calories to suppress catabolism. Enteral nutrition should be used if the patient is able to tolerate feeds; parenteral nutrition with lipids should be initiated immediately with the goal of initiating enteral nutrition as soon as possible. Intact protein should be started within 24 to 48 hours to prevent deficiency. Carnitine therapy should be continued (Acosta, 2010; Sarafoglou et al., 2009). An emergency protocol is also essential, outlining the goals to suppress protein catabolism and inhibit plasma accumulation of toxic metabolites and ammonia by giving an energy glucose supply above the basal metabolic demand, treating the illness, decreasing natural protein intake to decrease organic acid production, enhancing detoxifying mechanisms and urinary excretion of organic acids, preventing carnitine depletion, and aggressively treating hyperammonemia, dehydration, and acidosis (Acosta, 2010; Sarafoglou et al., 2009). The long-term management goals of PROP and MMA are to promote normal growth and development as well as reduce metabolic crisis. Several medications and supplements are used to manage patients with MMA and PROP. Carnitine is given in pharmacologic doses because it facilitates the excretion of toxic acyl compounds and prevents a secondary deficiency (Acosta, 2010; Sarafoglou et al., 2009). Coenzymes are also given in therapeutic doses to enhance any residual enzyme activity; vitamin $B_{12}$ is given for MMA and biotin is provided for PROP (Acosta, 2010). Metronidazole has been used to reduce gut bacteria that synthesizes propionate (Acosta, 2010; Sarafoglou et al., 2009). Soluble fiber or laxatives may help to reduce constipation and decrease gut proprionate. Bicitra (sodium citrate and citric acid) is often prescribed as a urine-alkalizing agent (Acosta, 2010). MMA and PROP patients require frequent monitoring of metabolic labs, including urine ketones (Acosta, 2010). Gastrostomy tube placement is often recommended for treatment during illness or to provide nutrition because anorexia can occur as an inherent component of these diseases (Acosta, 2010). Liver and/or kidney transplant may be indicated (Acosta, 2010; Sarafoglou et al., 2009). Bone health should be assessed (Acosta, 2010).

The outcomes of nutrition management for MMA and PROP depend upon disease severity, enzyme function, and age at presentation. Aggressive medical intervention, effective chronic nutrition management, and in some cases transplantation have resulted in patients living longer and more healthy lives than previously. Despite advances, the majority of patients with severe disease still suffer from chronic, debilitating symptoms (Acosta, 2010).

## Disorders of Nitrogen Metabolism

### Urea Cycle Disorders

**Urea cycle disorders (UCD)** are a group of autosomal recessive (except for ornithine transcarbamylase) disorders involving five different enzymes in the urea cycle. Urea cycle disorders are among the most common inborn errors of metabolism affecting the liver (Acosta & Yannicelli, 2001; Lanpher, Gropman, Chapman, Lichter-Konecki, & Summar, 2011; Morioka et al., 2005). The six disorders that make up urea cycle disorders include the following: ornithine transcarbamylase (OTC), carbamyl phosphate synthetase 1 (CPS1), argininosuccinate synthetase (citrullinemia), N-acetyl glutamate synthase (NAGS), argininosuccinate lyase (ASL or ASA Lyase), and

**urea cycle disorders (UCD)** Inborn errors of urea synthesis caused by a deficiency of the enzymes in the urea cycle (carbonyl phosphate synthetase, n-acetylglutamate synthetase, ornithine transcarbamylase, arginosuccinic acid synthase, arginosuccinate lyase, and arginase).

arginase (ARGI) (Lanpher et al., 2011). Urea cycle disorders are characterized by a defect in waste metabolism (nitrogen). When protein is broken down, it produces nitrogen that is then converted to ammonia and must go through the urea cycle in the liver (Lanpher et al., 2011). The urea cycle is the sole source of endogenous production of arginine, ornithine, and citrulline. It is also the principal mechanism for the clearance of waste nitrogen and the metabolism of other nitrogenous metabolic compounds (Lanpher et al., 2011) (see **Figure 5.3**). When one of the enzymes is not functioning properly, there is a rapid accumulation of ammonia in the blood that can be lethal (Lanpher et al., 2011). The incidence of urea cycle disorders in the United States is 1 in 25,000–100,000 (Morioka et al., 2005; Sarafoglou et al., 2009).

The severity of the urea cycle disorder is based on position of the defective enzyme in the cycle and the severity of the defect (Lanpher et al., 2011; Sarafoglou et al., 2009). For example, OTC in males is a complete absence of OTC enzyme activity and is one of the most severe disorders. Approximately 15% of females who are carriers of OTC develop hyperammonemia during their lifetime and may require chronic medical management (Lanpher et al., 2011). CPS1 controls the first step of the urea cycle and is the most severe of the urea cycle disorders (Lanpher et al., 2011). NAGS is caused by severe deficiency or total absence of enzyme activity of any of the first four enzymes (CPS1, OTC, ASS, and ASL) (Lanpher et al., 2011). Citrullinemia, caused by a deficiency in argininosuccinate synthetase, has two forms that can vary greatly in presentation (neonatal or adult onset) and severity. ASL or ASA lyase is characterized by a deficiency in argininosuccinate lyase and can present in the neonatal period or later in life. ARGI is a more subtle disorder involving neurologic symptoms and causes hyperargininemia; however, neonatal hyperammonemia has been rarely reported (Lanpher et al., 2011).

Although UCDs are detected on NBS, individuals often present with signs and symptoms prior to

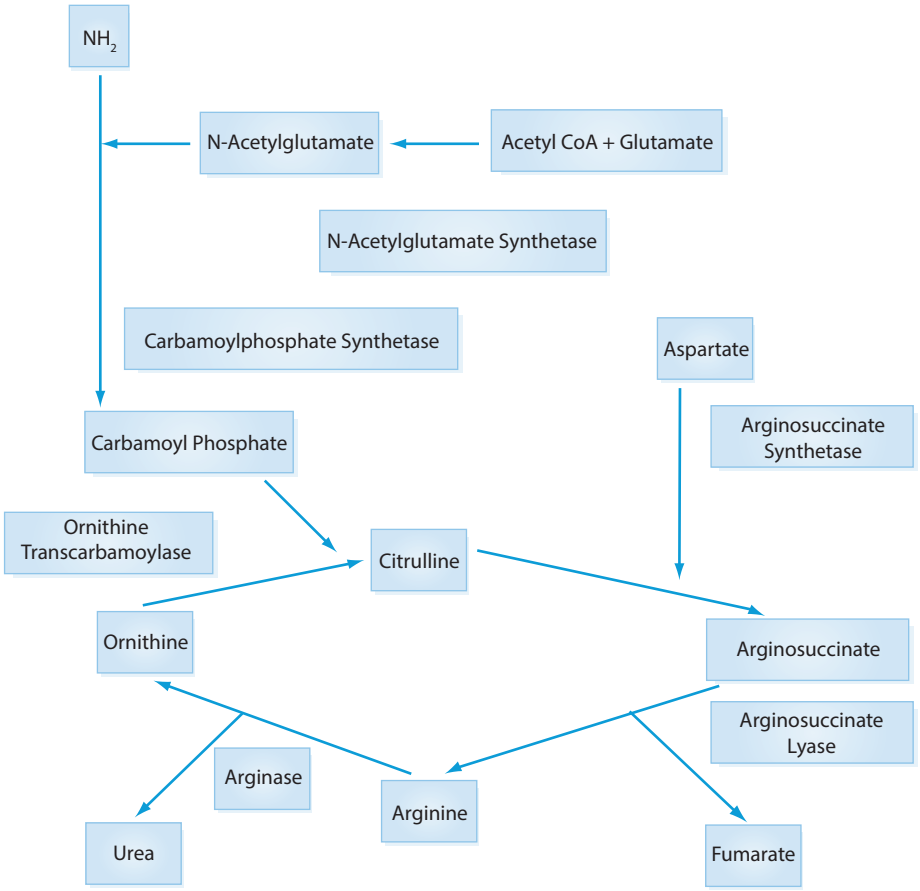



**FIGURE 5.3** Urea cycle

NBS results. Neonatal onset UCDs int he first few days of life with symptoms of vomiting, lethargy, drowsiness, poor feeding/failure to feed, and hypotonia (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009). These clinical symptoms progress rapidly into loss of thermoregulation, hyperventilation, hypoventilation, ataxia, stupor, cerebral edema, seizures, coma, shock, and death (Acosta, 2010; Acosta & Yannicelli, 2001; Lanpher et al., 2011; Sarafoglou et al., 2009).

### Nutrition Management of Urea Cycle Disorders

The backbone of nutrition management in UCD is a restriction of all protein in the diet. All protein breakdown enters the urea cycle so, unlike in other disorders, no specific amino acid (AA) is restricted but all protein (Acosta, 2010; Leonard, 2001). There are specific guidelines based on age and severity of disease to determine the overall restriction of intact protein (Lanpher et al., 2011). It is recommended to supplement diet with nonessential AA-free medical food that is high in BCAA to reduce nitrogen waste and meet essential amino acid (EAA) needs (Acosta, 2010). Individuals with severe disorders might tolerate less than the RDA in total protein (intact and EAA-rich medical food) (Lanpher et al., 2011). General guidelines are to start with 50% of total protein from intact protein supply and provide the remainder from EAA-rich medical food (Acosta, 2010; Lanpher et al., 2011; Urea Cycle Disorders Conference Group [UCD], 2001). It is important to monitor plasma AA levels to prevent and correct deficiencies quickly (Acosta, 2010; Leonard, 2001). Energy needs are generally higher, especially in the neonatal period, because all efforts to prevent catabolism should be taken. However, it is necessary to balance needs to prevent excess weight gain and obesity, especially later in childhood (Acosta, 2010; Leonard, 2001). Fluid recommendations are similar to those of healthy individuals but need to be closely monitored because both dehydration and overhydration can lead to problems in this population (Acosta, 2010).

The initial few days of life are extremely important for the outcomes in UCD and immediate initiation of nutrition management is critical; for that reason confirmation of diagnosis is not needed to initiate treatment (Acosta, 2010; UCD, 2001). All protein should be quickly removed from the diet and replaced with very high energy intakes provided by intravenous lipids and glucose to reverse catabolism (Acosta, 2010; UCD, 2001). Within 48 to 72 hours, EAA-rich medical food should be added to prevent further protein catabolism (Acosta, 2010; UCD, 2001). Although oral or enteral feedings are the preferred source of nutrition, parental nutrition is most often necessary to ensure that adequate calories are provided for individuals with neonatal onset (Acosta, 2010; UCD, 2001). Nitrogen-scavenging drugs (i.e., sodium benzoate, phenylacetate), which are compounds that remove nitrogen by alternative pathways, should be added as soon as possible and electrolytes should be monitored closely during this time (Acosta, 2010; UCD, 2001). If ammonia levels do not decrease with high calories and nitrogen scavengers, dialysis is necessary (Acosta, 2010; UCD, 2001). An additional important part of therapy is preventing catabolism and hyperammonemia episodes by minimizing stress at all times (Sarafoglou et al., 2009). Sick day diets that are high in calories and low in protein or protein-free with adequate fluid are necessary. Gastrostomy tubes should be considered for quick administration of calories and improved adherence to sick day diets (Acosta, 2010; UCD, 2001). Catabolism and dehydration need to be completely avoided; special precautions should be in place for all medical procedures (Acosta, 2010; Leonard, 2001; UCD, 2001).

The primary goal of long-term management is to prevent hyperammonemia episodes while also promoting growth and development (Acosta, 2010; Leonard, 2001; Sarafoglou et al., 2009; UCD, 2001). Along with protein-restricted diets, nitrogen scavenger drugs are needed lifelong (Lanpher et al., 2011; Leonard, 2001). There are other treatment recommendations that vary by disorder (i.e., citrulline supplementation, which allows for removal of two nitrogen molecules from circulation) for individuals with OTC, NAGS, and CPS1 (Lanpher et al., 2011). For individuals with ASL and ASA lyase, supplementation of arginine prevents arginine deficiency and assists with alternative pathways of nitrogen excretion (Lanpher et al., 2011). For individuals with severe disorders and those with recurrent hyperammoniemia episodes, liver transplant is a viable treatment option (UCD, 2001).

The long-term outcomes and results of nutrition management have shown improved survival with rapid nutritional and medical management over the last decade, but overall outcomes continue to be poor overall (Acosta, 2010). Severe urea cycle disorders have a 50% to 65% mortality rate (Acosta, 2010). Individuals who survive

the neonatal period demonstrate poor cognitive outcomes with neurological deficiencies and mental retardation (Acosta, 2010; Acosta & Yannicelli, 2001; Hansen & Horslen, 2008).

## Disorders of Fatty Acid Oxidation

### Mitochondrial Fatty Acid Oxidation Disorders

Fatty acid oxidation disorders (FAODs) are a group of rare inborn errors of metabolism that result from an impairment in mitochondrial oxidation of fatty acids, resulting in an accumulation of fatty acids in the body. The mitochondria are responsible for the oxidation of fatty acids with carbon lengths of 20 or less. Mitochondrial β-oxidation involves the transport of activated acyl-CoAs into the mitochondria with sequential removal of 2-carbon acetyl-CoA units. These units are then used as fuel for the tricarboxylic acid (TCA) cycle or the production of ketone bodies, which subsequently form reducing equivalents to be used by the electron transport chain for the production of ATP (Shils, Olson, Shike, & Ross, 1999).

Free fatty acids must be activated by a corresponding acyl-CoA thioester before undergoing β-oxidation. Short- and medium-chain carboxylic acids (< 10 carbons in length) can directly enter the mitochondrial matrix, where they are activated. However, long-chain fats (> 10 carbons in length) are activated in the cytoplasm and require active transport into the mitochondria. A transporter enzyme and carnitine are both required. Long-chain acyl-CoAs are conjugated to carnitine by carnitine palmitoyl transferase I (CPT-1), an enzyme located on the outer mitochondrial membrane. CPT-1 catalyzes the conversion of long-chain acyl-CoAs to acylcarnitines. The acylcarnitines are then transported into the mitochondria. Once inside the mitochondrial matrix, the reaction is reversed by carnitine palmitoyl transferase II (CPT-2), producing free carnitine and long-chain fatty acyl-CoA (Acosta, 2010; Acosta & Yannicelli, 2001; Shils et al., 1999).

After entering the mitochondria, acyl-CoAs of all chain lengths undergo a series of enzymatic reactions that result in a release of a 2-carbon unit acetyl-CoA and a new acyl-CoA molecule that is two carbons shorter in length. This results in a repeating 4-enzyme cycle, with each cycle releasing one molecule of acetyl-CoA. The first reaction is performed by one of five acyl-CoA dehydrogenases, depending upon the structure and chain length of the fatty acid substrate, including very long-chain, long-chain, medium-chain, and short-chain acyl-CoA dehydrogenases CoA (Acosta, 2010; Acosta & Yannicelli, 2001; Sarafoglou et al., 2009; Shils et al., 1999). Genetic defects of any of these acyl-CoA dehydrogenases result in an inability to appropriately metabolize the corresponding carbon chain, thereby causing a fatty acid oxidation disorder of very long-chain, long-chain, medium-chain, or short-chain fatty acids.

Fatty acids are a primary metabolic fuel for the body during times of prolonged fasting and are a direct source of energy for heart and skeletal muscle (Acosta & Yannicelli, 2001). During periods of reduced calorie intake resulting from fasting, febrile illness, and/or increased muscular exertion, the normal endocrine response is mobilization of lipid stores and generation of free fatty acids at the plasma membrane to meet the body's increased energy demands. Any person affected by a FAOD presents with an abnormal response to fasting, characterized by low ketone production during times of illness or stress when energy demands are increased (Acosta & Yannicelli, 2001). Typical symptoms of untreated fatty acid oxidation disorders include lethargy, muscle weakness, seizures, coma, hepatomegaly, cardiomyopathy, and possibly death. On autopsy, fatty infiltration of organs is often found. Additionally, siblings of children who have died from sudden infant death syndrome (SIDS) should be evaluated for a possible defect of fatty acid oxidation (Acosta & Yannicelli, 2001). Symptoms may first occur in the newborn period and are often induced from fasting or illness with vomiting and diarrhea (Acosta & Yannicelli, 2001). However, the development of pigmentary retinopathy or peripheral neuropathy is a symptom unique to long-chain-hydroxy-acyl-CoA dehydrogenase deficiency (Acosta & Yannicelli, 2001).

The incidence of fatty acid oxidation defects varies, depending on the type of defect. However, the overall incidence in some populations may be as high as 1 in 9,000 births (Acosta & Yannicelli, 2001). A fatty acid oxidation defect may be diagnosed under tandem mass spectroscopy when newborn screening is performed. If NBS testing results indicate concern for FAOD, an acylcarnitine profile is performed for confirmatory testing of an FAOD diagnosis. Each fatty acid oxidation

disorder has a unique acylcarnitine profile; these test results facilitate diagnosis of the specific type of FAOD. The presence of urine organic dicarboxcylic aciduria is also suggestive of an FAO defect. Individuals with an altered acylcarnitine profile or urine organic acids, or the symptoms often associated with fatty acid oxidation disorders (listed later), should have confirmatory testing performed by a metabolic center.

Short-chain acyl CoA dehydrogenase (SCAD) deficiency involves a defect in the oxidation of short-chain fatty acids ($C_{4-6}$) that prevents the body from converting short-chain fatty acids into energy, especially during periods of fasting. The incidence is approximately 1 in 40,000–100,000 newborns (Genetics Home Reference, 2009c).

Medium-chain acyl CoA dehydrogenase (MCAD) deficiency involves a defect in the oxidation of medium-chain fatty acids ($C_{6-10}$). It is the most common defect in patients with an FAOD. According to Genetic Home Reference (Genetics Home Reference, 2009b), the estimated incidence of MCAD deficiency in the United States is 1 in 17,000 people. It is more common among individuals of northern European ancestry.

Very long-chain 3-hydroxyacyl-CoA dehydrogenase (VLCAD) deficiency and long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency involve a defect in the oxidation of fatty acids greater than 10 carbon chains in length. The incidence of LCHAD deficiency is unknown. However, an estimate based on a Finnish population indicates that 1 in 62,000 pregnancies is affected, but the incidence is probably much lower in the United States (Genetics Home Reference, 2009a). VLCAD deficiency is estimated to affect 1 in 40,000–120,000 people (Genetics Home Reference, 2009d).

### Nutrition Management of Fatty Acid Oxidation Disorders

The mainstay for treating mitochondrial β-oxidation defects is avoidance of fasting. By not allowing patients with fatty acid oxidation disorders to become dependent on β-oxidation for energy needs, the accumulation of intermediate metabolites and symptoms can be kept to a minimum (Shils et al., 1999). The acceptable time periods between feedings vary depending on the child's age and size in relation to available glycogen stores; for example, feedings every 4 hours for infants, every 6 hours for children, and every 8 hours for adults (Acosta & Yannicelli, 2001).

In addition to avoidance of fasting, those with SCAD and MCAD deficiency should consume frequent feedings of a carbohydrate-rich diet. A mixture of raw cornstarch to water (1:2 ratio) may be used in children older than 9 months of age at bedtime (Acosta & Yannicelli, 2001). Raw cornstarch is a high-fiber, complex carbohydrate containing high amounts of amylose that is slowly metabolized, thereby aiding to maintain blood glucose levels overnight. A low-fat diet is not required as a result of the fact that in the average diet, less than 5% of daily calorie intake is contributed by medium- or short-chain fatty acids unless medium-chain triglycerides are purposefully given as a supplement (Acosta, 2010).

For very long-chain and long-chain fatty acid defects, medium-chain triglyceride (MCT) supplementation is often prescribed because this form of triglyceride uses an alternate metabolic pathway. MCT supplementation can be achieved with consumption of a nutritional formula high in MCTs or with the addition of MCT oil. Recommended diet composition during infancy is to provide 40–45% of energy from total fat intake (MCT, long-chain fats, and supplements). Depending on the severity of the mutation, long-chain fats should be restricted to 10–20% of fat intake, with the remaining provided by MCT (Rohr & van Calcar, 2008). In children and adults, total fat intake should be restricted to 35% of energy with 10–20% of total energy provided by long-chain fat and the remainder supplied as MCT (Rohr & van Calcar, 2008). An alternative method for determining MCT intake is 2–3 g MCT per kilogram of body weight in infancy and 1–1.5 g/kg MCT for children and adults. However, it is crucial to confirm the diagnosis of very long-chain or long-chain FAOD prior to the initiation of this dietary treatment; giving medium-chain triglycerides to patients with medium- or short-chain defects is harmful.

All FAOD diets should provide at least 3% of total calories from linoleic acid and 1% from alpha-linolenic acid to prevent essential fatty acid deficiency (EFAD). If the patient's diet does not meet recommended EFA intakes, the diet should be supplemented with oils rich in linoleic and alpha-linolenic acid, such as flaxseed, walnut, canola, and corn oils. It is important to include the long-chain fats from these oils when quantifying a patient's daily "allowance" for total long-chain fat intake.

The nutrient requirements for individuals with fatty acid oxidation disorders are similar to those

of normal individuals. Energy intake should be adequate to support age-appropriate growth in infants and children and to maintain normal body mass index (BMI) in adults. Protein intake should provide 10–15% of total calories, with the remaining 30% of total calories provided from fat sources (comprised of long-chain fats, oils to provide EFAs, and medium-chain triglycerides, if warranted based on the type of FAOD). Upon initial diagnosis, energy intakes at 15–20% above the RDA may be prudent to ensure prevention of metabolic decompensation caused by catabolism.

In all individuals, those with and without FAOD, febrile illness and trauma can cause the body to undergo catabolism of body fat and protein stores. During times of febrile illness or trauma, the goal for patients affected by FAOD is to ensure that they have adequate hydration, maintain electrolyte balance, deter catabolism, decrease intake of fatty acids, prevent hypoglycemia, and minimize the accumulation of toxic fatty acyl groups (Acosta & Yannicelli, 2001).

During illness, it is recommended the patient consume extra carbohydrate-containing foods and beverages (e.g., juices, sports drinks, electrolyte replacements, gelatin, and popsicles) to maintain adequate hydration and prevent hypoglycemia. Ingestion of carbohydrate-containing foods at a dose of 5–7 g/kg every 2 to 3 hours is often recommended. Supplemental fats in the usual diet, such as oils to prevent EFA deficiency and medium-chain triglycerides, should be removed from the diet. If the individual is unable to tolerate the ingestion of oral foods and fluids (i.e., there is vomiting or diarrhea), admission to the hospital for dextrose- and electrolyte-containing intravenous fluid management may be needed.

The addition of L-carnitine may aid in preventing an accumulation of fatty acyl groups and may completely reverse symptoms of carnitine transporter deficiency. Supplementation of L-carnitine at a dose of 100 mg/kg/day may be prescribed (Acosta & Yannicelli, 2001; Shils et al., 1999). However, the efficacy of carnitine supplementation in fatty acid oxidation defects, other than primary carnitine transporter deficiency, has been questioned. Riboflavin is an essential cofactor for acetyl-CoA dehydrogenase. Some patients with specific SCAD mutations have demonstrated clinical improvement when given pharmacological doses of oral riboflavin supplementation at doses of 100–200 mg/kg/day (Acosta, 2010; Acosta & Yannicelli, 2001; Shils et al., 1999). Additionally, glycine supplementation may aid in the excretion of excess acetyl-CoAs and other metabolites. Glycine supplementation at a dose of 300 mg/kg/d may be helpful in patients with multiple dehydrogenase deficiency (Acosta & Yannicelli, 2001; Shils et al., 1999). Because docosohexanoic acid (DHA) is often limited in the diet, some metabolic centers may supplement patients with DHA at a dose of 65 mg/day for children weighing less than 20 kg and 130 mg/day for those weighing more than 20 kg (Rohr & van Calcar, 2008).

The outcome of individuals with FAODs, like most metabolic disorders, varies depending on the severity of the defect, timing of diagnosis, and the long-term metabolic control (Acosta & Yannicelli, 2001).

## Disorders of Carbohydrate Metabolism

Disorders of carbohydrate metabolism are inherited conditions caused by a deficiency in any enzyme responsible for the breakdown of carbohydrates. Types of disorders of carbohydrate metabolism include, but are not limited to, the following: glycogen storage diseases, galactosemia, hereditary **fructose intolerance** (see **Figure 5.4**), mucopolysaccharidoses, and pyruvate dehydrogenase (PDH) deficiency. The dietary interventions for galactosemia and PDH deficiency are detailed in this section.

**fructose intolerance**
An inborn error of metabolism caused by the lack of the enzyme fructose-1-phosphate aldolase (aldolase B).

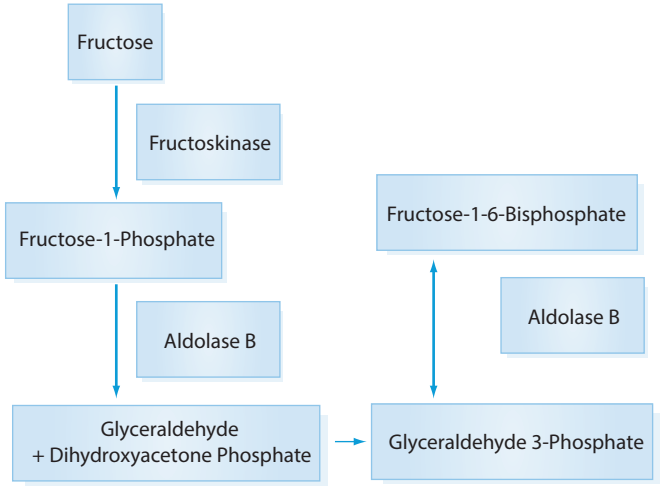



**FIGURE 5.4** Fructose metabolism

## Galactosemia

**galactosemia** An inborn error of carbohydrate metabolism caused by a deficiency of galactosyl-1-phosphate uridyl-transferase, which inhibits galactose conversion to glucose, causing severe medical problems after the ingestion of galactose.

**Galactosemia** is an inherited condition that affects the metabolism of galactose, a hexose that is most commonly bound with glucose to form the milk sugar lactose. It is inherited in an autosomal recessive manner and is caused by a defect in one of three enzymes in the Leloir pathway that converts galactose to glucose primarily in the liver (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009). Galactokinase (GALK) is responsible for converting galactose to galactose-1-phosphate. A deficiency in GALK occurs in 1:50,000–1,000,000 live births (Sarafoglou et al., 2009). Galactose-1-phosphate-uridyltransferase (GALT) converts galactose-1-phosphate to glucose-1-phosphate and is the second step in galactose metabolism. GALT deficiency causes classical galactosemia and has been reported to occur in 1:10,000–30,000 live births (Acosta, 2010). The most common GALT mutations are Duarte-1 and Duarte-2. These mutations are milder and are considered a variant form of classical galactosemia. Many patients with GALT deficiency have a Duarte mutation in combination with another mutation, causing a partial impairment in GALT function, resulting in 14–25% of normal enzyme activity and not requiring dietary intervention (Ficicioglu, Hussa, Yager, & Segal, 2008). Uridine diphosphate-galactose-4-epimerase (GALE) is responsible for the conversion of UDP-galactose to UDP-glucose (Acosta, 2010); a deficiency of this galactosemia-causing enzyme occurs in 1:6,200–1:64,800 live births (Sarafoglou et al., 2009).

Newborns affected with galactosemia are usually diagnosed by state newborn screening using the Beutler fluorescent test. A positive newborn screen result should be followed up with additional testing to confirm or rule out the diagnosis and determine the necessary course of treatment. Confirmatory tests include quantitative analysis of the GALT enzyme and red blood cell levels of galactose and galactose-1-phosphate (Acosta, 2010). In certain cases, a newborn may not be detected by newborn screening or may become ill before the newborn screening results are available. Any patient with symptoms of galactosemia should receive a full work-up to determine whether a type of galactosemia is causing the symptoms. Symptoms vary, depending on whether there is a partial or complete defect, which enzyme is deficient, and biochemical findings. Symptoms of GALT deficiency may be present as early as the second day of life and are caused by the accumulation of galactose-1-phosphate and the abnormal glycosylation of glycoprotein and glycolipids. As noted, without treatment, infants with classical GALT mutations may die, usually from sepsis. It is important to note that symptoms of GALE deficiency depend on the severity of the mutations and can be completely benign. The long-term symptoms of all types of galactosemia are poorly understood and cannot be attributed to the biochemical findings (Sarafoglou et al., 2009).

The goal of nutrition intervention in those with galactosemia is to promote normal nutrition status including normal growth and development in infants and children and a healthy body mass index in adults (Acosta & Yannicelli, 2001). A study by Panis demonstrated normal in utero growth for patients with GALT deficiency but decreased height and weight velocity in girls and a failure in both genders to achieve target midparental height (Sarafoglou et al., 2009). This was not noted in patients with GALE or GALK deficiency. Infants with GALT deficiency may require additional calories, up to 110–120 kcal/kg/day. Calorie needs beyond infancy are similar to the general population and can be estimated using standard predictive equations. Protein requirements in GALT and GALK deficiency may be higher, up to 3.75 g/100 kcal or 15% of calories in infants, because of the more rapid digestion of soy protein isolate compared to casein (Acosta, 2010). Fluid needs in galactosemia are similar to those of unaffected infants.

### Nutrition Management of Galactosemia

A diet free of lactose and low in galactose should be initiated for all patients with confirmed GALT and GALK deficiency as well as for those with severe forms of GALE deficiency (Acosta, 2010; Fridovich-Keil, Bean, He, & Schroer, 2011). The exception is patients with a Duarte mutation in combination with another GALT mutation. These patients have sufficient residual GALT enzyme and in a pilot study were shown not to require dietary intervention (Ficicioglu, Thomas, et al., 2008). The initiation of diet will reverse the symptoms seen in infancy in classical GALT and GALK deficiencies (Acosta, 2010). For infants, breast milk or standard milk-based infant formulas should be replaced with powdered soy infant formula.

Ready-to-feed and liquid concentrate soy formula contain bound galactose as a result of the presence of carrageenan, and thus should be avoided. It is not possible to provide breast milk to a patient with galactosemia because of the lactose present in human milk, which persists because of endogenous galactose production even if the lactating mother eliminates all galactose and lactose from her diet (Gleason, Rasberry, & van Calcar, 2010). Patients with persistently high galactose-1-phosphate levels despite receiving powdered soy formula may benefit from an elemental formula, which is completely free of galactose and has been shown to rapidly decrease galactose-1-phosphate levels (Ficicioglu, Hussa, Yager, & Segal, 2008). Data have not shown that elemental formulas prevent the long-term symptoms of galactosemia. For this reason, and, because of their high cost, elemental formulas are not routinely recommended for all patients with galactosemia.

After 1 year of age, an infant soy or elemental formula is no longer age-appropriate. Toddlers with galactosemia should be transitioned to a pediatric or "step 2" powdered soy formula, which is nutritionally complete and very low in galactose (Gleason et al., 2010). Other options include calcium- and vitamin D–enriched soy milk, rice milk, almond milk, and hemp milk. With the exception of soy milk, these milks are low in protein, so other dietary sources of protein are necessary to support growth. Moreover, rice milk is low in calories as compared to soy milk. In addition to calcium and vitamin D supplements, an age-appropriate multivitamin may be necessary once a patient is transitioned off a soy-based pediatric formula.

Caregivers must be instructed how to read food labels and identify unsafe ingredients as solid foods are introduced. Foods and ingredients that contain galactose must be omitted from the diet of those with galactosemia (Gleason et al., 2010). These contain and are not limited to any milk products, liquid or solid, margarine, chocolate, organ meats, lactoalbumin, hydrolyzed protein, and garbanzo beans. Following a diet restricted in galactose and free of lactose is important in the management of a patient with galactosemia, but the body's endogenous production of galactose far exceeds that consumed in the diet and likely exceeds that which a person with galactosemia can metabolize (Fridovich-Keil & Walter, 2008). This may explain why a child on a carefully controlled diet can have persistently high galactose-1-phosphate level and still experience long-term complications of galactosemia (Berry et al., 1997; Bosch, 2011).

In addition to the diet, long-term management of patients with galactosemia should include routine monitoring of galactose-1-phosphate levels, with a goal of less than 2 mg/dL; however, this may not be possible for patients with classical galactosemia (Sarafoglou et al., 2009). Regular monitoring of calcium and vitamin D intake and bone health status is essential. Patients with galactosemia are at increased risk for low bone density despite intake of calcium, magnesium, zinc, vitamin D, and protein intake above the RDA. Although the mechanisms involved in low bone density are not well understood in this population, dual-energy X-ray absorptiometry (DEXA) can help assess bone density status and guide recommendations for supplementation. Patients should undergo evaluation by a neuropsychologist because of the neurological and cognitive complications associated with galactosemia. Females with galactosemia should be monitored for signs of premature ovarian failure. Labs such as IgF-1, IgFBP2, free T4, and TSH can be evaluated in children with growth failure (Acosta, 2010).

Patients with galactosemia often have issues such as cognitive and neurological deficits and premature ovarian failure despite compliance to diet. There are many possible reasons for these issues, including the following: endogenous galactose production (Berry et al., 1997; Bosch, 2011), over- or underrestriction of galactose (Acosta, 2010), damage before initiation of diet, or damage caused by exposure to galactose in utero (Acosta & Yannicelli, 2001).

### Pyruvate Dehydrogenase Deficiency

Pyruvate dehydrogenase (PDH) deficiency is a disorder of carbohydrate metabolism that is characterized by lactic acidosis and neurological symptoms. It is also known as pyruvate dehydrogenase complex deficiency, or PDHC deficiency (Genetics Home Reference, 2012).

The pyruvate dehydrogenase complex is responsible for converting pyruvate, the end product of glycolysis, into acetyl CoA, which is a major fuel for the Krebs cycle and a substrate for fatty acid synthesis. Pyruvate can also be carboxylated to oxaloacetate via pyruvate carboxylase, transaminated to alanine via aminotransferases, or anaerobically converted to lactate by lactate dehydrogenase. In PDH deficiency, the normal metabolism of carbohydrates is inhibited and the

majority of ATP production is prevented (Acosta & Yannicelli, 2001). The pyruvate dehydrogenase complex (PDC) encompasses multiple copies of several enzymes called E1, E2, and E3. Several catalytic enzymes, E1α, E1β, E2, E3, and protein X, are involved in the enzyme complex. Each of these enzymes plays a part in the chemical reaction that converts pyruvate to acetyl-CoA. The E1 enzyme, also called pyruvate dehydrogenase, comprises four subunits: two E1α subunits and two E1β subunits. Gene mutations in the instructions for making the E1α gene are the most common causes of PDH deficiency (Genetics Home Reference, 2012). Gene mutations that provide instructions for the E1β, E2 enzyme, E3 enzyme, E3 binding protein, and pyruvate dehydrogenase phosphatase result in reduced functioning of one component of the complex, and thereby impair the activity of the whole complex. With decreased function of the complex, the pyruvate builds up and through another chemical reaction is converted to lactic acid, causing mild to severe lactic acidosis and elevated plasma alanine and pyruvate concentrations (Acosta & Yannicelli, 2001; Genetics Home Reference, 2012). The prevalence of pyruvate dehydrogenase deficiency is unknown (Genetics Home Reference, 2012). However, approximately 70% of known cases of PDH deficiency are mutations of the E1α subunit, which is encoded on the X chromosome (Sarafoglou et al., 2009).

When diagnosing PDH deficiency, the most common lab values identified are an elevated serum lactate and pyruvate level, but a normal lactate to pyruvate ratio. The ratio is usually normal because the respiratory chain activity and nicotinamide adenine dinucleotide (NADH) oxidation is maintained. However, during periods of acute illness, the lactate to pyruvate ratio may be elevated (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009). Additionally, serum alanine levels are often elevated in PDH deficiency. Measurement of PDC enzyme activity in cultured skin fibroblasts, cultured lymphocytes, skeletal muscle, and other organs are indicative of PDH deficiency (Sarafoglou et al., 2009).

The clinical presentation of PDH deficiency varies, ranging from fatal congenital lactic acidosis to mild ataxia or neuropathy. The clinical conditions present in two major categories, metabolic and neurologic. Metabolic presentations often occur in the newborn period or early infancy with severe lactic acidosis, usually resulting in death. Neurological conditions for patients surviving infancy may present with developmental delay, hypotonia, seizures, ataxia, and peripheral neuropathy (Sarafoglou et al., 2009).

### Nutrition Management of Pyruvate Dehydrogenase Deficiency

Successful treatment of PDH deficiency remains limited. However, the ketogenic diet has been used with the aim to bypass the lack of PDC activity by replacing blood glucose with sufficient amounts of β-hydroxybutyrate and acetoacetate to serve as alternative energy sources for the nervous system and fatty acids as fuel sources for skeletal muscle and the heart (Sarafoglou et al., 2009). A detailed discussion on the ketogenic diet can be found later in this chapter.

Dichloroacetate (DCA) is an inhibitor of PDH kinase, which maintains the activity of PDH. Supplementation of DCA can reduce the cerebral lactate and cerebrospinal fluid concentrations of lactate and pyruvate. This treatment can be beneficial in eliminating or reducing seizures, infantile spasms, and myoclonic seizures (Acosta & Yannicelli, 2001). However, because DCA is not approved by the Food and Drug Administration (FDA) and is potentially toxic, it should only be used with approved clinical trials (Sarafoglou et al., 2009). High doses (100–3,000 mg/day) of thiamine supplementation have also been recommended to prolong the half-life of any residual PDH. Although some case reports have suggested it may be beneficial, no controlled trials have been done, and its benefit remains unclear (Sarafoglou et al., 2009). If serum carnitine levels are depleted, the patient should also be supplemented with L-carnitine at doses of 25–50 mg/kg/d to normalize plasma free carnitine levels (Acosta & Yannicelli, 2001; Sarafoglou et al., 2009).

An average ketogenic diet provides 10% of calories from carbohydrate, 15% of calories from protein, and at least 75% of calories from fat. Recommended energy intakes should be sufficient to support normal weight gain in infants and children and appropriate weight-for-height in adults (Acosta & Yannicelli, 2001). For patients with intractable seizures, calorie intakes of 75–125% of the RDAs have been used to maintain ketosis. Ketogenic diets are prescribed in ratios of fat to carbohydrate plus protein. For example, one of the more strict ketogenic diets provides a 4:1 ratio, which is 90% of total calories from fat with the remaining 10% composed of carbohydrate and protein. Adequate protein intake is necessary to

prevent failure to thrive, weight loss, low plasma transthyretin concentrations, osteopenia, and hair loss (Acosta & Yannicelli, 2001).

The efficacy of the ketogenic diet for treatment of PDH deficiency is not well established. However, initiation of the diet at an early age may improve neurologic function and delay death (Acosta & Yannicelli, 2001). The recommended length of time to remain on the diet differs among clinicians, but it is often recommended for a duration of 2 years. Those patients that respond positively to the diet remain seizure-free or show continued improvement after cessation of the diet (Acosta & Yannicelli, 2001).

## *Failure to Thrive*

***Claire Blais, RD, CDE, LDN***

*Failure to thrive*, or FTT, is a term used to describe a child whose growth is significantly less than expected, when compared to the child's peers (Bergman & Graham, 2005). There is no one agreed-upon definition or set of anthropometric measures to use for diagnosing failure to thrive (Cole & Lanham, 2011; Olsen, 2006). See **Table 5.1** for common criteria used for making the diagnosis of failure to thrive. One study found that 27% of infants met criteria according to at least one definition (Olsen et al., 2007), and another noted that up to 39% of infants cross two major percentile lines before stabilizing into a growth pattern (Mei, Grummer-Strawn, Thompson, & Dietz, 2004). Therefore, no one single anthropometric measure should be used when making the diagnosis; this further highlights the importance of clinical judgment when working with a child with failure to thrive.

### CRITICAL Thinking

Never let definitions outweigh your clinical judgment. These classifications are meant to help guide your practice, not replace it. If you have a child who was born at low birth weight and is plotting consistently below the third percentile on the weight for age chart, would you classify her with failure to thrive? Some previously accepted definitions would say yes—weight for age below the third percentile classifies as failure to thrive. But use your judgment; perhaps this patient is just following her own growth curve. Someone has to be in the bottom 3%!

### Classification of Failure to Thrive

In 1972, Waterlow established criteria for comparing weight and height of a malnourished child with standards for age- and sex-matched peers. He described a weight for height deficit as wasting, indicating acute malnutrition, and a height for age deficit as stunting, indicating chronic malnutrition. The **Waterlow criteria**, which are similar to those factors listed in Table 5.1 (and found at www.medicalcriteria.com/site/index.php?option=com_content&view=article&id=275%3Amalnutrition&catid=66%3Anutrition&Itemid=80&lang=en), are still widely accepted today as among the primary classifications of pediatric malnutrition.

**Waterlow criteria** A set of calculations used to classify the severity of malnutrition in children.

Several researchers have proposed that the term *failure to thrive* may not be adequately descriptive of faltering growth or pediatric undernutrition (Bergman & Graham, 2005; Olsen, 2006) and that instead clinicians should consider the three factors that lead to energy imbalance. These are inadequate energy intake, inefficient energy utilization, and increased energy expenditure (see **Table 5.2**).

**TABLE 5.1 Waterlow Criteria**

| | | |
|---|---|---|
| Acute malnutrition (wasting) | | |
| $\frac{\text{Actual weight (kg)} \times 100}{\text{Expected weight (kg) for ht (cm) at 50th \%ile}}$ | Grade 0 (normal) | > 90% |
| | Grade I (mild) | 81–90% |
| | Grade II (moderate) | 70–80% |
| | Grade III (severe) | < 70% |
| Chronic malnutrition (stunting) | | |
| $\frac{\text{Actual height (cm)} \times 100}{\text{Expected height (cm) for age at 50th \%ile}}$ | Grade 0 (normal) | > 95% |
| | Grade I (mild) | 90–95% |
| | Grade II (moderate) | 85–89% |
| | Grade III (severe) | < 85% |

Data from Waterlow, C. (1972). Classification and definition of protein-calorie malnutrition. *British Medical Journal*, 3, 566–569.

**TABLE 5.2** Risk Factors for the Development of OFFT and NOFFT

| Risk Factors for OFTT | Risk Factors for NOFTT |
|---|---|
| Bronchopulmonary dysplasia | Poverty |
| Cleft palate | Abuse |
| Cystic fibrosis | Maternal/paternal deficits in appropriate feeding practices |
| Developmental delay | Feeding difficulties |
| Fetal alcohol syndrome | Factors leading to poor bonding |
| HIV/AIDS | Unwanted pregnancy |
| Intrauterine growth restriction | Mother of young age (< 17 years) |
| Prematurity | Depression |
| Low birth weight | Psychopathology (including Munchausen syndrome by proxy) |
| Very low birth weight | |
| Extremely low birth weight | |
| Short gut syndrome | |

Data from Corrales, K. M., & Utter, S. L. (2004). Failure to thrive. In P. Q. Samour, K. K. Helm, & C. E. Lang (Eds.), *Handbook of pediatric nutrition* (2nd ed., pp. 395–412). Burlington, MA: Jones & Bartlett Learning; Kleinman, R.E. (Ed.) (2004). *Pediatric nutrition handbook* (5th ed.). Elk Grove Village, IL: American Academy of Pediatrics; and Feldman, R., Keren, M., Gross-Rozval, O., & Tyano, S. (2004). Mother–Child touch patterns in infant feeding disorders: Relation to maternal, child, and environmental factors. *J Am Acad Child Adolesc Psychiatry, 43*(9), 1089–1097.

There are a variety of causes for the three factors, which are broken up into two classes: organic, or those that can be attributed to an identifiable disease or disorder, and nonorganic, which are considered social, behavioral, or environmental causes. Traditionally, cases of failure to thrive were classified as either organic or nonorganic, but it is increasingly becoming understood that the development of failure to thrive is multifactorial and complex, and most cases present with both organic and nonorganic risk factors (Emond, Drewett, Blair, & Emmett, 2007).

### The Cultural Diversity of Poverty

Poverty is the most omnipresent of the social risk factors for failure to thrive. One study documented that 13% of patients with poor growth are homeless, which makes access to the medical care needed to prevent and correct malnutrition difficult (Frank & Zeisel, 1988). It is crucial in proper treatment of your patients that you consider their socioeconomic status in designing an individualized care plan.

## Assessment of the Patient with Failure to Thrive

Assessment of the patient with failure to thrive should include a thorough medical, social, and nutritional evaluation. The medical evaluation should include a complete history and physical, paying careful attention to pre- and perinatal history, clinical examination, and family history, especially that of older siblings (Gahagan, 2006). The medical evaluation should also assess for lead toxicity, anemia, and zinc deficiency. A low serum alkaline phosphatase may indicate zinc deficiency. During the clinical examination, the physician should observe for signs of neglect, which may include poor general hygiene, poor oral health, or diaper rash (Stephens, Gentry, Michener, Kendall, & Gauer, 2008).

Given that the vast majority of cases of failure to thrive present with some form of inadequate caloric intake, nutritional rehabilitation should be attempted before extensive laboratory workup is commenced. A social evaluation should be conducted to assess the family dynamics and economic situation (Jaffe, 2011). During all evaluations, careful attention should be paid to caregiver–child interaction, including eye contact, physical proximity, and verbalization.

The assessment performed by the nutrition professional is most essential. A thorough assessment of intake and output is necessary to evaluate the child's nutriture truly. If the infant is breastfeeding, the registered dietitian should ask whether the child feeds at both breasts at each feeding, how long the child sucks at each breast, and how many times daily the child feeds. If the infant is formula feeding, careful attention must be paid to formula preparation. A common error in formula feeding is

improper dilution of the formula (Gahagan, 2006). The dietitian should determine how the formula is prepared, how much the child feeds, and how often. It is important here to be specific. Many parents are tempted to say, "Two to 3 ounces every 3 to 4 hours." This could be as little as 12 oz of formula or as much as 24 oz of formula. A detailed 3- to 5-day food record is the most accurate method, but it is not always possible, in which case a 24-hour recall can be very effective (Corrales & Utter, 2005).

For the child who is also taking solid foods, it is important to ask what types and textures are being offered, how long the child takes to eat, what the portion size is and how much the child actually eats of it, and the timing of meals and snacks. The dietitian should also inquire into food allergies or intolerances, including the child's reaction to the food item. If possible, a direct observation of the feeding would be helpful. An in-home evaluation is ideal, but often an office or hospital setting must suffice. In addition to evaluating intake, the dietitian should also garner an understanding of the child's output. Number and consistency of stools and number of wet diapers (or strict measurement of urine output in the acute care setting) can be helpful in determining the child's absorptive capacity. The dietitian should also ask about spits or emesis and, if at all possible, try to quantify the frequency and volume of such spits and emesis. It is important to inquire about the child's activity level as well because fidgety or very active children can be difficult to feed and could also be burning off more calories than they are consuming. It is also the responsibility of the dietitian to plot the growth curves carefully.

The nutrition evaluation should also include an assessment of kilocalorie and protein needs for catch-up growth. The equation for catch-up growth is as follows:

$$\frac{(\text{Ideal Body Weight for Height} \times \text{RDA for Weight Age})}{\text{Current Weight}}$$

where ideal body weight for height is the weight at the 50th percentile for the current height, RDA is the Recommended Dietary Allowance, and the weight age is the age at which the current weight is at the 50th percentile (Corrales & Utter, 2005). This equation can be used for both kilocalorie needs and protein needs. For example, an 8-month-old male who weighs 7 kg and is 68 cm long has a weight age of 4.5 months and an ideal body weight for length of 8 kg. You would then multiply 8 kg × 108 kcal/kg and divide by 7 kg. The result is 123 kcal/kg. If the RDA for weight age is equal to the RDA for current age, then it is generally acceptable to use 120 kcal/kg as the standard reference for this value. For example, a 5-month-old female who weighs 5 kg and measures 59 cm long has a weight age of 2.5 months and an ideal body weight for length of 5.7 kg. The RDA for her weight age is the same as the RDA for her chronologic age, so you multiply 5.7 kg × 120 kcal/kg and divide by 5 kg to get 134 kcal/kg.

Calculation of needs for catch-up growth is always an estimate. It is important to exercise clinical judgment. If a child's catch-up needs calculate out to be substantially more than his or her current level of intake, it is best to make small increases in the level of intake, following weight gain and adjusting as needed. Efforts to promote catch-up should continue until the child regains his or her previously established growth percentiles (Corrales & Utter, 2005). The best measure of whether a child is meeting his or her needs for growth is by following the child's weight gain after interventions. If the child is gaining weight at or above the expected rate for age, the child is receiving adequate kilocalories.

## Nutritional Interventions

Many failure to thrive cases are initially treated in the outpatient setting with behavioral and nutritional interventions. Regardless of the cause of the failure to thrive, the primary intervention is to increase intake of kilocalories. For the breastfed infant, consultation with a lactation specialist may be beneficial to ensure optimal milk production and nursing technique (Gahagan, 2006). In many cases, supplementation with formula or fortified breast milk may be necessary to achieve adequate weight gain and can be discontinued when catch-up growth is achieved. In the formula-fed infant, once it has been determined that the formula is being prepared correctly and adequate volume is being offered to the infant, formula should be concentrated to a higher caloric density. This can be done by concentration up to 24 kcal/oz using powdered infant formula and water, and then by the addition of modular kilocalorie supplements (typically

in the form of fat or glucose polymers) as needed to achieve weight gain, up to 30 kcal/oz. It is not generally recognized as safe to concentrate beyond 24 kcal/oz strictly from powder because the potential renal solute load becomes too high for the infant and vomiting and dehydration could occur (Corrales & Utter, 2005). Increase concentration by 2 kcal/oz every 2–3 days as tolerated until weight velocity is achieved.

For the child who is taking additional solid foods, adding kilocalories to food without adding volume becomes imperative to maximize their intake. Parents should be taught label reading to offer their infants younger than 12 months old high-kilocalorie baby foods. These foods can also be fortified with small amounts of infant cereal, glucose polymer modular supplements, vegetable oil, or margarine. Children older than 1 year of age can be offered drinks with added powdered skim milk, instant breakfast drink mix powder, glucose polymer modular supplements, table sugar, or vegetable oil. Cheese, gravy, butter or margarine, and cream cheese can be added to solid foods, in addition to the supplements listed previously for beverage addition. Juice should be eliminated from the diet (Cole & Lanham, 2011; Corrales & Utter, 2005). Oral nutrition supplementation with high-calorie nutritionally complete drinks may be offered if intake from normal foods alone is insufficient (King & Davis, 2010).

It is important to teach caregivers to develop a standard mealtime routine with their children. Some suggestions for what to tell caregivers are as follows (University of Iowa, 2002):

- Set definitive meal and snack times, and limit their duration to 30 minutes or less.
- Do not allow eating between scheduled meals and snacks, regardless of how much was eaten at the previous feeding.
- Always feed your child at the table in a high chair or a booster seat.
- Praise the positive behaviors.
- Ignore the negative behaviors.
- Allow your child to get messy while eating.
- Model good eating behavior.

If nutritional and behavioral interventions fail to promote weight gain, more intensive therapies such as medications or nutrition support may be considered. Appetite stimulants such as megestrol acetate and cyproheptadine have been shown to be effective in older children with growth failure related to cystic fibrosis and cancer-related cachexia (Couluris et al., 2008; Homnick et al., 2004), but they have not been well studied in other cases of FTT, and the American Academy of Pediatrics and the U.S. Food and Drug Administration (FDA) both believe the costs outweigh the benefits of the use of this appetite stimulant in infants with failure to thrive (Corrales & Utter, 2005).

The multidisciplinary healthcare team should jointly decide when nutrition support should be considered. The amount of supplemental nutrition to be provided generally depends on how much the child can reliably take by mouth because oral nutrition will always be the preference. For infants younger than age 1 year, standard infant formulas can be given. It is best to start with the standard dilution of 20 kcal/oz and advance by 2 kcal/oz daily as tolerated by the infant until the goal concentration is met. The same rules for concentrating infant formula and breast milk as for oral feedings apply for tube feedings as well. For children older than 1 year of age, a standard 30 kcal/oz pediatric formula can be used, provided there is no cow's milk protein allergy present. If the child is allergic, there is a variety of hydrolyzed protein and hypoallergenic formulas available on the market. Full-strength feedings should be initiated at a low rate and advanced slowly as tolerated, monitoring for vomiting, diarrhea, or abdominal distension. If it becomes evident that the child will need aggressive nutrition supplementation for a period of several weeks to months, it would be wise to consider more permanent enteral access, typically in the form of a gastrostomy tube.

The prognosis of children with failure to thrive depends largely on the severity of the illness and the risk factors present. Evidence from studies on the effects on cognitive development in children with a history of failure to thrive in infancy has shown an adverse effect on intellectual outcomes in older childhood, but it is unclear whether this effect is clinically significant (Rudolf & Logan, 2005). A multidisciplinary team (a registered dietitian, a speech-language pathologist, and an occupational therapist, at a minimum) approach to the treatment of the patient with failure to thrive is the most effective method of intervention in these patients. In many cases, prognosis can be excellent if the diagnosis is made early enough and intervention is commenced immediately (Corrales & Utter, 2005).

## Summary

Individualized medical nutrition therapy must be initiated immediately upon diagnosis of an inborn error of metabolism. Nutrition therapy is necessary to prevent further adverse health outcomes and to optimize growth and development. Medical nutrition therapy must be continuously monitored to ensure that the individual needs for each patient are being met and that blood levels of key substances are being monitored. In addition, for some inborn errors of metabolism, consumption of foods may cause physical distress such as gastrointestinal pain, and the individual may be reluctant to eat foods or may develop anorexia. In planning nutrition support, a formal dietary prescription must be carefully written, including all macronutrients and key micronutrients as well as fluid. A plan for monitoring the nutrition support must also be enacted to ensure that both physical and biochemical status are monitored regularly. Last, compliance with the medical nutrition therapy is a very challenging area and must be constantly supported to enable parents and patients to maintain their diet.

Failure to thrive describes children whose growth is significantly below that of their peers. This term has been used for centuries but remains controversial (Olsen, 2006). There exists no one agreed-on definition for failure to thrive but rather several clinically acceptable diagnostic criteria. This chapter touched on some of these diagnostic criteria as well as accepted methods for classifying malnutrition in the pediatric population. It reviewed necessary components of the medical and nutritional evaluation of the patient with failure to thrive and described several techniques found to be effective in treatment of failure to thrive. Because there are so many factors that can contribute to the development of failure to thrive, outcomes are variable. Continued education and heightened awareness of clinicians is necessary to identify and treat failure to thrive in at-risk populations.

## Case Study 1

### Inborn Errors of Metabolism

Laura Harkness, PhD, RD, and Sara Snow, MS, RD

A 5-day-old infant presented to the pediatrician with poor feeding, irritability, lethargy, and vomiting. Upon examination, there was poor tendon reflex.

Laboratory studies were obtained and included urinalysis, complete blood count, blood glucose, and serum electrolytes. Subsequent laboratory tests included blood gases. Key laboratory findings included the following:

| | |
|---|---|
| Blood pH | 7.35 |
| Potassium | 5.5 mM |
| Glucose | 160 mg/dL |
| Ketones | 200 mg/dL |
| Chloride | 94 mM |
| Bicarbonate | 12 mEq/L |
| Urine pH | 4.3 |
| + ketones in urine | |

**Questions**

1. What kind of metabolic imbalance does this infant have?
2. What other laboratory findings would be useful?

## Case Study 2

### Failure to Thrive

Claire Blais, RD, CDE, LDN

MN is a 2-year, 2-month-old boy admitted to the hospital with failure to thrive. As the clinical dietitian, you are consulted to evaluate this patient on his first day of admission. In your chart review, you determine that he was a full-term infant and was strictly breastfed until 6 months old, and then he began to take rice cereal and eventually strained fruits and vegetables were added to his diet. During your interview, MN's mom reports that MN is difficult to feed. He throws food on the floor, plays with it, pushes the spoon away, laughs at his older brothers (ages 4 and 7 years), and generally does not seem interested in eating. It takes him longer than an hour to eat each meal and snack, and it is not a pleasant experience for the patient or his mother.

| | |
|---|---|
| Birth weight | 7 lb, 2 oz |
| Birth length | 20.5 inches |
| 12-month check-up | 19 lb, 6 oz |
| Current weight | 9.86 kg |
| Current length | 90.5 cm |
| Labs | Unremarkable, albumin WNL |
| Meds | None |

24-Hour Recall

Breakfast: 6–8 oz orange juice, ½ cup toasted oat o-shaped cereal, ½ banana, sliced. MN mostly throws the cereal on the floor and mashes the banana into his hair.
Morning snack: 4–6 oz apple juice, 2–3 wheat crackers, 1 string cheese
Lunch: 8 oz whole milk, 1 cup spaghetti with tomato sauce. MN very much enjoys the milk, but most of the spaghetti ends up on his clothes and in the high chair.

Afternoon snack: 4 oz water, ½ cup cooked carrot coins
Dinner: 8 oz whole milk, 1 to 2 chicken nuggets, a few bites of corn
Dessert: 8 oz whole milk, a few bites of chocolate brownie

**Questions**

1. Other than the information provided here, what other questions would you ask MN's mom?
2. What other services do you believe should be consulted on this patient while he is in the hospital?
3. Plot MN's weights on a NCHS growth chart. Describe his weight trend.
4. Estimate MN's calorie and protein needs.
5. What suggestions can you give to his mom?

## Issues to Debate

The assessment and provision of adequate protein and optimal levels of essential amino acids are necessary for normal growth and development in children with inborn errors of metabolism. However, there is scant evidence regarding the amount of protein and amino acids needed to support normal development. In addition, there is little evidence to establish tolerance levels for amino acid levels in individuals with inborn errors of metabolism. Frequently, medical nutrition therapy is based on case studies with little research evidence to support the recommendations of the therapy. In addition, patient compliance with the medical nutrition therapy is problematic.

1. How does the healthcare provider ensure optimal growth and development for patients?
2. How does the healthcare provider write a prescription for medical nutrition therapy when little evidence is available to justify the prescription?
3. How does the healthcare provider work with patients and families to ensure compliance with medical nutrition therapy?

In failure to thrive:

1. When is it appropriate to feed an infant nasogastrically? Moreover, when does it become imperative?
2. At what point should permanent enteral access be considered in an infant or toddler with failure to thrive?

## References


Acosta, P. (2010). *Nutrition management of patients with inherited metabolic disorders*. Sudbury, MA: Jones and Bartlett.

Acosta, P., & Yannicelli, S. (2001). *The Ross Metabolic Formula System: Nutrition support protocols* (4th ed.). Columbus, OH: Abbott Laboratories.

Anastasoaie, V., Kurzius, L., Forbes, P., & Waisbren, S. (2008). Stability of blood phenylalanine levels and IQ in children with phenylketonuria. *Molecular Genetics and Metabolism*,*95*, 17–20.

Arnold, G. L., Koeberl, D. D., Matern, D., Barshop, B., Braverman, N., Burton, B., . . . Longo, N. (2008). A Delphi-based consensus clinical practice protocol for the diagnosis and management of 3-methylcrotonyl CoA carboxylase deficiency. *Molecular Genetics and Metabolism*, *93*, 363–370.

Barschak, A. G., Sitta, A., & Deon, M. (2007). Erythrocyte glutathione peroxidase activity and plasma selenium concentration are reduced in maple syrup urine disease patients during treatment. *International Journal of Developmental Neuroscience*, *25*, 335–338.

Behrman, R. E., Kliegman, R. M., & Jenson, H. B. (2000). *Nelson textbook of pediatrics* (16th ed.). Philadelphia, PA: Saunders.

Belanger-Quntana, A., Burlina, A., Harding, C. O., & Muntau, A. C. (2011). Up to date knowledge on different treatment strategies for phenylketonuria. *Molecular Genetics and Metabolism*, *104*(Suppl.), S19–25.

Bergman, P., & Graham J. (2005). An approach to "failure to thrive." *Australian Family Physician, 34*, 725–729.

Berry, G. T., Nissim, I., Gibson, J. B., Mazur, A. T., Lin, Z., Elsas, L. J., . . . Segal, S. (1997). Quantitative assessment of whole body galactose metabolism in galactosemia patients. *European Journal of Pediatrics*, *156*(Suppl. 1), S43–S49.

Bosch, A. M. (2011). Classical galactosemia: Dietary dilemmas. *Journal of Inherited Metabolic Disease*, *34*, 257–260.

Burton, B. K., Adams, D. J., Grange, D. K., Malone, J. I., Jurecki, E., Bausell, H., . . . Swan, K. T. (2011). Tetrahydrobiopterin therapy for phenylketonuria in infants and young children. *Journal of Pediatrics*, *158*(3), 410–415.

Cakir, B., Teksam, M., Kosehan, D., Akin, K., & Koktener, A. (2011). Inborn errors of metabolism presenting in childhood. *Journal of Neuroimaging*, *21*(2), e117–e133.

Castorina, M., Rigante, D., Antuzzi, D., Cannizzaro, G. S., & Ricci, R. (2008). Different outcome in isovaleric acidemia might be related to unsatisfactory diet compliance. *Scandinavian Journal of Gastroenterology*, *43*, 767–768.

Chuang, D. T., & Shih, V. E. (2001). Maple syrup urine disease (branched-chain ketoaciduria). *Metabolic and Molecular Bases of Inherited Disease*, *8*, 1971–2005.

Cole, S. Z., & Lanham, J. S. (2011). Failure to thrive: An update. *American Family Physician, 83*, 829–834.

Corrales, K. M., & Utter, S. L. (2005). Growth failure. In P. Q. Samour, K. K. Helm, & C. E. Lang (Eds.), *Handbook of pediatric nutrition* (3rd ed., pp. 391–406). Sudbury, MA: Jones and Bartlett.

Couce, M. L., Castiñeiras, D. E., Bóveda, M. D., Baña, A., Cocho, J. A., Iglesias, A. J., . . . Fraga, J. M. (2011). Evaluation

and long-term follow-up of infants with inborn errors of metabolism identified in an expanded screening programme. *Molecular Genetics and Metabolism*, *104*, 470–475.

Coude, F. X., Ogier, H., Grimber, G., Parvy, P. H., Charpentier, C., & Saudubray, J. M. (1982). Correlations between blood ammonia concentrations and organic acid accumulation in isovaleric and propionic academia. *Pediatrics*, *69*, 115–117.

Delis, D., Michelakakis, H., Katsarou, E., & Bartsocas, C. S. (2001). Thiamin-responsive maple syrup urine disease: Seizures after 7 years of satisfactory control. *Journal of Inherited Metabolic Disease*, *24*, 683–684.

Couluris, M., Mayer, J. L., Freyer, D. R., Sandler, E., Xu, P., & Krischer, J. P. (2008). Effect of cyproheptadine hydrochloride (periactin) and megestrol acetate (megace) on weight in children with cancer/treatment-related cachexia. *Journal of Pediatric Hematology/Oncology, 30*, 791–797.

*Dietitian's guide: Phenylade PheBloc LNAA phenylalanine free relaxed diet therapy*. (2010). Applied Nutrition Corporation. Retrieved from www.industrycortex.com/datasheets/profile/1503756083/lnaa-lnaa-phenylalanine-free-relaxed-diet-therapy-phenylalanine-f

Elsas, L. J., & Acosta, P. B. (1999). Nutrition support of inherited metabolic disorders. *Modern Nutrition in Health and Disease, 9*, 1003–1056.

Emond, A., Drewett, R., Blair, P., & Emmett, P. (2007). Postnatal factors associated with failure to thrive in term infants in the Avon Longitudinal Study of Parents and Children. *Archives of Disease in Childhood, 92*, 115–119.

Ficicioglu, C., Hussa, C., Yager, C., & Segal, S. (2008). Effect of galactose free formula on galactose-1-phosphate in two infants with classical galactosemia. *European Journal of Pediatrics, 167*, *595–596*.

Ficicioglu, C., Thomas, N., Yager, C., Gallagher, P. R., Hussa, C., Mattie, A., . . . Forbes, B. J. (2008). Duarte (DG) galactosemia: A pilot study of biochemical and neurodevelopmental assessment in children detected by newborn screening. *Molecular Genetics and Metabolism, 95*, 206–212.

Fomon, S. J., Haschke, F., Zeigler, E. E., & Nelson, S. E.(1982). Body composition of reference children from birth to age 10 years. *American Journal of Clinical Nutrition, 35*, 1169–1175.

Frank, D. A., & Zeisel, S. H. (1988). Failure to thrive. *Pediatric Clinics of North America, 35*(6), 1187–1206.

Fridovich-Keil, J., Bean, L., He, M., & Schroer, R. (2011). Epimerase deficiency galactosemia. *Gene Reviews*. Retrieved from www.ncbi.nlm.nih.gov/books/NBK51671/

Fridovich-Keil, J. L., & Walter, J. H. (2008). Chapter 72: Galactosemia. Online Metabolic and Molecular Basis of Inherited Disease. Retrieved from www.ommbid.com/OMMBID/a/c.html/carbohydrates/galactosemia

Gahagan, S. (2006). Failure to thrive: A consequence of undernutrition. *Pediatrics in Review, 27*, e1–e11.

Genetics Home Reference. (2009a). Long-chain 3-hydroxyacyl-CoA dehydrogenase deficiency. Retrieved from http://ghr.nlm.nih.gov/condition/long-chain-3-hydroxyacyl-coa-dehydrogenase-deficiency

Genetics Home Reference. (2009b). Medium-chain acyl-CoA dehydrogenase deficiency. Retrieved from http://ghr.nlm.nih.gov/condition/medium-chain-acyl-coa-dehydrogenase-deficiency

Genetics Home Reference. (2009c). Short-chain acyl-CoA dehydrogenase deficiency. Retrieved from http://ghr.nlm.nih.gov/condition/short-chain-acyl-coa-dehydrogenase-deficiency

Genetics Home Reference. (2009d). Very long-chain acyl-CoA dehydrogenase deficiency. Retrieved from http://ghr.nlm.nih.gov/condition/very-long-chain-acyl-coa-dehydrogenase-deficiency

Genetics Home Reference. (2012). Pyruvate dehydrogenase deficiency. Retrieved from http://ghr.nlm.nih.gov/condition/pyruvate-dehydrogenase-deficiency

Genetics Home Reference. (2013a). Inborn errors of metabolism. Retrieved from http://ghr.nlm.nih.gov/glossary=inbornerrorsofmetabolism

Genetics Home Reference. (2013b). What are the types of genetic tests? Retrieved from http://ghr.nlm.nih.gov/handbook/testing/uses

Gleason, L., Rasberry, M., & van Calcar, S. (2010). *Understanding galactosemia: A diet guide*. Columbus, OH: Abbott Nutrition.

Gropper, S. S., Naglak, M. C., Nardella, M., Plyler, A., Rarback, S., & Yannicelli, S. (1993). Nutrient intakes of adolescents with phenylketonuria and infants and children with maple syrup urine disease on semisynthetic diets. *Journal of the American College of Nutrition*, *12*, 108–114.

Guthrie, R. (1961). Blood screening for phenylketonuria. *Journal of the American Medical Association*, *178*, 863.

Guthrie, R. (1996). The introduction of newborn screening for phenylketonuria: A personal history. *European Journal of Pediatrics*, *155*, S4–*5*.

Hansen, K., & Horslen, S. (2008). Metabolic liver disease in children. *Liver Transplantation, 14*, 713–733.

Henstenburg, J. D., Mazur, A. T., Kaplan, P. B., & Stallings, V. A. (1990). Nutritional assessment and body composition in children with maple syrup urine disease. *Journal of the American Dietetic Association*, *90*, 32A.

Holmes Morton, D., Strauss, K. A., Robinson, D. L., Puffenberger, E. G., & Kelley, R. I. (2002). Diagnosis and treatment of maple syrup urine disease: A study of 36 patients. *Pediatrics*, *109*, 999–1008.

Homnick, D. N., Homnick, B. D., Reeves, A. J., Marks, J. H., Pimentel, R. S., & Bonnema, S. K. (2004). Cyproheptadine is an effective appetite stimulant in cystic fibrosis. *Pediatric Pulmonology, 38*, 129–134.

Jaffe, A.C. (2011). Failure to thrive: Current clinical concepts. *Pediatrics in Review, 32*, 100–107.

Kaplan, P., Ficicioglu, C., Mazur, A. T., Palmieri, M. J., & Berry, G. T. (2006). Liver transplantation is not curative for methylmalonic acidopathy caused by methylmalonyl-CoA mutase deficiency. *Molecular Genetics and Metabolism, 88*(4), 322–326.

King, C., & Davis, T. (2010). Nutritional treatment of infants and children with faltering growth. *European Journal of Clinical Nutrition, 64*, S11–13.

Lanpher, B. C., Gropman, A., Chapman, K. A., Lichter-Konecki, U., & Summar, M. L. (2011). *Urea cycle disorders review*. Seattle, WA: Urea Cycle Disorders Consortium.

Leonard, J. V. (2001). The nutritional management of urea cycle disorders. *Journal of Pediatrics*, *138*(1), S40–S45.

Loots, D. T., Mienie, L. J., & Erasmus, E. (2007). Amino-acid depletion induced by abnormal amino-acid conjugation

and protein restriction in isovaleric academia. *European Journal of Clinical Nutrition*, *61*, 1323–1327.

Loukas, Y. L., Soumelas, G. S., Dotsikas, Y., Georgiou, V., Molou, E., Thodi, G., . . . Papadoopoulos, K. (2010). Expanded newborn screening in Greece: 30 months of experience. *Journal of Inherited Metabolic Disease*, *33*(Suppl. 3), 341–348. doi:10.1007/s10545-010-9181-8

Matalon, R., Koch, R., Michals-Matalon, K., Moseley, K., Surendran, S., Tyring, S., . . . Guttler, F. (2004). Biopterin responsive phenylalanine hydroxylase deficiency. *Genetics in Medicine*, *6*(1), 27–32.

Mei, Z., Grummer-Strawn, L. M., Thompson, D., & Dietz, W. H. (2004). Shifts in percentiles of growth during early childhood: Analysis of longitudinal data from the California Child Health and Development Study. *Pediatrics, 113*, e717–e727.

Millington, D. S., Kodo, N., Norwood, D. L., & Roe, C. R. (1990). Tandem mass spectrometry: A new method for acylcarnitine profiling with potential for neonatal screening for inborn errors of metabolism. *Journal of Inherited Metabolic Disease*, *13*, 321–324.

Morioka, D., Kasahara, M., Takada, Y., Shirouzu, Y., Taira, K., Sakamoto, S., . . . Tanaka, K. (2005). Current role of liver transplantation for treatment of urea cycle disorders: A review of worldwide English literature and 13 cases at Kyoto University. *Liver Transplantation*, *11*, 1332–1342.

National Institutes of Health Consensus Development Panel, National Institutes of Health Consensus Development Conference Statement. (2001). Phenylketonuria (PKU): Screening and management, October 16–18, 2000. *Pediatrics*, *108*, 972–982.

National Newborn Screening and Global Resource Center. (2012). History and overview of newborn screening. Retrieved from http://genes-r-us.uthscsa.edu/resources/newborn/overview.htm

Olsen, E. M. (2006). Failure to thrive: Still a problem of definition. *Clinical Pediatrics, 45*, 1–6.

Olsen, E. M., Petersen, J., Skovgaard, A. M., Weile, B., Jørgensen, T., & Wright, C. M. (2007). Failure to thrive: The prevalence and concurrence of anthropometric criteria in a general infant population. *Archives of Disease in Childhood, 92*, 109–114.

Polinati, P. P., Eskelin, P., & Tyni, T. (2009). Fatty acid oxidation disorders. *eLS*. doi:10.1002/9780470015902.a0003067.pub2

Raghuveer, T. S., Garg, U., & Graff, W. D. (2006). Inborn errors of metabolism in infancy and early childhood: An update. *American Family Physician*, *73*(11), 1981–1990.

Rohr, F., & van Calcar, S. (Eds.). (2008).*Very long chain acyl CoA dehydrogenase deficiency (VLCADD)*. OMIM #201475. Retrieved from www.gmdi.org/index.php?option=com_content&view=article&id=43

Ross, L. F. (2010). An ethical and policy analysis of elective transplantation for metabolic conditions diagnosed by newborn screening. *Journal of Pediatrics*, *56*(1), 139–144.

Rudolf, M. C., & Logan, S. (2005). What is the long term outcome for children who fail to thrive? A systematic review. *Archives of Disease in Childhood, 90*, 925–931.

Sarafoglou, K., Hoffman, G. F., & Roth, K. S. (2009). *Pediatric endocrinology and inborn errors of metabolism*. New York, NY: McGraw-Hill.

Scaini, G., Jeremias, I. C., Morais, M. O. S., Borges, G. D., Munhoz, B. P., Leffa, D. D., . . . Streck, E. L. (2012). DNA damage in an animal model of maple syrup urine disease. *Molecular Genetics and Metabolism*, *106*, 169–174.

Schulze, A., Mayatepek, E., & Hoffmann, G. F. (2002). Evaluation of 6-year application of the enzymatic colorimetric phenylalanine assay in the setting of neonatal screening for phenylketonuria. *International Journal of Clinical Chemistry, 317*(1–2), 27–37.

Scriver, C. R., Waters, P. J., & Sarkissian, C. (2000). PAHdb: A locus-specific knowledgebase. *Human Mutation*, *15*(1), 99–104.

Shellmar, D. A., DeVito Dabbs, A., Dew, M. A., Noll, R. B., Feldman, H., Strauss, K. A., . . . Mazareigos, G. V. (2011). Cognitive and adaptive functioning after liver transplantation for maple syrup urine disease: A case series. *Pediatric Transplantation*, *15*, 58–64.

Shils, M., Olson, J. A., Shike, M., & Ross, A. C. (Eds.). (1999). *Modern nutrition in health and disease* (9th ed.). Baltimore: Lippincott Williams & Wilkins.

Singh, R. H., & Quirk, M. E. (2011). Using change in plasma phenylalanine concentrations and ability to liberalize diet to classify responsiveness to tetrahydrobiopterin therapy in patients. *Molecular Genetics and Metabolism*, *104*(4), 485–491.

Solis, J. O., & Singh, R. H. (2002). Management of fatty acid oxidation disorders: A survey of current treatment strategies. *Journal of the American Dietetic Association*, *102*(12), 1800–1803.

Staddler, S. C., Polanetz, R., & Maier, E. M. (2006). Newborn screening for 3-methylcrotonyl-CoA carboxylase deficiency: Population heterogeneity of MCCA and MCCB mutations and impact on risk assessment. *Human Mutation*, *27*, 748–759.

Stephens, M. B., Gentry, B. C., Michener, M. D., Kendall, S. K., & Gauer, R. (2008). What is the clinical work up for failure to thrive? *Journal of Family Practice, 57*, 264–266.

Strauss, K. A., Mazareigos, G. V., Sindhi, R., Squires, R., Finegold, D. N., Vockley, G., . . . Morton Holmes, D. (2006). Elective liver transplantation for the treatment of classical maple syrup urine disease. *American Journal of Transplantation*, *6*, 557–564.

Strauss, K. A., & Morton, D. H. (2003). Branched-chain keto-acid dehydrogenase deficiency: Maple syrup urine disease. *Current Treatment Options in Neurology*, *5*, 329–341.

University of Iowa. (2002). "Mealtime routine" suggestions. Adapted from Intensive Course in Pediatric Nutrition, University of Iowa, Iowa City, May 7–10.

Urea Cycle Disorders Conference Group. (2001). Consensus statement from a Conference for the Management of Patients with Urea Cycle Disorders. *Journal of Pediatrics*, *138*(1), S1–S5.

Van Calcar, S. C., MacLeod, E. L., Gleason, S. T., Etzel, M. R., Clayton, M. K., Wolff, J. A., & New, D. M. (2009). Improved nutritional management of phenylketonuria by using a diet containing glycomacropeptide compared with amino acids. *American Journal of Clinical Nutrition*, *89*(4), 1068–1077.

Walsh, K. S., & Scott, M. N. (2010). Neurocognitive profile in a case of maple syrup urine disease. *Clinical Neuropsychologist*, *24*, 689–700.

Waterlow, C. (1972). Classification and definition of protein-calorie malnutrition. *British Medical Journal, 3*, 577–579.

Watson, M. S., Lloyd-Puryear, M. A., Mann, M. Y., Rinaldo, P., & Howell, R. R. (Eds.). (2006). Newborn screening: Toward a uniform screening panel and system. *Genetics in Medicine*, *8*(5 Suppl.), 12S–252S. Retrieved from www.acmg.net/resources/policies/NBS/NBS-sections.htm

Zhou, M., Lu, G., Gao, C., Wang, Y., & Sun, H. (2012). Tissue-specific and nutrient regulation of the branched-chain alpha-keto acid dehydrogenase phosphatase, protein phosphatase 2Cm (PP2Cm). *Journal of Biological Chemistry*, *287*(28), 23397–23406.

# Special Section on Neonatal Intensive Care Nutrition: Prematurity and Complications

*Liesje Nieman, RD, CNSD, LDN*



## CHAPTER OUTLINE



## Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Define prematurity and discuss the impact of prematurity on growth and nutrition.
2. Determine appropriate goals for growth for infants in the neonatal intensive care unit.
3. Identify enteral formulas that are inappropriate for infants born prematurely.
4. Explain the rationale for the use of postdischarge premature infant formulas.
5. Discuss the importance of evaluating developmental readiness, as opposed to just chronologic or corrected age, when introducing complementary foods.

6. Describe the impact of feeding disorders and reflux on nutrient intake.
7. Identify appropriate uses of parenteral nutrition in infants.
8. Discuss the limitations and risks of parenteral nutrition.
9. Explain under what circumstances a multivitamin supplement is appropriate when an infant is on enteral feedings.

Because of advancements in medical treatments and technology, survival of extremely premature infants (i.e., < 26 weeks gestation) is improving (Chan, Ohlsson, Synnes, Chein, & Lee, 2001). This state of extreme **prematurity** brings about a host of unique nutrition-related issues. The importance of the nutritionist's role in the neonatal intensive care unit (NICU) has been brought to the forefront in recent years. Many find it quite rewarding to work with premature infants because of the clinician's ability to significantly affect outcomes, such as growth velocity, healing, and transition from parenteral to enteral nutrition support. Registered dietitian involvement in the NICU has been associated with improved nutritional intake and infant growth, shortened hospital length of stay, and reduced related costs (Olsen, Richardson, Schmid, Ausman, & Dwyer, 2005).

**prematurity** Less than 37 weeks gestation at birth.

## Nutrition Assessment

Premature infants are born at less than 37 weeks gestation. Infants are further classified by their birth weight as follows: low birth weight, less than 2,500 g; **very low birth weight (VLBW)**, less than 1,500 g; **extremely low birth weight**, less than 1,000 g; and "micropremies," less than 750 to 800 g. To evaluate in utero growth, an infant's birth anthropometrics are plotted on the Lubchenco intrauterine growth chart. An infant is appropriate for gestational age if his or her weight plots between the 10th and 90th percentiles, **small for gestational age (SGA)** if below the 10th percentile, and large for gestational age if above the 90th percentile.

**very low birth weight (VLBW)** Birth weight of less than 1,500 g.

**extremely low birth weight** Birth weight of less than 1,000 g.

**small for gestational age (SGA)** Birth weight plots below the 10th percentile.

Symmetric growth restriction is defined as weight, length, and head circumference of an infant plotting below the 10th percentile; infants with this type of growth restriction are more prone to poor outcomes for later growth and development. An infant whose weight and length are SGA but whose head circumference is above the 10th percentile has asymmetric growth restriction (e.g., head spared). **Intrauterine growth restriction** often is used interchangeably with SGA, but these two terms have different denotations. An infant with intrauterine growth restriction has not achieved his or her full in utero growth potential, whereas an infant defined as SGA born to parents who are both less than the 5th percentile for height may have achieved his or her full genetic potential in utero and is therefore not defined as intrauterine growth restricted (Griffin, 2002). If an infant requires catch-up growth, generally the head circumference catches up first, followed by weight and then length. Catch-up growth in head circumference generally occurs in the first 3 to 8 months of life (Bernstein, Heimler, & Sasidharan, 1998).

**intrauterine growth restriction** When an infant has not achieved his or her full in utero growth potential.

There are several growth chart options for plotting premature infants. Using corrected age, infants may initially be plotted either on the Oregon growth record for infants (i.e., Babson Benda intrauterine and postnatal growth chart) or the Fenton growth chart. When plotting infants and children on growth charts, corrected age is generally used on average until 24 months, with a minimum of 18 months and maximum of 36 months.

### Calculating Corrected or Adjusted Age

Calculating an infant's corrected age "adjusts" for the infant's prematurity. It may also be referred to as postconceptional or postmenstrual age. The calculation is as follows (Bernbaum, 2000): chronological age – weeks or months of prematurity = corrected age

For example, an infant born at 28 weeks gestational age is 3 months (12 weeks) premature (based on a term pregnancy of 40 weeks). If the infant's actual age is 6 months, the corrected age is 3 months. For example: 6 months – 3 months = 3 months

The Babson Benda chart represents cross-sectional data from a small group of newborns from 26 to 40 weeks gestation along with longitudinal data from more than 4,000 term infants through the first year of life. Male and female data were combined (Babson & Benda, 1976). Its limits include the small sample size (which provides low confidence in the extremes of the data), the 26 weeks start, and the 500-g graph increments. Two standard deviations above the mean roughly corresponds with 97.5 percentile, two standard deviations below the mean corresponds with 2.5 percentile, 1 standard deviation above the mean corresponds with 84 percentile, and 1 standard deviation below the mean corresponds with 16 percentile (Groh-Wargo & Cox, 1997).

In creating the Fenton growth chart, the literature was searched from 1980 to 2002 for more recent data to complete the pre- and postterm sections of the chart. Data were selected from population studies with large sample sizes. Comparisons were made between the new chart and the Babson Benda graph. To validate the growth chart, the growth results from the National Institute of Child Health and Human Development Neonatal Research Network were superimposed on the new chart. The updated growth chart allows a comparison of an infant's growth as early as 22 weeks gestation, up to the term infant at 50 weeks postconceptional age (i.e., 10 weeks term). Comparison of the size of infants based on the National Institute of Child Health and Human Development data at a weight of 2 kg provides evidence that on average preterm infants are growth retarded with respect to weight and length, whereas their head size has caught up to birth percentiles. Other advantages of this chart include the 100-g graph increments and the percentile curves (3rd, 10th, 50th, 90th, and 97th) rather than standard deviation. As with all meta-analyses, the validity of this growth chart is limited by the heterogeneity of the data sources. Further validation is needed to illustrate the growth patterns of preterm infants to older ages (Fenton, 2003).

The Infant Health and Development Program charts (i.e., Casey charts) include separate charts for VLBW and low-birth-weight infants born prematurely and separate charts within each weight category for boys and girls. This growth chart starts at "–2 months of age." The benefit of using these charts over others is that growth is compared with other premature infants. However, these charts do not delineate "normal" or "abnormal" growth, and the data were collected before the availability of enriched formulas. Different from the NCHS growth charts, nutrition interventions should be initiated when measurements fall below the 50th percentile on the Infant Health and Development Program charts or when growth percentiles change rapidly (Groh-Wargo & Cox, 1997).

### Growth Goals

Although most preterm infants demonstrate adequate growth before discharge, very few experience complete catch-up. At discharge, most premature infants are smaller than the fetus or newborn infant of comparable postconceptional age (Agosti, Vegni, Calciolari, Marini, & the Investigators of the "GAMMA" Study Group, 2003). During 38 to 48 weeks postconceptional age, prematurely born infants may show rates of weight gain similar to the rapid weight gain that normally occurs during 32 to 36 weeks gestation in utero (Groh-Wargo & Cox, 1997). This period of catch-up growth often correlates with optimal caloric intake and the absence of medical compromise. Until 1,800 g or 37 weeks gestation, expected growth velocity is 15 to 20 g/kg of current weight per day (Steward & Pridham, 2002; Ziegler, Thureen, & Carlson, 2002). Beyond 37 weeks gestation, growth velocity of premature infants is generally compared with the expected velocity of healthy reference standards. Catch-up growth may be 10% to 50% above expected. An infant should be referred for further nutrition evaluation if he or she is not gaining at least 20 g/day from term to 3 months corrected age, 15 g/day from 3 to 6 months corrected age, 10 g/day from 6 to 9 months corrected age, and 6 g/day from 9 to 12 months corrected age (Gairdner & Pearson, 1988; Giuo, Roche, & Moore, 1988; Roche & Himes, 1982). An infant should be referred to a dietitian if head circumference growth is less than 0.5 cm/week from term to 3 months, less than 0.25 cm/week from 3 to 6 months, and greater than 1.25 cm/week at any time during infancy (Groh-Wargo & Cox, 1997).

### Estimating Nutrient Needs

Infants have a higher metabolic rate and energy requirement per unit of body weight than children and adults (Pierro, 2002). Energy requirements for infants are broken down as follows: 40 to 70 kcal/kg/day for maintenance metabolism, 50 to 70 kcal/kg/day for growth (tissue synthesis and energy stored), and up to 20 kcal/kg/day to cover losses via excrement (Pierro, 2002). Caloric needs for a newborn infant fed enterally are approximately 100 to 120 kcal/kg/day, whereas those who receive parenteral nutrition (PN) require fewer calories (i.e., 80 to 100 kcal/kg/day) because energy is not needed to cover losses in excrement nor is energy being used for the thermogenic effect of food.

The caloric needs of premature infants are even higher than the caloric needs of full-term newborn infants. Current research suggests that VLBW neonates require a daily minimum of 60 kcal/kg/day, including 2.5 g/kg/day of amino acids to prevent catabolism, and 80 to 90 kcal/kg/day, including 2.7 to 3.5 g/kg/day of amino acids to maintain growth rates similar to those observed in utero (Ibrahim, Jeroudi, Baier, Dhanireddy, & Krouskop, 2004). In contrast, other research demonstrated that preterm newborns are minimally anabolic with parenteral intakes of 2 g/kg/day of amino acids and 50 to 60 kcal/kg/day (Thureen & Hay, 2001). Enterally fed premature infants require approximately 110 to 130 kcal/kg/day and 90 to 110 kcal/kg/day via PN. Some medical circumstances may require even higher caloric intakes to support adequate growth (e.g., bronchopulmonary dysplasia, congenital heart disease).

## Enteral Feeding and Prematurity

Breast milk produced by mothers who have given birth prematurely contains greater concentrations of the following as compared with term breast milk: immune proteins, total lipid, medium-chain fatty acids, vitamins, some minerals (e.g., calcium, sodium), and trace elements (Rao & Georgieff, 2005). Breast milk is classified as preterm only for the 2 to 4 weeks after birth, regardless of how prematurely the infant was born. Studies have shown that **necrotizing enterocolitis (NEC)** occurs less frequently when human milk is administered as compared with formula, which is especially pertinent to the preterm infant population (Lucas & Cole, 1990).

**necrotizing enterocolitis (NEC)** An inflammation or death of the intestinal tract.

Some mothers are unwilling or unable to provide breast milk for their infant. Although pasteurized donor breast milk has not been studied as extensively as mothers' own milk, pasteurized donor human milk can provide many of the components and benefits of human milk while eliminating the risk of transmission of infectious agents. "Pasteurization does affect some of the nutritional and immunologic components of human milk, but many immunoglobulins, enzymes, hormones, and growth factors are unchanged or minimally decreased" (Wight, 2001). In addition, gastric emptying has been shown to be faster and feed tolerance better with human milk (Yu, 2005).

However, the exclusive feeding of unfortified breast milk in premature infants has been associated with poor growth velocity and nutritional deficits (Kashyap, Schulze, & Forsyth, 1990). It has been observed that formula-fed infants have a higher rate of growth than do breastfed infants. Premature human milk provides insufficient protein, sodium, calcium, and phosphate to meet the needs of the premature infant (Schanler, 2001). There are commercially available multicomponent human milk fortifiers that provide additional calories, protein, calcium, phosphate, and vitamins. A meta-analysis study (Kuschel & Harding, 2004) concluded the following:

- Infants receiving fortified human milk demonstrated greater weight gains, greater length gains, and greater head growth in the short term.
- There is no significantly increased risk of NEC in infants receiving fortified human milk.
- Mean serum alkaline phosphatase and bone mineral content values were not statistically different between control and treatment groups.
- No long-term advantage was shown in terms of either growth or neurodevelopmental outcomes.

Fortification of breast milk may be initiated when feeds are tolerated at 100 to 150 mL/kg/day, and a minimum intake of 150 mL/kg/day of preterm milk fortified to 24 calories/oz (i.e., 120 kcal/kg/day) is required to achieve optimal nutrition.

When breast milk is not available, premature formulas are generally indicated for infants

who weigh less than 1,850 to 2,000 g (American Academy of Pediatrics and American College of Obstetrics and Gynecology, 2002; Bernstein et al., 1998). Infants who require higher intakes of calcium and phosphorus (e.g., those with osteopenia) may continue to receive these formulas until they weigh 3,500 g (Carver, Wu, & Hall, 2001). The protein in premature infant formulas is whey predominant, which may result in greater cystine intake and retention as well as greater taurine stores. In addition, the use of whey-predominant formula may decrease the potential for development of lactobezoars in premature infants. Premature formulas contain greater amounts of protein than standard term infant formulas, and the fat content is a combination of vegetable oils, providing both long-chain and medium-chain triglycerides. The high medium-chain triglyceride content may enhance calcium absorption; promote nitrogen retention, lipogenesis, and weight gain; and improve fat absorption. Mineral content (e.g., calcium, phosphorus, magnesium) of premature formulas is higher than term infant formulas to support optimal bone mineralization (Groh-Wargo, Thompson, & Cox, 2000).

It is the responsibility of the registered dietitian and the neonatologist to strictly monitor the use of these formulas and to transition to a postdischarge premature formula when medically appropriate. Intake of premature infant formula exceeding 500 mL/day will likely provide excessive amounts of vitamin A and vitamin D (Lucas, King, Bishop, & King, 1992; Nako, Fukushima, & Tomomasa, 1993). Examples of premature formulas include Enfamil Premature Lipil (Mead Johnson Nutritionals) and Similac Special Care Advance (Ross Products Division, Abbott Laboratories).

Premature infants should receive multivitamin supplementation if the daily volume intake of premature formulas is inadequate (i.e., < 500 mL). Premature infants receiving postdischarge premature formulas (i.e., Enfacare Lipil, Neosure Advance) at intakes of less than 750 mL/day require multivitamin supplementation. There is no indication to provide multivitamin supplementation to full-term infants receiving term infant formulas (e.g., Enfamil Lipil, Similac Advance) to meet their caloric needs (i.e., 108 kcal/kg/day). Premature infants receiving full **enteral nutrition** via exclusive breast milk fortified with human milk fortifier do not require multivitamin supplementation, but supplementation with iron sulfate should be considered. Multivitamin supplementation is indicated for exclusively breastfed premature infants to meet the recommended daily vitamin D intake of 400 IU.

**enteral nutrition** The delivery of nutrients into the digestive system.

Premature infants are prone to anemia because of their inadequate iron stores and iatrogenic blood losses. All premature infants should receive iron supplementation by 1 month of age at doses of 2 mg/kg/day for breastfed infants and at doses of 1 mg/kg/day for those receiving preterm or postdischarge formulas. Iron may be administered to premature infants who are tolerating enteral feeds (i.e., > 100 mL/kg/day) as early as 2 weeks of age. This early supplementation can reduce the incidence of iron deficiency and the need for blood transfusions, as compared with the late administration of iron starting at 2 months of age (Franz, Mihatsch, Sander, Kron, & Pohlandt, 2000).

Infants may not tolerate large volumes of breast milk or formula for a variety of reasons, such as disease of the pulmonary, cardiac, hepatic, renal, or GI systems. If adequate volumes cannot be provided by mouth, supplemental tube feedings are recommended. If fluid needs are met (i.e., > 130 to 150 mL/kg/day for infants without medical conditions requiring fluid restriction) and additional caloric intake is needed, increasing the concentration of the breast milk or formula is recommended (Wheeler & Hall, 1996). Care should be taken to avoid administering a potentially excessive renal solute load (i.e., > 30 to 35 mOsm/100 kcal). Often, formulas concentrated to 24 to 27 kcal/oz are tolerated, and infants with mature renal systems have been noted to tolerate formulas concentrated to 30 kcal/oz. If vitamin, mineral, and protein needs are met with the feeding volume and growth remains inadequate, modular products are often added. Modular products may be carbohydrate (e.g., Polycose, Ross Products Division, Abbott Laboratories; Moducal, Mead Johnson Nutritionals), fat (e.g., Microlipid, Mead Johnson Nutritionals, vegetable oil), or protein (e.g., Beneprotein, Novartis Medical Nutrition). When diverting from a standard formula dilution, it is essential to evaluate the final product's macronutrient distribution and to ensure that vitamin and mineral intake is appropriate. Recommended macronutrient distribution is as follows: 7% to 16% calories from protein, 30% to 55% calories from fat, and 30% to 60% calories from carbohydrate (Groh-Wargo et al., 2000).

Often referred to as postdischarge premature formulas or nutrient-enriched formulas, EnfaCare Lipil (Mead Johnson Nutritionals) and Similac NeoSure Advance (Ross Products Division, Abbott Laboratories) provide more calories, protein, vitamins, and minerals than standard term formulas to support rapid growth and prevent nutrient depletion without providing excessive nutrients (Groh-Wargo et al., 2000). Carver and associates (2001) demonstrated that premature infants fed postdischarge formula had improved growth compared with those fed a term formula, with the most significant beneficial effects seen among infants with birth weights less than 1,250 g. Greater gains in weight and head circumference were seen in the infants fed postdischarge formula, especially within the first 1 to 2 months after discharge. This study and several others support the use of nutrient-enriched formulas for preterm infants after discharge (Groh-Wargo et al., 2000). For optimal growth and bone mineralization, providing postdischarge premature formulas to infants born prematurely is recommended until catch-up growth is completed or until 9 to 12 months corrected age, whichever occurs first (Groh-Wargo et al., 2000; Henderson, Fahey, & McGuire, 2005; Lucas et al., 1992).

For infants born appropriate for gestational age at 34 to 37 weeks gestation, clinical judgment should determine the appropriate duration for the use of postdischarge premature formulas, considering each patient case individually. Bhatia (2005) recommended transition from a postdischarge premature formula to a standard term formula at 4 to 6 months corrected age if all growth parameters are above the 25th percentile. A recent Cochrane Review concluded that "the limited available data do not provide strong evidence that feeding preterm or low birth weight infants following hospital discharge with calorie and protein-enriched formula compared with standard term formula affects growth rates or development up to 18 months post-term" (Henderson et al., 2005). Further studies focusing on VLBW, extremely low birth weight, and micropremie infants are needed and may reveal a significant effect of postdischarge premature formulas on growth rates and development.

Ultimately, the primary care physician is responsible for ensuring that premature infants continue on a postdischarge formula for an appropriate duration. The current standard of practice is to use postdischarge premature formulas until the infant reaches 9 to 12 months corrected age. The cost for postdischarge premature formulas is approximately 10% to 30% more than standard formulas, depending on geographic location and place of purchase (e.g., grocery vs. drug store). Although these postdischarge formulas cost slightly more than term formulas, the benefits usually outweigh the costs. For patients who meet financial eligibility requirements, the Special Supplemental Nutrition Program for Women, Infants, and Children does cover these formulas.

Preterm infants with birth weights less than 1,800 g fed soy protein–based formulas had less weight gain, less linear growth, and lower serum albumin than those fed cow's milk–based formula. Preterm infants fed soy protein–based formulas also had lower phosphorus levels and higher alkaline phosphatase levels, thus increasing the risk for development of **osteopenia of prematurity** (Callenbach, Sheehan, Abramson, & Hall, 1981; Kulkarni, Hall, & Rhodes, 1980). Therefore, the American Academy of Pediatrics (AAP) concluded that soy protein–based formulas should not be fed to infants weighing less than 1,800 g at birth (American Academy of Pediatrics [AAP] Committee on Nutrition, 1998; Shenai, Jhaveri, & Reynolds, 1981).

**osteopenia of prematurity** Reduction in bone volume to below normal levels, often seen in infants born prematurely.

### Feeding Evaluation

The introduction and maintenance of oral feeding in premature infants presents many unique challenges. One study recently surveyed NICU feeding practices, and results revealed inconsistent and often contradictory strategies in attempting to transition high-risk infants from gavage to oral feedings (Premji, McNeil, & Scotland, 2004). Effective sucking, intact gag reflex, and intact cough reflex are requirements for safe and successful oral feeding and generally correlate with an intact neurologic system and physiologic maturity. Preterm infants are particularly prone to feeding issues because of their difficulty coordinating the suck-swallow-breathe pattern, physiologic instability (i.e., respiratory or cardiac problems), behavioral state, and minimal energy reserve (Premji et al., 2004). Immature GI systems also contribute to feeding problems. A small percentage of premature infants never fully orally feed and require long-term enteral nutrition support (i.e., tube feedings) (Geggie, Dressler-Mund, Creighton, & Cormack-Wong, 1999).

### Powdered Formula in the NICU

The following statement was released by the Food and Drug Administration (FDA) in 2002[1]:

> The literature suggests that premature infants and those with underlying medical conditions may be at highest risk for developing *E. sakazakii* infection. Several outbreaks have occurred in neonatal intensive care units worldwide.... The Centers for Disease Control and Prevention (CDC) has communicated information to FDA about a fatal infection due to *E. sakazakii* meningitis in a neonatal intensive care unit in the United States. In CDC's subsequent investigation, a cluster of neonates with *E. sakazakii* infection or colonization were identified in association with a powdered infant formula containing these bacteria. As background information for health professionals, FDA wants to point out that powdered infant formulas are not commercially sterile products. Powdered milk-based infant formulas are heat-treated during processing, but unlike liquid formula products they are not subjected to high temperatures for sufficient time to make the final packaged product commercially sterile. FDA has noted that infant formulas nutritionally designed for consumption by premature or low birth weight infants are available only in commercially sterile liquid form. However, so-called "transition" infant formulas that are generally used for premature or low birth weight infants after hospital discharge are available in both noncommercially sterile powder form and commercially sterile liquid form. Some other specialty infant formulas are only available in powder form.... In light of the epidemiological findings and the fact that powdered infant formulas are not commercially sterile products, FDA recommends that powdered infant formulas not be used in neonatal intensive care settings unless there is no alternative available.

1. Courtesy of the FDA (www.fda.gov/Food/RecallsOutbreaksEmergencies/SafetyAlertsAdvisories/ucm111299.htm).

### Enteral Nutrition

Early trophic feeding, also referred to as minimal feedings or GI priming, has been shown to decrease the time to attain full feeds, the time to regain to birth weight, the duration of PN, the number of days feedings are held for residuals or intolerance, and duration of hospital stay, without increasing the incidence of NEC (Landers, 2003; Tyson & Kennedy, 2005). In addition, benefits of trophic feeding into the intact intestine are maturation of neonatal intestinal digestive, absorptive, and motor and immune function; improved neonatal feeding tolerance and behavior; maintenance of intestinal function during starvation and catabolic states; and prevention of intestinal bacterial overgrowth and bacterial translocation (Sondheimer, 2004). Trophic feedings also have been shown to improve nitrogen and mineral retention (Landers, 2003). Feedings are classified as trophic if they comprise less than 25% of the patient's nutritional needs or provide less than 20 mL/kg/day (Sondheimer, 2004). Delayed enteral feeding leads to gut mucosal atrophy, decreased enzyme activity, and impaired gut growth and immunity.

Premature infants often do not tolerate enteral feedings because of their small stomach capacity and immature GI tract (e.g., dysmotility and decreased digestive enzymes). Gastric emptying and intestinal transit times are significantly delayed when compared with the term infant (Weckwerth, 2004). "Organized" gut motility does not begin until 32 to 34 weeks gestation (Thureen & Hay, 2001). Supplemental PN is often required while enteral feedings advance to their goal volume.

#### Bolus Versus Continuous Feedings

In a study of infants with birth weights of 750 to 1,500 g, time to full feeds, time to regain birth weight, discharge, and nitrogen retention/balance studies were no different between the continuous and intermittent (e.g., gavage feeds every 3 hours) feeding groups (Silvestre, Morbach, Brans, & Shankaran, 1996). Another study assessing feeding tolerance in premature infants found the time to reach full feeds (160 mL/kg/day) was significantly higher in the continuous gastric infusion than in the intermittent gastric bolus. There was no significant difference noted in time to regain to body weight, however. Feeding intolerance was defined as gastric residuals greater than 21% of feed volume and/or emesis. Abdominal distention was also monitored. A plausible explanation is that there needs to be

some stomach distention by a minimum volume of feeds to optimize gut peristalsis/gastric emptying (Dollberg, Kuint, Mazkereth, & Mimouni, 2000). Another study also reported less intolerance to intermittent feedings versus continuous infusions (Schanler, Shulman, Lau, O'Brian Smith, & Heitkemper, 1999). There are several advantages to intermittent feeds: easier to administer, facilitate monitoring of gastric residuals, provide optimal nutrient delivery, enhance surges of gut hormones, and allow easier transition to oral feeding schedule. Advantages of continuous feedings are minimal gastric distention, reduced adverse pulmonary effects (i.e., less pressure on the diaphragm), improved tolerance in infants with apnea and bradycardia, and increased nutrient absorption with GI disease.

#### Transpyloric Versus Gastric Feedings

Transpyloric feedings (e.g., nasoduodenal and orogastric jejunum) have been associated with increased mortality in preterm infants. Also, reports of GI disturbance were significantly higher in infants fed via transpyloric tubes. No evidence of significantly different rates of growth was found when comparing transpyloric with gastric feedings (McGuire & McEwan, 2004).

## Introduction of Solid Foods

The introduction of solid foods should be based on a premature infant's developmental readiness, not chronologic age. Corrected age is a more true reflection of an infant's developmental stage than chronologic age is, and many preterm infants are not developmentally ready for solid foods until at least 6 months corrected age. Feeding guidelines for the premature infant should also take into account the degree of immaturity at birth because extremely premature infants are more likely to experience developmental delay. Anticipatory guidance for caregivers of neurologically impaired infants should stress the safe introduction of solids; introduction of solid foods before developmental readiness, based solely on corrected age, may result in "forced feedings" of solids and decreased intake of breast milk or formula, often reducing protein and mineral intake (Marino et al., 1995). Introducing solid foods or complex consistencies before developmental readiness also may increase the risk for aspiration. Reported inappropriate feeding practices include the early introduction of solids, early feeding of cow's milk, and feeding of low-fat cow's milk. These practices may have reflected infants who were perceived in terms of chronologic age rather than age corrected for prematurity (Dhillon & Ewer, 2004).

### Feeding Disorders

Feeding problems are often encountered during follow-up of high-risk neonates (Groh-Wargo et al., 2000). A delay in feeding skills may be related to frequent or prolonged illness, oral defensiveness, decreased exposure to oral feeding, or parental misperception of infants' feeding cues. A common feeding aversion in infants with complex medical issues is "tactile defensiveness," in which infants have an adverse response to having objects placed in or near the oral cavity (Wheeler & Hall, 1996). Infants with neurologic or anatomic abnormalities may have poor suck–swallow coordination, excessive tongue thrust, problems with gag reflex, or gastroesophageal reflux (Groh-Wargo et al., 2000; Hawdon, Beauregard, Slattery, & Kennedy, 2000). Respiratory and cardiac diseases often increase the work of breathing, compromising an infant's ability to eat by decreasing feeding endurance (Bernstein et al., 1998).

Marino and associates (1995) observed an increased incidence (63%) of gastroesophageal reflux in a group of preterm infants. Gastroesophageal reflux is commonly identified in infants born prematurely; however, there is a lack of randomized trials regarding the standardization of treatments for gastroesophageal reflux (Rommel, DeMeyer, Feenstra, & Veereman-Wauters, 2003). Treatment strategies range from using a "reflux sling" to medications to thickening feeds with cereal, depending on the belief of the neonatologist.

Neonates who have the most complex or severe conditions are most at risk for the development of "disorganized" or "dysfunctional feeding." In addition, medical interventions often delay the introduction and establishment of oral (sucking) feeding. It is possible that the interventions required and the disordered feeding patterns are both consequences of the degree of severity of illness or prematurity (Ritchie, 2002). Feeding disorders may be a consequence of improved neonatal survival. Rommel and colleagues (2003) demonstrated a significant correlation between prematurity/dysmaturity and feeding disorders. Children with feeding disorders had significantly lower birth weights for gestational age,

which implies that feeding problems could be related to intrauterine growth restriction. Data showed that infants born before 34 weeks gestation had more GI and oral sensory problems (Rommel et al., 2003).

It may be difficult for premature infants to take adequate nutrients via oral feeds because effective feeding requires coordination of suck, swallow, and breathing. Feedings lasting more than 30 minutes may tire infants and result in a net energy loss. Supplemental tube feedings may be an effective adjunct therapy for these infants. Options for administering supplemental tube feedings include providing oral feedings followed by bolus tube feedings every 3 to 4 hours around the clock to meet a minimum daily volume requirement, or providing oral/bolus feedings during the day with supplemental continuous tube feedings via pump overnight. Several factors influence which method of supplemental tube feedings is chosen, including but not limited to parenteral preference, aspiration risk, compromised respiratory status, and dysmotility. Proposed criteria (Gray & King, 1997) for referral to a feeding therapist include the following:

- Suck or swallow incoordination or weak suck
- Delayed or absent progress to spoon feedings or table foods within expected time
- Discomfort, increased fussiness, distress, severe irritability, and/or arching during feedings
- Disrupted breathing or apnea during feedings
- Lethargy, decreased arousal during feedings, or tires easily and has difficulty finishing a feeding
- Feeding lasting longer than 30 minutes for an infant or 45 minutes for a child
- Abnormal oral-motor anatomy or physiology: lips, tongue, jaw, or palate
- Choking, excessive gagging, or recurrent coughing during feedings, toward the end of feeding, or between feedings
- Recurrent vomiting during or after a feeding
- Unexplained food refusal or inability to take adequate nutrition to support growth
- Physical symptoms or conditions such as dysphagia, recurrent pneumonia, aspiration, hypertonicity, hypotonicity, and failure to thrive

## Fluid and Electrolyte Management

It is essential to reevaluate an infant's fluid status daily, at least for the first week of life. In the first day of life, a full-term infant requires as little as 60 mL/kg/day to meet fluids needs. As an infant matures, changes in renal solute load, stool water output, and infant growth increase fluid needs (Groh-Wargo et al., 2000). In preterm infants fluid needs are 80 mL/kg/day on day of life 1, increasing to 120–160 mL/kg/day as the infant matures (Groh-Wargo et al., 2000). Preterm infants have increased insensible water losses resulting from the immaturity of their skin and respiratory losses.

Close monitoring of electrolyte status is essential. Electrolytes are often not administered on the first day of life, with the exception of calcium (Groh-Wargo et al., 2000). Potassium is generally added once normal kidney status and good urine output are established, and sodium is often added once diuresis begins (Groh-Wargo et al., 2000). Daily adjustment to electrolyte intake often is necessary. Electrolyte requirements of preterm and full-term infants are generally similar.

## Parenteral Nutrition

**Parenteral nutrition (PN)** is the delivery of nutrients into the circulatory system. If one anticipates that an infant will not receive enteral feedings within the first 6 to 12 hours after birth, PN should be initiated once electrolyte and fluid stability are achieved. Infants in the NICU often require PN for at least the first days of life to maximize caloric intake until enteral feedings are adequate. Premature infants have very minimal caloric reserves given that most fetal nutrient stores are deposited during the last 3 months of pregnancy. For example, in a preterm infant weighing 1,000 g, fat contributes only 1% of total body weight as compared with a term infant (3,500 g), which is about 16% fat (Anderson, 1996). If adequate nutrition support cannot be achieved and fat and glycogen stores have been exhausted, infants begin to catabolize protein stores for energy.

**parenteral nutrition (PN)** The delivery of nutrients into the circulatory system.

PN support should be initiated immediately when medical conditions contraindicate enteral feedings. PN should be continued until the medical situation allows for safe initiation of enteral feedings, and then PN may be weaned as the enteral feedings are advanced to their goal. Timing of the discontinuation of PN varies, but many clinicians are comfortable doing so when enteral feedings are tolerated at 100 to 120 mL/kg/day.

### Initiating Parenteral Nutrition

PN can be administered through a peripheral vein, which is called peripheral parenteral nutrition. Peripheral parenteral nutrition requires a relatively large volume to allow for adequate administration of nutrients. In the ill infant, nutrient requirements often cannot be met with peripheral parenteral nutrition because of fluid restriction. Limiting dextrose concentration to 10% to 12.5% with a final osmolality of 900 mOsm/kg is recommended to minimize risk of phlebitis and infiltration (AAP Committee on Nutrition, 1983).

Total parenteral nutrition requires central vein access and allows for administration of a solution with a higher osmolality (i.e., > 900 mOsm/kg). Venous access is not defined by the initial point of entry but by the position of the catheter tip. With central venous lines the catheter tip terminates in the superior vena cava or the right atrium of the heart. Examples of central venous lines that are found in infants include umbilical venous catheters, nontunneled lines, and tunneled lines. Umbilical venous catheters are generally placed after birth and are removed within 2 weeks because of increased risk of infection (Pereira, 1995). Nontunneled lines include femoral, jugular, subclavian, and peripherally/percutaneously inserted central catheters (PICCs). PICC lines are generally placed when a long-term intravenous access is needed and duration may vary from several days to months. PICC lines may be central or peripheral, depending on the placement of the catheter tip. If the PICC line terminates in the superior vena cava, it is considered to be central, and termination in other vessels needs to be individually evaluated to determine the maximum dextrose concentration that can be safely infused. A Broviac line is the tunneled line typically used in pediatric patients, comparable with a Groshong or Hickman in adults. Broviac lines are often placed when an infant is expected to be discharged on home PN support. X-ray confirmation of central line placement is essential before administration of a solution with a high osmolality. Dextrose concentration is generally limited to a maximum of 25% to 30%.

Parenterally, carbohydrates are administered in the form of dextrose, which provides 3.4 cal/g. For optimal brain function, initiating with 4 to 6 mg/kg/min a day is recommended (i.e., D7.5 10%). Typically, advancement of 1 to 2 mg/kg/minute a day to a maximum of 11 to 14 mg/kg/minute a day is tolerated; however, preterm neonates may have unpredictable glycemic control. Glucose intolerance (e.g., stress-induced hyperglycemia) persisting greater than 24 to 48 hours with a glucose infusion rate (i.e., milligrams dextrose per kilogram per minute a day) of less than 4 to 6 may warrant the initiation of an insulin infusion to allow for provision of more dextrose.

Protein is administered as a crystalline amino acid solution, which provides 4 cal/g. TrophAmine and Aminosyn PF are amino acid solutions most appropriate for use in infants because of the addition of taurine, *N*-acetyl-L-tyrosine, glutamic acid, and aspartic acid (Groh-Wargo et al., 2000). TrophAmine is formulated to promote growth in neonates and young infants and to achieve a plasma amino acid pattern similar to that of normal postprandial breastfed infants (Heird, Dell, & Helms, 1987). Research has shown a potential decrease in PN-associated cholestasis with TrophAmine (Wright, Ernst, Gaylord, Dawson, & Burnette, 2003). Another benefit of TrophAmine is that when L-cysteine is added, there is an increased solubility of calcium and phosphorus to promote optimal bone mineralization (Schmidt, Baumgartner, & Fischlschweiger, 1986). TrophAmine is initiated with 2 to 3 g amino acid/kg/day for infants expected to have normal renal function, titrating by 1 g amino acid/kg/day as tolerated to a goal of 3 to 4 g amino acid/kg/day, the higher end of the range being most appropriate for the smallest infants (e.g., < 1,000 g) (Porcelli & Sisk, 2002).

Parenteral fat is a lipid emulsion of either soybean oil or a combination of safflower and soybean oils. Intravenous lipids provide 10 cal/g regardless of the product concentration and are available in 10% (1.1 cal/mL) and 20% (2 cal/mL) concentrations. For infants, the 20% concentration is preferred over the 10% product because it allows adequate lipid intake in less volume and improved clearance of phospholipids and triglycerides (Groh-Wargo et al., 2000). Not only are lipids a concentrated source of calories, but they also provide essential fatty acids for cell membrane integrity and brain development. Lipids also help to prolong the integrity of peripheral lines because of their lower osmolality. It is crucial to provide a minimum of 0.5 to 1.0 g lipids/kg/day to prevent essential fatty acid deficiency, which can develop in premature infants during the first week of life and as early as the second day of life (Groh-Wargo et al., 2000). Essential fatty acid

deficiency is defined as a triene-to-tetraene ratio of at least 0.4 (Kerner, 1996). Lipids are initiated with 1 to 2 g/kg/day, with titration as tolerated to a maximum of 3 to 3.5 g/kg/day. Premature infants have a maximum lipid oxidation rate of 0.125 g/kg/minute, whereas term infants may tolerate up to 0.15 g/kg/minute.

Most cases requiring PN support result from either GI malformations or NEC. GI malformations include omphalocele, gastroschisis, intestinal atresia, volvulus, and Hirschsprung disease. An omphalocele is a congenital herniation of internal organs into the base of the umbilical cord, with a covering membranous sac. In contrast, gastroschisis is a defect in the abdominal wall resulting from rupture of the amniotic membrane, usually accompanied by protrusion of internal organs. Both conditions require surgical repair. Recovery from omphalocele is often quicker than that from gastroschisis because the bowel was protected by the membranous sac during pregnancy. In gastroschisis, the bowel is often quite dilated and inflamed because of exposure to amniotic fluids. Intestinal atresia is the absence of a normal opening (i.e., the intestinal lumen has a closure where it should be a continuous segment); this condition requires surgical repair as well. A volvulus is a twisting of the intestines, causing an obstruction. Hirschsprung disease (i.e., congenital megacolon) often presents as constipation and is characterized by the absence of ganglion cells in a distal intestinal segment. Various surgical treatments exist for treating Hirschsprung disease, such as a diverting colostomy or resection of the aganglionic bowel with a "pull through" of the normal bowel down to the distal rectum (Groh-Wargo et al., 2000).

NEC is an inflammation or death of the intestinal tract. Some cases of NEC are classified as "medical" because a period of bowel rest and administration of antibiotics are sufficient to promote a full recovery. Feedings are typically held for 7 to 14 days. Surgical NEC are those cases that require surgical intervention, such as peritoneal drain placement and/or intestinal resection.

The cause of NEC is unknown. Historically, rapid titration of feedings was implicated as the cause of NEC, but more recently this association has been questioned. Several retrospective studies suggested that rapid advancement of feedings was associated with increased NEC (Anderson & Kliegman, 1991; McKeown, Marsh, & Amarnath, 1992). Anderson and Kliegman (1991) concluded that "the feed increment rate from initiation of feeds to day of maximum feeds was 27.8 ± 16 ml/kg/day for the NEC patients and 16.8 ± 11 for the control patients." However, a recent prospective randomized controlled study concluded that among infants weighing between 1,000 and 2,000 g at birth, starting and advancing feedings at 30 mL/kg/day had no significant impact on incidence of development of NEC and resulted in fewer days to reach full-volume feedings as compared with 20 mL/kg/day (Caple, Armentrout, & Huseby, 2004). That study confirmed earlier findings by Rayyis, Ambalavanan, Wright, and Carlo (1999) that a 35-mL/kg/day feed advancement did not affect the incidence of NEC.

Because standards have not been set for evaluating gastric residuals in premature infants, there are many differences between NICUs in interpreting gastric residuals. A recent retrospective study concluded, however, that a gastric residual volume greater than 33% of a previous feed was associated with a higher risk for NEC (Cobb, Carlo, & Ambalavanan, 2004). A meta-analysis found that feeding with donor human milk was associated with a significantly reduced relative risk of NEC; infants who received donor human milk were three times less likely to develop NEC and four times less likely to have confirmed NEC than were infants who received formula milk (McGuire & Anthony, 2003).

GI malformations and NEC can lead to **short bowel syndrome (SBS)**, also known as short gut syndrome, which may be composed of weight loss or inadequate weight gain, muscle wasting, diarrhea, rapid GI transit time, malabsorption, dehydration, and hypokalemia. SBS is a malabsorptive state caused by a significant bowel resection or congenital defect. SBS may present with a decreased ability to secrete GI regulatory peptides and trophic hormones as well as compromised GI immune function (J. Vanderhoof, 2004). Causes of SBS in the NICU often include NEC, gastroschisis, volvulus, or multiple intestinal atresias. Generally, a full-term infant has approximately 200 to 250 cm of small bowel (J. A. Vanderhoof, 2003). SBS may be defined as less than 75 to 100 cm of small bowel. After a bowel resection, the remaining small bowel adapts in the following ways: mucosal hyperplasia, villus lengthening, increased crypt depth, and bowel dilatation (Andorsky et al., 2001). Provision of enteral

**short bowel syndrome (SBS)** A malabsorptive state caused by a significant bowel resection or congenital defect.

nutrition is the main factor influencing bowel adaptation after a resection.

In some cases, the postresection gut adaptation is sufficient to allow for a patient to wean off PN and sustain adequate growth on enteral nutrition. Given the complications of PN (e.g., line sepsis, cholestatic liver disease, and poor bone mineralization) in the NICU, weaning from PN support should be a priority. Many SBS patients, however, require at least supplemental PN to sustain adequate growth and/or hydration. An infant with an intact ileocecal valve and at least 10 to 30 cm of small bowel may be able to wean from PN successfully. Without an ileocecal valve, at least 30 to 50 cm of small bowel are generally needed to wean from PN (Kurkchubasche, Rowe, & Smith, 1993; Quiros-Tejeira, Ament, & Reyen, 2004). Andorsky and associates (2001) observed that the mean residual bowel length in the group of patients able to be weaned from PN was 88.6 cm as compared with 71.7 cm in the group unable to be weaned from PN. Factors associated with a reduction in duration of PN include percentage of enteral feeding days when breast milk or an amino acid–based formula was given and percentage of caloric intake received by the enteral route 6 weeks after intestinal resection (Andorsky et al., 2001). Some studies associated the presence of an ileocecal valve with a decreased duration of PN, but at least four other studies did not support this theory (Andorsky et al., 2001). Early restoration of intestinal continuity (i.e., ostomy "take-down") appears to correlate with less severe PN-related cholestasis (Andorsky et al., 2001).

Patients with SBS are prone to morbidity and mortality from progressive liver failure and sepsis (Andorsky et al., 2001). PN cholestasis is defined as a conjugated bilirubin greater than 2 mg/dL. Typically, patients with cholestasis are jaundiced and may have pale, clay-colored stools as a result of fat malabsorption. Neonates are especially prone to PN cholestasis because of their diminished bile acid pool size and key hepatobiliary transporters and their reduced enterohepatic circulation of bile acids (i.e., decreased bile flow) (Karpen, 2002). Karpen (2002) observed that patients with elevated conjugated bilirubin at approximately 4 months of age or older may have irreversible PN cholestasis. Recognizing end-stage liver disease as early as possible is essential, and referral for liver or liver plus small bowel transplantation should be made when medically appropriate (Karpen, 2002).

Goals of nutrition therapy for the infant with SBS should include promoting normal growth and development, maximizing intestinal adaptation, and minimizing complications. Initially, the focus of nutritional therapy is maintaining fluid and electrolyte balance, which is often accomplished through intravenous fluid (e.g., one-half normal saline with KCl) replacement of ostomy losses (J. Vanderhoof, 2004). Once fluid and electrolyte losses have resolved and GI function has returned (i.e., Salem sump is placed to gravity drainage without increased abdominal distention), trophic enteral feeds should be initiated. Andorsky and associates (2001) demonstrated that the use of breast milk showed the highest correlation with shorter PN courses. If breast milk is not available, hydrolyzed or amino acid–based formulas are generally used to facilitate absorption (J. Vanderhoof, 2004). Pasteurized donor breast milk should also be considered if the infant's mother is unable or unwilling to provide breast milk. Amino acid–based formulas may improve outcomes in SBS because there seems to be a higher incidence of allergy in children with SBS. Also, the infant amino acid–based formula used in several studies (e.g., Neocate, SHS, Inc.) contains a high percentage of fat from long-chain fatty acid sources, which have been shown to stimulate mucosal adaptation better than medium-chain triglycerides in animal models (Andorsky et al., 2001; Bines, Francis, & Hill, 1998). An amino acid–based formula recently approved for use in infants (e.g., Elecare, Ross Laboratories) has also been administered to SBS patients with good tolerance. Elecare and Neocate include a similar amino acid base; however, Elecare contains significantly more medium-chain triglycerides when compared with Neocate (33% of total fat vs. 5%), facilitating optimal fat absorption during cholestasis.

Continuous enteral feedings are advantageous over bolus feedings because of enhanced absorption, which is facilitated by saturation of transporters in the gut 24 hours per day. Aggressive enteral nutrition reduces the need for PN as early as possible and helps to ameliorate the development and progression of PN cholestasis; moreover, aggressive enteral nutrition stimulates gut adaptation (J. Vanderhoof, 2004). Factors hindering advancement of enteral feeds include stool losses increasing more than 50% over baseline, ostomy output greater than 40 to 50 mL/kg/day, and ostomy output strongly positive for reducing substances (J. Vanderhoof, 2004).

Once weaned from PN support, SBS patients should routinely be screened for fat-soluble vitamins (A, D, E, and K), calcium, magnesium, and zinc deficiencies. Once a patient is tolerating full enteral feeds and PN has been discontinued, providing the following vitamin supplementation is recommended: TPGS-E (e.g., Liqui-E or Nutr-E-Sol) at 25 IU/kg/day plus 2 mL liquid multivitamin (e.g., Poly-vi-sol) to provide adequate vitamins A and D and vitamin K (e.g., phytonadione) from 1 to 2.5 mg weekly up to 5 to 10 mg daily, according to need (Francavilla, Miniello, Lionetti, & Armenio, 2003; Karpen, 2002). Karpen (2002) stated that the "use of this combination vitamin treatment is superior to use of available water-soluble vitamin supplements" (e.g., vitamins A, D, E, and K). Dosing of vitamin supplements can be adjusted as needed based on the results of monthly measurements of vitamin A, 25-OH vitamin D, vitamin E, and coagulation profile.

Caring for infants with SBS often is a challenging undertaking. Daily or weekly changes are made to increase enteral nutrition support and to minimize the need for PN, with continual monitoring of growth and laboratory results as well as monitoring for symptoms of intolerance of enteral nutrition. NICU dietitians' close collaboration with the medical and surgical teams as well as the gastroenterologist and hepatologist, is essential to facilitating a positive outcome for the SBS patient.

Other medical conditions that may warrant the use of PN support include intractable nonspecific diarrhea, extracorporeal membrane oxygenation, congenital diaphragmatic hernia, and vasopressor support (e.g., dopamine or dobutamine). Extracorporeal membrane oxygenation and vasopressor support are not absolute contraindications to enteral feedings; however, many neonatologists are hesitant to enterally feed infants with hemodynamic instability because of risk of bowel ischemia. Diaphragmatic hernia often requires PN until surgical repair of the hernia occurs and the stomach and/or intestines are returned to a lower place in the abdominal cavity and are functioning (e.g., "stooling").

### Vitamins and Trace Elements in Parenteral Nutrition

Currently available pediatric parenteral multivitamins do not optimally meet the vitamin needs of preterm infants (Greene, Hambidge, Schanler, & Tsang, 1988). The available formulation provides higher amounts of thiamin, riboflavin, pyridoxine, and cyanocobalamin and lower amounts of fat-soluble vitamins (e.g., vitamin A) than recommended.

Available trace element products do not adequately meet the needs of preterm neonates either. Specifically, if sufficient zinc is administered, manganese is provided in excessive amounts. However, short-term use of a standard neonatal trace element product is considered to be safe. Many institutions with NICUs have begun developing their own trace element combinations from individual trace element products to better meet the needs of their smallest patients. In cases of specific medical conditions, such as renal (e.g., chromium, selenium) or hepatic (e.g., copper, manganese) disease, the clinician may need to alter specific trace elements.

Zinc is important for the maintenance of cell growth and development. When PN is supplemental to enteral nutrition or of short duration, zinc is the only trace element that requires supplementation. Some conditions that require additional zinc intake include elevated urinary zinc excretion (e.g., high-output renal failure) and increased GI excretion (e.g., high-volume stool/ostomy losses, fistula/stoma losses).

Copper is an essential constituent of many enzymes. Current daily recommendations are adequate to prevent deficiency in preterm infants. Copper deficiency is more likely to occur with high-dose zinc therapy because of the inverse relationship of these two minerals. Clinical manifestations of copper deficiency include hypochromic anemia that is unresponsive to iron therapy, neutropenia, and osteoporosis. Rapid growth in VLBW infants increases the risk of deficiency. Conditions requiring higher copper intake include increased biliary losses resulting from jejunostomy and losses via external biliary drainage. These conditions may require an additional 10 to 15 µg/kg/day. Historically, copper was withheld in PN for patients with cholestasis; however, cases of copper deficiency have recently been reported when copper was withheld in PN (Hurwitz, Garcia, Poole, & Kerner, 2004). It is recommended in patients with cholestasis to reduce supplementation by 50% (i.e., 10 µg/kg/day), monitor monthly serum copper levels and ceruloplasmin, and adjust supplementation accordingly.

Manganese is an important component of several enzymes. Manganese deficiency has not been documented in humans. However, manganese toxicity has been reported (Rao & Georgieff, 2005). Manganese supplementation in PN should be withheld in patients with cholestasis or other

liver function impairment (Fok et al., 2001; Rao & Georgieff, 2005). Fok and associates (2001) provided evidence suggesting that high manganese intake contributes to the development of cholestasis. Manganese should therefore be used with caution in PN provided to infants because they are more susceptible to cholestasis (Fok et al., 2001). Monthly serum manganese levels should be monitored and supplementation adjusted as needed.

Selenium is a component of the enzyme glutathione peroxidase, which is involved in protecting cell membranes from peroxidase damage through detoxification of peroxides and free radicals (Rao & Georgieff, 2005). Supplementation with selenium is recommended in long-term PN (i.e., > 1 month). Decreasing selenium intake when renal dysfunction is present is recommended.

Chromium potentiates the action of insulin and is required for growth in light of its role in glucose, protein, and lipid metabolism (Rao & Georgieff, 2005). Decreasing chromium intake with renal dysfunction is recommended.

Molybdenum supplementation is recommended in cases when exclusive PN exceeds 4 weeks. Deficiency of molybdenum has not been reported in premature infants; however, one adult case of deficiency has been documented (Rao & Georgieff, 2005).

Iodine is often omitted from PN given that iodine-containing disinfectants and detergents are used on the skin and absorbed. The Committee on Clinical Practice Issues of the American Society of Clinical Nutrition recommended parenteral intakes of iodine at 1.0 µg/kg/day for the preterm infant to avoid any risk of deficiency (Rao & Georgieff, 2005).

Iron supplementation should be considered only among long-term PN-dependent patients who are not receiving frequent blood transfusions. Iron supplementation of 100 µg/kg/day may be safely delayed until 3 months of age in term infants. Preterm infants *not* receiving blood products, with the exception of platelets, may benefit from iron supplementation of 100 to 200 µg/kg/day at 2 months of age (Rao & Georgieff, 2005). Monitoring iron status is imperative with iron supplementation because there is a risk of iron overload (Mirtallo et al., 2004).

### Other Parenteral Nutrition Additives

Neonates and infants are unable to endogenously produce carnitine. Premature neonates also have limited tissue carnitine stores. Premature infants less than 34 weeks gestation receiving PN without carnitine can develop carnitine deficiency 6 to 10 days after birth (Schmidt-Sommerfield, Penn, & Wolf, 1982). Human milk is a good source of carnitine. PN does not contain carnitine unless it is added to the PN solution. A recent meta-analysis concluded that there was no evidence to support routine supplementation of parenterally fed neonates with carnitine (Cairns & Stalker, 2000). However, four of the six studies included in this review demonstrated positive effects on either growth or lipid tolerance. Clinicians may provide 10 to 20 mg carnitine/kg/day without risk of adverse effects (Crill, Christensen, Storm, & Helms, 2006; Putet, 2000).

Cysteine, a conditionally essential amino acid, is not a component of crystalline amino acid solutions because it is unstable and forms an insoluble precipitate (Groh-Wargo et al., 2000). In adults, cysteine can be synthesized from methionine; however, preterm infants lack adequate hepatic cystathionase to facilitate this conversion. Commonly recommended dosing for L-cysteine hydrochloride is 40 mg/g amino acids (Mirtallo et al., 2004). One benefit of the addition of L-cysteine hydrochloride to PN is the decrease in the pH of the solution, which increases the solubility of supplemental calcium and phosphorus. It should be noted, however, that the addition of cysteine to PN warrants close monitoring of an infant's acid–base status because it may predispose infants to acidosis, and acetate may need to be added to the solution.

Addition of heparin to PN solutions "reduces the formation of a fibrin sheath around the catheter, may reduce phlebitis … and increases the duration of catheter patency" (Groh-Wargo et al., 2000). Heparin also stimulates the release of lipoprotein lipase, which may improve lipid clearance. Adding 0.25 to 1.0 units heparin per milliliter of PN solution is recommended. However, there is an increased risk of anticoagulation with the higher doses of heparin.

### Parenteral Nutrition and Biochemical Monitoring

Before initiation of PN support, the following biochemical indices should be checked: basic metabolic panel, calcium, magnesium, phosphorus, liver function tests (i.e., alkaline phosphatase, alanine and aspartate aminotransferases, and gamma-glutamyltransferase), total bilirubin, conjugated or direct bilirubin, prealbumin, albumin, and triglyceride. The basic metabolic panel, calcium,

magnesium, phosphorus, and triglyceride levels should be checked daily for 3 days after the initiation of PN support or until indices are stable. Weekly liver function tests, total bilirubin, conjugated or direct bilirubin, prealbumin, albumin, and triglyceride should be monitored. Other biochemical studies (e.g., iron status, vitamin levels, ionized calcium, serum zinc, copper, manganese) may be warranted on an individual basis. Patients on long-term PN, such as those with SBS, need monthly monitoring of vitamin and mineral status.

### Parenteral Nutrition Complications

Short-term potential adverse effects of PN include infection, hyperglycemia, electrolyte abnormalities, disturbance of acid–base balance, hypertriglyceridemia, bacterial translocation, and compromised gut integrity. With long-term PN, adverse effects may include infection, PN cholestasis, metabolic complications, disturbance of acid–base balance, osteopenia, risk of vitamin/mineral deficiency or toxicity, and continued risk of bacterial translocation. Nosocomial infections appear to result either from improper care of the catheter and/or frequent use of the catheter for purposes other than delivery of nutrients (e.g., blood draws, medication administration).

Episodes of line infection can hasten the rising of the bilirubin level. Further, cholestasis may develop in 90% of infants after the first line infection (Beath et al., 1996; Sondheimer, Asturias, & Cadnapaphornchai, 1998). A multidisciplinary nutrition support service and early discharge on home PN has been shown to reduce the incidence of central venous catheter infection (Beath et al., 1996; Knafelz et al., 2003).

Often, the terms *osteopenia* and *rickets* are used interchangeably. However, osteopenia is defined as a "reduction in bone volume to below normal levels especially due to inadequate replacement of bone lost to normal lysis," whereas rickets is defined as "a deficiency disease that affects the young during the period of skeletal growth, is characterized especially by soft and deformed bones, and is caused by failure to assimilate and use calcium and phosphorus normally due to inadequate sunlight or vitamin D" (Medline Plus Medical Dictionary, 2013a, 2013b). Some experts proposed that the term *rickets* should be reserved for radiographic or physical findings. Fractures may occur in osteopenic premature infants, with or without radiologic features of rickets. The incidence of rickets or fractures in infants with a body weight less than 1,500 g ranges from 20% to 32%, increasing to 50% to 60% in infants with a body weight less than 1,000 g.

Fetal accretion rates of calcium and phosphorus reach their peak during the third trimester, with upward of about 80% of fetal skeletal mineralization taking place during this time period. It has been reported that the clinical onset of osteopenia of prematurity generally occurs in the 6th to 12th week postnatally. The diagnosis of rickets usually occurs between 2 and 4 months chronologic age (Greer, 1994; Groh-Wargo et al., 2000).

#### Osteopenia of Prematurity

Risk factors for osteopenia of prematurity:

- < 30 weeks gestation
- < 1,000 g body weight
- Delayed establishment of full enteral feeds and/or prolonged PN
- Enteral feeds with low mineral content or bioavailability (unfortified breast milk, standard term formula, soy formula)
- Chronic use of medications that increase mineral excretion
- Cholestatic jaundice

Etiology of osteopenia of prematurity:

- Increased mineral needs for growth and bone accretion
- Decreased calcium and phosphorus intake:
  - Parenteral nutrition
  - Fluid restriction
- Enteral feedings: Unsupplemented formula or unfortified human milk
- Increased calcium and phosphorus losses:
  - Diuretics (furosemide and spironolactone [Aldactone]) ➔ hypercalcuria
  - Sodium bicarbonate ➔ increases mineral excretion
- Corticosteroids ➔ increase of bone resorption and a decrease of bone formation, increase of urinary calcium/ creatinine, and decrease in serum phosphorus
- Decreased calcium and phosphorus reserves:
  - Prematurity
  - Placental insufficiency (severe preeclampsia)
- Other factors:
  - Aluminum contaminants (divalent cations) in PN components
  - Deposits in bone, displacing calcium and phosphorus
- Vitamin D deficiency:
  - Inadequate vitamin D intake (rare)
  - Liver/renal disease (affects vitamin D metabolism)

High 1,25-$[OH]_2$ vitamin D values are often associated with low serum phosphate levels, thus confirming phosphate insufficiency as a primary factor in the pathogenesis of osteopenia of prematurity. Vitamin D deficiency is quite rare in premature

infants; exceptions to this include patients with liver disease, renal disease, and malabsorption.

Ideally, the goal of therapeutic interventions for osteopenia of prematurity is to achieve the intrauterine rate of bone mineralization. However, this would require enteral intakes of about 200 mg calcium/kg/day and about 90 mg phosphorus/kg/day, assuming 65% absorption of calcium (at best) and 80% absorption of phosphorus. In some instances, the most realistic goal is to prevent severe osteopenia with fractures and rickets. In patients receiving PN support, maximal retention can be accomplished when providing between 1.3:1 and 1.7:1 Ca/P by weight, or 1.1 to 1.3:1 by molar ratio. Ca/P ratios less than 1:1 by weight (0.8:1 by molar ratio) and alternating daily infusions of calcium and phosphorus are not recommended (Klein, 1998; Prestridge, Schanler, Shulman, Burns, & Laine, 1993).

### Emerging Issues

It has come to light that various products used during PN compounding have a high aluminum content, which can be especially dangerous for infants and children. Preterm infants are extremely vulnerable to aluminum toxicity because of immature renal function and the likelihood for long-term PN (Mirtallo et al., 2004). The FDA mandated that by July 2004 manufacturers of products used in compounding PN measure the aluminum content of their products and disclose it on the label. The FDA identified 5 µg/kg/day as the maximum amount of aluminum that can be safely tolerated, and amounts exceeding this limit may be associated with central nervous system or bone toxicity (Miratallo et al., 2004). It is essential for pharmacists, dietitians, physicians, and nurses to collaborate to reduce the use of higher-aluminum-content products. However, it is difficult to achieve the recommended aluminum intake level set by the FDA when patients are receiving multiple medications and PN. A reasonable goal for clinicians is to minimize aluminum exposure.

## Additional Neonatal Diagnoses and Nutritional Issues

Bronchopulmonary dysplasia is chronic lung disease that primarily affects premature infants with severe respiratory distress syndrome and is most commonly caused by intermittent positive pressure ventilation and oxygen therapy in the neonatal period. The lower the body weight and gestational age of the infant, the higher the incidence and severity of bronchopulmonary dysplasia. The etiology of growth impairment and compromised neurologic status in infants with bronchopulmonary dysplasia is multifactorial. Studies have shown reduced growth rates for weight, length, and head circumference during the first 2 years of life for patients with bronchopulmonary dysplasia (Davidson et al., 1990; Johnson, Cheney, & Monsen, 1998; Marks et al., 2006). Factors that contribute to this inadequate growth include elevated energy expenditure, fluid restrictions limiting nutrition intake, recurrent illnesses with periods of caloric and nutrient deficits, and chronic steroid use.

## Summary

Advanced technologic and medical interventions enable clinicians to support infants born prematurely. It is a challenge to provide adequate nutrition to promote growth similar to fetal growth velocity and to support bone mineralization similar to fetal accretion rates. Neonatologists, registered dietitians, and neonatal clinical teams strive to provide optimal nutrition support to premature infants; however, clinicians sometimes cannot provide optimal nutrition support because of barriers encountered throughout an infant's admission to the NICU. This special section defined prematurity, identified the potential adverse impact prematurity can have on oral feeding, described appropriate goals for growth, discussed feeding options, and explained appropriate uses of PN.

## References


Abad-Sinden, A., Verbrugge, K. C., & Buck, M. L. (2001). Assessment, prevention and management of metabolic bone disease in very low birthweight infants: The role of the neonatal nutritionist. *Nutrition in Clinical Practice*, *16*, 13–19.

Agosti, M., Vegni, C., Calciolari, G., Marini, A., & the Investigators of the "GAMMA" Study Group. (2003). Post-discharge nutrition of the VLBW infant: Interim results of the multicentric GAMMA study. *Acta Paediatrica*, *41*(Suppl.), 39–43.

American Academy of Pediatrics and American College of Obstetrics and Gynecology. (2002). *Guidelines for perinatal care* (5th ed.). Elk Grove Village, IL: American Academy of Pediatrics.

American Academy of Pediatrics Committee on Nutrition. (1983). Commentary on parenteral nutrition. *Pediatrics*, *71*, 547.

American Academy of Pediatrics Committee on Nutrition. (1998). Soy protein–based formulas: Recommendations for use in infant feeding. *Pediatrics*, *101*, 148–153.

Anderson, D. (1996). Nutrition in the care of the low birth weight infant. In L. K. Mahan & S. Escott-Stump (Eds.), *Krause's food, nutrition, and diet therapy* (9th ed.). Philadelphia, PA: Saunders.

Anderson, D., & Pittard, W. B. (1997). Parenteral nutrition for neonates. In R. Baker, S. Baker, & A. Davis (Eds.), *Pediatric parenteral nutrition*. New York, NY: International Thomson Publishing.

Anderson, D. M., & Kliegman, R. M. (1991). The relationship of neonatal alimentation practices to the occurrence of endemic necrotizing enterocolitis. *American Journal of Perinatology*, *8*, 62–67.

Andorsky, D. J., Lund, D. P., Lillehei, C. W., Jaksic, T., Dicanzio, J., Richardson, D. S., … Duggan, C. (2001). Nutritional and other postoperative management of neonates with short bowel syndrome correlates with clinical outcomes. *Journal of Pediatrics*, *139*, 27–33.

Babson, S. G., & Benda, G. I. (1976). Growth graphs for the clinical assessment of infants of varying gestational age. *Journal of Pediatrics*, *89*, 814–820.

Beath, S. V., Davies, P., Papadopoulou, A., Khan, A. R., Buick, R. G., Corkery, J. J., … Booth, I. W. (1996). Parenteral nutrition-related cholestasis in postsurgical neonates: Multivariate analysis of risk factors. *Journal of Pediatric Surgery*, *31*, 604–606.

Bernbaum, J. C. (2000). *Preterm infants in primary care: A guide to office management*. Columbus, OH: Ross Products Division, Abbott Laboratories.

Bernstein, S., Heimler, R., & Sasidharan, P. (1998). Approaching the management of the neonatal intensive care unit graduate through history and physical assessment. *Pediatric Clinics of North America*, *45*, 79.

Bhatia, J. (2005). Post-discharge nutrition of preterm infants. *Journal of Perinatology*, *25*, S15–S16.

Bines, J., Francis, D., & Hill, D. (1998). Reducing parenteral requirement in children with short bowel syndrome: Impact of an amino acid–based complete formula. *Journal of Pediatric Gastroenterology and Nutrition*, *26*, 123–128.

Cairns, P. A., & Stalker, D. J. (2000). Carnitine supplementation of parenterally fed neonates. *Cochrane Database of Systematic Reviews*, (4), CD000950.

Callenbach, J. C., Sheehan, M. B., Abramson, S. J., & Hall, R. T. (1981). Etiologic factors in rickets in VLBW infants. *Journal of Pediatrics*, *98*, 800–805.

Caple, J., Armentrout, D., & Huseby, V. (2004). Randomized, controlled trial of slow versus rapid feeding volume advancement in preterm infants. *Pediatrics*, *114*(6), 1597–1600.

Carver, J. D., Wu, P. K., & Hall, R. T. (2001). Growth of preterm infants fed nutrient-enriched or term formula after hospital discharge. *Pediatrics*, *107*, 683–689.

Catache, M., & Leone, C. R. (2003). Role of plasma and urinary calcium and phosphorus measurements in early detection of phosphorus deficiency in very low birthweight infants. *Acta Paediatrica*, *92*, 76–80.

Chan, K., Ohlsson, A., Synnes, A., Lee, D. S., & Chein, L. Y. (2001). Survival, morbidity, and resource use of infants of 25 weeks gestation or less. *American Journal of Obstetrics and Gynecology*, *185*, 220–226.

Cobb, B. A., Carlo, W. A., & Ambalavanan, N. (2004). Gastric residuals and their relationship to necrotizing enterocolitis in very low birth weight infants. *Pediatrics*, *113*, 50–53.

Crill, C. M., Christensen, M. L., Storm, M. C., & Helms, R. A. (2006). Relative bioavailability of carnitine supplementation in premature neonates. *Journal of Parenteral and Enteral Nutrition*, *30*, 421–425.

Davidson, S., Schrayer, A., Wielunsky, E., Krikler, R., Lilos, P., & Reisner, S. H. (1990). Energy intake, growth, and development in ventilated very-low-birth-weight infants with and without bronchopulmonary dysplasia. *American Journal of Diseases of Children*, *144*, 553–559.

Dhillon, A. S., & Ewer, A. K. (2004). Diagnosis and management of gastro-oesophageal reflux in pre-term infants in neonatal intensive care units. *Acta Paediatrica*, *93*, 88–93.

Dollberg, S., Kuint, J., Mazkereth, R., & Mimouni, F. B. (2000). Feeding tolerance in preterm infants: Randomized trial of bolus and continuous feeding. *Journal of the American College of Nutrition*, *19*, 797–800.

Faerk, J., Peitersen, B., Petersen, S., & Michaelsen, K. F. (2002). Bone mineralisation in premature infants cannot be predicted from serum alkaline phosphatase or serum phosphate. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, *87*, F133–F136.

Fenton, T. R. (2003). A new growth chart for preterm babies: Babson and Benda's chart updated with recent data and a new format. *BMC Pediatrics*, *16*, 13.

Fok, T. F., Chui, K. K. M., Cheung, R., Ng, P. C., Cheung, K. L., & Hjelm, M. (2001). Manganese intake and cholestatic jaundice in neonates receiving parenteral nutrition: A randomized controlled study. *Acta Paediatrica*, *90*, 1009–1015.

Foman, S. J. (1993). *Nutrition of normal infants*. St. Louis, MO: Mosby.

Francavilla, R., Miniello, V. L., Lionetti, M. E., & Armenio, L. (2003). Hepatitis and cholestasis in infancy: Clinical and nutritional aspects. *Acta Paediatrica*, *441*(Suppl.), 101–104.

Franz, A. R., Mihatsch, W. A., Sander, S., Kron, M., & Pohlandt, F. (2000). Prospective randomized trial of early versus late enteral iron supplementation in infants with a birth weight of less than 1301 grams. *Pediatrics*, *106*, 700–706.

Gairdner, D., & Pearson, J. (1988). *Growth and development record: Preterm–2 years, length/weight/head circumference*. Welwyn Garden City, Hertfordshire, England: Castlemead Press.

Geggie, J. H., Dressler-Mund, D. L., Creighton, D., & Cormack-Wong, E. R. (1999). An interdisciplinary feeding team approach for preterm, high-risk infants and children. *Canadian Journal of Dietetic Practice and Research*, *60*, 72–77.

Giuo, S., Roche, A. F., & Moore, W. M. (1988). Reference data for head circumference and 1-month increments from 1 to 12 months of age. *Journal of Pediatrics*, *113*, 490.

Gray, K., & King, W. (1997). Feeding assessment. In J. Cox (Ed.), *Nutrition manual for at-risk infants and toddlers*. Chicago, IL: Precept Press.

Greene, H. L., Hambidge, K. M., Schanler, R., & Tsang, R. C. (1988). Guidelines for the use of vitamins, trace elements, calcium, magnesium, and phosphorus in infants

and children receiving total parenteral nutrition: Report of the Subcommittee on Pediatric Parenteral Nutrition Requirements from the Committee on Clinical Practice Issues of the American Society for Clinical Nutrition. *American Journal of Clinical Nutrition*, *48*, 1324–1342.

Greer, F. R. (1994). Osteopenia of prematurity. *Annual Reviews in Nutrition*, *14*, 169–185.

Griffin, I. J. (2002). Postdischarge nutrition for high risk neonates. *Clinics in Perinatology*, *9*, 327–344.

Groh-Wargo, S., & Cox, J. H. (1997). Prematurely born infants. In J. Cox (Ed.), *Nutrition manual for at-risk infants and toddlers*. Chicago, IL: Precept Press.

Groh-Wargo, S., Thompson, M., & Cox, J. (2000). *Nutritional care for high-risk newborns* (Rev. 3rd ed.). Chicago, IL: Precept Press.

Hatun, S., Ozkan, B., Orbak, Z., Doneray, H., Cizmecioglu, F., Toprak, D., & Calikoglu, A. S. (2005). Vitamin D deficiency in early infancy. *Journal of Nutrition*, *135*, 279–282.

Hawdon, J. M., Beauregard, N., Slattery, J., & Kennedy, G. (2000). Identification of neonates at risk of developing feeding problems in infancy. *Developmental Medicine and Child Neurology*, *42*, 235–239.

Heird, W. C., Dell, R. B., & Helms, R. A. (1987). Amino acid mixture designed to maintain normal plasma amino acid patterns in infants and children requiring parenteral nutrition. *Pediatrics*, *80*, 401–408.

Henderson, G., Fahey, T., & McGuire, W. (2005). Calorie and protein-enriched formula versus standard term formula for improving growth and development in pre-term or low birth weight infants following hospital discharge. *Cochrane Database of Systematic Reviews*, (18), CD004696.

Hurwitz, M., Garcia, M. G., Poole, R. L., & Kerner, J. (2004). Copper deficiency during parenteral nutrition: A report of four pediatric cases. *Nutrition in Clinical Practice*, *19*, 305–308.

Ibrahim, H. M., Jeroudi, M. A., Baier, R. J., Dhanireddy, R., & Krouskop, R. W. (2004). Aggressive early total parenteral nutrition in the low-birth-weight infants. *Journal of Perinatology*, *24*, 482–486.

Johnson, D. B., Cheney, C., & Monsen, E. R. (1998). Nutrition and feeding in infants with bronchopulmonary dysplasia after initial hospital discharge: Risk factors for growth failure. *Journal of the American Dietetic Association*, *98*, 649–656.

Karpen, S. (2002). Update on the etiologies and management of neonatal cholestasis. *Clinics in Perinatology*, *29*, 159–180.

Kashyap, S., Schulze, K. F., & Forsyth, M. (1990). Growth, nutrient retention, and metabolic response of low-birth-weight infants fed supplemented and unsupplemented preterm human milk. *American Journal of Clinical Nutrition*, *52*, 254–262.

Kerner, J. A. (1996). Parenteral nutrition. In W. A. Walker, P. R. Durie, J. R. Hamilton, J. A. Walker-Smith, & J. B. Watkins (Eds.), *Pediatric gastrointestinal disease: Pathophysiology, diagnosis, management* (Vol. 2). St. Louis, MO: Mosby.

Klein, G. L. (1998). Metabolic bone disease of total parenteral nutrition. *Nutrition*, *14*, 149–152.

Knafelz, D., Gambarara, M., Diamanti, A., Papadatou, B., Ferretti, F., Tarissi De Iacobis, I., & Castro, M. (2003). Complications of home parenteral nutrition in a large pediatric series. *Transplantation Proceedings*, *35*, 3050–3051.

Kulkarni, P. B., Hall, R. T., & Rhodes, P. G. (1980). Rickets in VLBW infants. *Journal of Pediatrics*, *97*, 249–252.

Kurkchubasche, A. G., Rowe, M. I., & Smith, S. D. (1993). Adaptation in short-bowel syndrome: Reassessing old limits. *Journal of Pediatric Surgery*, *28*, 1069–1071.

Kurl, S., Heinonen, K., & Lansimies, E. (2000). Effects of prematurity, intrauterine growth status, and early dexamethasone treatment on postnatal bone mineralisation. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, *83*, F109–F111.

Kuschel, C. A., & Harding, J. E. (2004). Multicomponent fortified human milk for promoting growth in preterm infants. *Cochrane Database of Systematic Reviews*, (1), CD000343.

Ladhani, S., Srinivasan, L., Buchanan, C., & Allgrove, J. (2004). Presentation of vitamin D deficiency. *Archives of Disease in Childhood*, *89*, 781–784.

Landers, S. (2003). Maximizing the benefits of human milk feeding for the preterm infant. *Pediatric Annals*, *32*, 298–306.

Lucas, A., & Cole, T. J. (1990). Breast milk and neonatal necrotizing enterocolitis. *Lancet*, *336*, 1519–1523.

Lucas, A., King, F., Bishop, N. B., & King, F. J. (1992). Randomized trial of nutrition for pre-term infants after discharge. *Archives of Disease in Childhood*, *67*, 324.

Marino, A. J., Assing, E., Carbone, M. T., Hiatt, I. M., Hegyi, T., & Graff, M. (1995). The incidence of gastroesophageal reflux in pre-term infants. *Journal of Perinatology*, *15*, 369–371.

Marks, K. A., Reichman, B., Lusky, A., Zmora, E., & Israel Neonatal Network. (2006). Fetal growth and postnatal growth failure in very-low-birthweight infants. *Acta Paediatrica*, *95(2)*, 236–242.

McGuire, W., & Anthony, M. Y. (2003). Donor human milk versus formula for preventing necrotising enterocolitis in preterm infants: Systematic review. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, *88*, F11–F14.

McGuire, W., & McEwan, P. (2004). Systematic review of transpyloric versus gastric tube feeding for preterm infants. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, *89*, F245–F248.

McKeown, R. E., Marsh, T. D., & Amarnath, U. (1992). Role of delayed feeding and of feeding increments in necrotizing enterocolitis. *Journal of Pediatrics*, *121*(5 Pt. 1), 764–770.

Medline Plus Medical Dictionary. (2013a). Osteopenia. Retrieved from www.merriam-webster.com/medlineplus/osteopenia

Medline Plus Medical Dictionary. (2013b). Rickets. Retrieved from www.merriam-webster.com/medlineplus/rickets

Mirtallo, J., Canada, T., Johnson, D., Kumpf, V., Petersen, C., Sacks, G., ... Guenter, P. (2004). Safe practices for parenteral nutrition. *Journal of Parenteral and Enteral Nutrition*, *28*, S39–S70.

Nako, Y., Fukushima, N., & Tomomasa, T. (1993). Hypervitaminosis D after prolonged feeding with a premature formula. *Pediatrics*, *92*, 862.

Olsen, I. E., Richardson, D. K., Schmid, C. H., Ausman, L. M., & Dwyer, J. T. (2005). Dietitian involvement in the neonatal intensive care unit: More is better. *Journal of the American Dietetic Association*, *105*, 1224–1230.

Pereira, G. (1995). Nutritional care of the extremely premature infant. *Neonatal and Perinatal Nutrition*, *22*, 61–75.

Pettifor, J. M. (2004). Nutritional rickets: Deficiency of vitamin D, calcium, or both? *American Journal of Clinical Nutrition*, *80*(6 Suppl.), 1725S–1729S.

Pierro, A. (2002). Metabolism and nutritional support in the surgical neonate. *Journal of Pediatric Surgery*, *37*, 811–822.

Porcelli, P. J., Jr., & Sisk, P. M. (2002). Increased parenteral amino acid administration to extremely low-birth-weight infants during early postnatal life. *Journal of Pediatric Gastroenterology and Nutrition*, *34*, 174–179.

Premji, S., McNeil, D. A., & Scotland, J. (2004). Regional neonatal oral feeding protocol: Changing the ethos of feeding preterm infants. *Journal of Perinatal and Neonatal Nursing*, *18*, 371–384.

Prestridge, L. L., Schanler, R. J., Shulman, R. J., Burns, P. A., & Laine, L. L. (1993). Effect of parenteral calcium and phosphorus therapy on mineral retention and bone mineral content in the very low birth weight infants. *Journal of Pediatrics*, *122*(Pt. 1), 761–768.

Putet, G. (2000). Lipid metabolism of the micropremie. *Clinics in Perinatology*, *27*, 57–69.

Quiros-Tejeira, R. E., Ament, M. E., & Reyen, L. (2004). Long-term parenteral nutritional support and intestinal adaptation in children with short bowel syndrome: A 25-year experience. *Journal of Pediatrics*, *145*, 157–163.

Rao, R., & Georgieff, M. (2005). Microminerals. In R. Tsang, A. Luca, R. Uauy, & S. Zlotkin (Eds.), *Nutritional needs of the preterm infant: Scientific basis and practical guidelines* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Rayyis, S. F., Ambalavanan, N., Wright, L., & Carlo, W. (1999). Randomized trial of "slow" versus "fast" feed advancements on the incidence of necrotizing enterocolitis in VLBW infants. *Journal of Pediatrics*, *134*, 293–297.

Rigo, J., De Curtis, M., Pieltain, C., Picaud, J. C., Salle, B. L., & Senterre, J. (2000). Bone mineral metabolism in the micropremie. *Clinics in Perinatology*, *27*, 147–170.

Ritchie, S. (2002). Primary care of the premature infant discharged from the neonatal intensive care unit. *American Journal of Maternal Child Nursing*, *27*, 76–85.

Ritz, E., Haxsen, V., & Zeier, M. (2003). Disorders of phosphate metabolism—pathomechanisms and management of hypophosphataemic disorders. *Best Practice and Research: Clinical Endocrinology and Metabolism*, *17*, 547–558.

Roche, A., & Himes, J. H. (1982). Incremental growth charts. *American Journal of Clinical Nutrition*, *35*, 629.

Rommel, N., DeMeyer, A., Feenstra, L., & Veereman-Wauters, G. (2003). The complexity of feeding problems in 700 infants and young children presenting to a tertiary care institution. *Journal of Pediatric Gastroenterology and Nutrition*, *37*, 75–84.

Schanler, R. J. (2001). The use of human milk for premature infants. *Pediatric Clinics of North America*, *48*, 207–219.

Schanler, R. J., Shulman, R. J., Lau, C., O'Brian Smith, E., & Heitkemper, M. M. (1999). Feeding strategies for premature infants: Randomized trial of gastrointestinal priming and tube-feeding method. *Pediatrics*, *103*, 434–439.

Schmidt, G. L., Baumgartner, T. G., & Fischlschweiger, W. (1986). Cost containment using cysteine HCl acidification to increase calcium/phosphate solubility in hyperalimentation solutions. *Journal of Parenteral and Enteral Nutrition*, *10*, 203–207.

Schmidt-Sommerfield, E., Penn, D., & Wolf, H. (1982). Carnitine blood concentrations and fat utilization in parenterally alimented premature newborn infants. *Journal of Pediatrics*, *100*, 260.

Shenai, J. P., Jhaveri, B. M., & Reynolds, J. W. (1981). Nutritional balance studies in VLBW infants: Role of soy formula. *Pediatrics*, *67*, 631–637.

Silvestre, M. A., Morbach, C. A., Brans, Y. W., & Shankaran, S. (1996). A prospective randomized trial comparing continuous versus intermittent feeding methods in very low birth weight neonates. *Journal of Pediatrics*, *128*, 748.

Sondheimer, J. M. (2004). A critical perspective on trophic feeding. *Journal of Pediatric Gastroenterology and Nutrition*, *38*, 237–238.

Sondheimer, J. M., Asturias, E., & Cadnapaphornchai, M. (1998). Infection and cholestasis in neonates with intestinal resection and long term parenteral nutrition. *Journal of Pediatric Gastroenterology and Nutrition*, *27*, 131–137.

Steward, D. K., & Pridham, K. F. (2002). Nutritional influences on the growth of extremely LBW infants. *Newborn and Infant Nursing Reviews*, *2*, 159–165.

Thureen, P. J., & Hay, W. W. (2001). Early aggressive nutrition in preterm infants. *Seminars in Neonatology*, *6*, 403–415.

Tyson, J. E., & Kennedy, K. A. (2005). Trophic feedings for parenterally fed infants. *Cochrane Database of Systematic Reviews*, *20*(3), CD000504.

U.S. Food and Drug Administration. (2002). Health professionals letter on *Enterobacter sakazakii* infections associated with use of powdered (dry) infant formulas in neonatal intensive care units. Retrieved from www.fda.gov/Food/RecallsOutbreaksEmergencies/SafetyAlertsAdvisories/ucm111299.htm

Vanderhoof, J. (2004). New and emerging therapies for short bowel syndrome in children. *Journal of Pediatric Gastroenterology and Nutrition*, *39*, S769–S771.

Vanderhoof, J. A. (2003). Enteral and parenteral nutrition in the care of patients with short-bowel syndrome. *Best Practice & Research Clinical Gastroenterology*, *17*, 997–1015.

Weckwerth, J. A. (2004). Monitoring enteral nutrition support tolerance in infants and children. *Journal of Parenteral and Enteral Nutrition*, *19*, 496–503.

Weisberg, P., Scanlon, K. S., Li, R., & Cogswell, M. E. (2004). Nutritional rickets among children in the United States: Review of cases reported between 1986 and 2003. *American Journal of Clinical Nutrition*, *80*(6 Suppl.), 1697S–705S.

Wheeler, R. E., & Hall, R. T. (1996). Feeding of premature infant formula after hospital discharge of infants weighing less than 1,800 grams at birth. *Journal of Perinatology*, *16*, 111.

Wight, N. E. (2001). Donor human milk for preterm infants. *Journal of Perinatology*, *21*, 249–254.

Wright, K., Ernst, K. D., Gaylord, M. S., Dawson, J. P., & Burnette, T. M. (2003). Increased incidence of parenteral nutrition-associated cholestasis with Aminosyn PF compared to TrophAmine. *Journal of Perinatology*, *23*, 444–450.

Yu, V. Y. (2005). Extrauterine growth restriction in preterm infants: Importance of optimizing nutrition in neonatal intensive care units. *Croatian Medical Journal*, *46*, 737–743.

Ziegler, E. E., Thureen, P. J., & Carlson, S. J. (2002). Aggressive nutrition of the VLBW infant. *Clinics in Perinatology*, *29*, 225–244.

CHAPTER 6

# Special Topics in Toddler and Preschool Nutrition: Vitamins and Minerals in Childhood and Children with Disabilities

*Aaron Owens, MS, RD,*
*Harriet H. Cloud, MS, RD,* FADA, and
*Sharon Collier, RD, LD, M Ed*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Recognize the purposes of vitamin and mineral functions in growth and development.
2. Review the definitions of developmental disabilities.
3. Identify the etiology and prevalence of various developmental disabilities.
4. Review the evidence-based practice for nutrition problems associated with selected syndromes and disabilities.
5. Compare legislative and community resources for children with developmental disabilities.

# *Vitamins and Minerals in Childhood*

*Aaron Owens, MS, RD, and Sharon Collier, MEd, RD, LDN*

Vitamins and minerals are nutrients that are essential to life because of their involvement in cellular metabolism, maintenance, and growth throughout the life cycle. Vitamins and minerals are often called micronutrients because in comparison with the four major nutrients—carbohydrate, protein, fat, and water—they are needed in relatively small amounts. Changing lifestyles and dietary patterns over the last two decades have conspired to increase a number of micronutrient imbalances in diets of children. Poor dietary patterns, such as skipping meals, a preference for low-nutrient-dense foods in place of nourishing ones, and frequent snacking, can contribute to suboptimal nutrition status during childhood (Kim, Kim, & Keen, 2005). The Commission on the Nutrition Challenges of the 21st Century (2000) stated that although protein-energy malnutrition remains a concern for many of the world's children, micronutrient malnutrition is recognized increasingly as a more widespread problem (Tanner & Finn-Stevenson, 2002).

In the United States, the Feeding Infants and Toddlers Study (FITS 2008) demonstrated that iron and zinc intake was of concern for the older infant population, whereas in the toddler group vitamin E, potassium, and fiber were of concern for adequacy. For this age group folate, preformed vitamin A, zinc, and sodium were in excess as compared to the Dietary Reference Intake (DRIs) (Dwyer Butte, Deming, Siega-Riz, & Reidy, 2010).

Malnutrition permeates all aspects of health, growth, cognition, motor, and social development. Micronutrient deficiencies, such as iron deficiency anemia, have been shown to positively correlate with decreased school performance. Lack of adequate micronutrients, especially zinc, selenium, iron, and the antioxidant vitamins, can also lead to clinically significant immune deficiency and infections in children. More than 50% of deaths of children in developing countries are a result of infections related to malnutrition. Irreversible and lifelong sequelae may prevent children from reaching their fullest potential (Neumann, Gewa, & Bwibo, 2004). Therefore, it is important that healthy dietary patterns are established during childhood.

## Vitamins

**Vitamins** are organic compounds that occur naturally in plants and animals and are distinct from carbohydrates, fats, and proteins. Vitamins are also typically found naturally in foods in minute amounts and are not synthesized by the body in adequate amounts to meet physiologic needs. Vitamin needs are often dependent on energy intake or other nutrient levels. Therefore, inadequate consumption of vitamins results in a specific deficiency syndrome. Vitamin functions are determined by their chemical and associated physical properties and serve essential roles in numerous metabolic processes. Few, however, show close chemical or functional similarities. By and large, vitamins function as coenzymes, which are fundamental parts of enzymes.

**vitamins** Organic compounds that occur naturally in plants and animals and are not synthesized by the body in adequate amounts to meet physiologic needs.

Vitamins are usually classified into two groups, based on their solubility. Some are soluble

in nonpolar solvents (i.e., vitamins A, D, E, and K), whereas others are soluble in polar solvents (i.e., vitamin C, thiamine, riboflavin, niacin, vitamin $B_6$, biotin, pantothenic acid, folate, and vitamin $B_{12}$). The **fat-soluble vitamins** tend to be absorbed and transported with dietary lipids, whereas the **water-soluble vitamins** are absorbed by passive and active processes. Vitamins remain in the body for varying amounts of time. Utilization of water-soluble vitamins begins the minute they are absorbed through the digestive system and remain in the body for 2 to 4 days. Water-soluble vitamins are not stored, with the exception of vitamin $B_{12}$, and are quickly excreted in the urine either intact or as water-soluble metabolites. Thus, water-soluble vitamins must be replenished regularly, and toxicities are virtually unknown. Fat-soluble vitamins stay in the body for longer periods of time and are usually stored in lipid tissue of organs, especially the liver. In general, the fat-soluble vitamins are excreted with the feces via enterohepatic circulation. Toxicity is probable with some but only when taken in very large doses (Mahan & Escott-Stump, 2008).

**fat-soluble vitamins** Including vitamins A, D, E, and K; soluble in polar solvents, absorbed and transported with dietary lipids, and excreted in the feces.

**water-soluble vitamins** Including vitamin C, thiamine, riboflavin, niacin, vitamin $B_6$, biotin, pantothenic acid, folate, and vitamin $B_{12}$; soluble in nonpolar solvents, absorbed by passive and active processes, and excreted in the urine.

## Water-Soluble Vitamins

### Vitamin B

Although each B vitamin has its own unique biological role and individual properties, as a group these nutrients are often thought of as a single entity because of their commonality. B vitamins work together in the body by maintaining healthy nerves, skin, hair, eyes, liver, and mouth and by preserving good muscle tone in the gastrointestinal tract. B vitamins are used as coenzymes in almost all parts of the body and also provide energy because they are necessary for metabolism of carbohydrate, fat, and protein. Emotional stress, surgery, illness, pregnancy, and breastfeeding all increase B vitamin requirements. Deficiency in a single B vitamin is rare; rather, people tend to have multiple deficiencies, making subtle deficiency symptoms more difficult to diagnose yet more likely to be present. Because of competition for absorption in the intestines, high doses of a single B vitamin should be avoided. When considering supplementation, the ratio of B vitamins is most beneficial when 1:1. In general, no toxicity of B vitamins has been reported.

### Vitamin $B_1$ (Thiamine)

Thiamine was the first B vitamin to be discovered in the 1920s, and it plays an essential role in metabolism of carbohydrates, a major source of energy in cells. As daily intake of complex and simple carbohydrates increases, the need for thiamine increases because extra carbohydrates use more thiamine. The cells of the brain and central nervous system are the first to show signs of thiamine deficiency because of extreme sensitivity to carbohydrate metabolism. During periods of physical and emotional stress, including times of fever, muscular activity, overactive thyroid, pregnancy, and lactation, thiamine requirements increase. Thiamine is also involved in converting fatty acids into steroid hormones, such as cortisone and progesterone, necessary for proper growth and maintenance of healthy skin. Thiamine may play a role in resistance to disease (Libermann & Branning, 2003).

Classic thiamine deficiency disease is **beriberi**, a disease that affects the gastrointestinal tract, cardiovascular system, and peripheral nervous system and is usually confined to alcoholics. Advanced symptoms of deficiency are indigestion, constipation, headaches, insomnia, and heavy weak legs, which is then followed by cramping or numb feet. Thiamine deficiency can also produce ventricular dilatation and dysfunction resulting in a damaged, enlarged, or irregularly beating heart (Prabhu & Dalvi, 2000). Subclinical deficiency symptoms are fatigue, apathy, mental confusion, inability to concentrate, poor memory, insomnia, anorexia, weight loss, decreased strength, emotional instability, depression, and irrational fears. Such symptoms have been reported in children with B-cell leukemia lymphoma being treated with chemotherapy and total parenteral nutrition. In this group, profound lethargy developed because of severe lactic acidosis caused by an impairment of pyruvate dehydrogenase complex. This was a result of a lack of its necessary cofactor, thiamine, in the total parenteral nutrition. Treatment with added thiamine resulted in an improved clinical and laboratory status (Svahn et al., 2003).

**beriberi** Classic thiamine deficiency disease that affects the gastrointestinal tract, cardiovascular system, and peripheral nervous system and is usually confined to alcoholics.

Mutations in pyruvate dehydrogenase have been associated with subacute necrotizing encephalopmyelopathy also known as Leigh's disease (Barnerias et al., 2010). This is a rare neurodegenerative disorder that occurs in early childhood and is characterized by focal necrotic brain lesions. Eventually this leads to death, typically before 5 years of age. Providing thiamine may help delay the mortality outcome but unfortunately will not prevent it.

Significant food sources include organ meats, pork, dried beans, peas, soybeans, peanuts, whole grains, wheat germ, rice bran, egg yolk, poultry, and fish. Foods with antithiamine activity include blueberries, red chicory, black currants, Brussels sprouts, red cabbage, and raw seafood. Ascorbic acid has been shown to protect against thiamine destruction in some of these foods. Thiamine is heat labile; therefore, pasteurization and canning in food processing may cause significant loss. This is why cereals, breads, and infant formulas are enriched with thiamine and other B vitamins (Erdman, Macdonald, & Zeisel, 2012).

#### Vitamin $B_2$ (Riboflavin)

Riboflavin plays an important role in the oxidation of amino acids, synthesis of fatty acids, and oxidation of glucose and thyroid hormone metabolism, processes that influence metabolism and energy production. An increased need for riboflavin is associated with tissue damage, including from burns, surgery, injuries, fever, and malignancies. The blood, too, requires riboflavin, a deficiency of which can lead to vitamin $B_2$ anemia. There is reasonably good evidence that poor riboflavin status interferes with iron handling and contributes to the etiology of anemia when iron intakes are low. Correcting a riboflavin deficiency in children improves the hematologic response of iron supplements. Riboflavin is also found in the pigment of the retina, enabling eyes to adapt to light, with photophobia resulting in times of vitamin $B_2$ deficiency. Current interest is focused on the role that riboflavin plays in determining circulating concentrations of homocysteine, a risk factor for cardiovascular disease.

Biochemical signs of depletion arise within only a few days of dietary deprivation. Riboflavin deficiency among Western children seems to be largely confined to adolescents, primarily girls, despite the diversity of riboflavin-rich foods available. Classic deficiency symptoms are cheilosis (tiny lesions and cracks in the corners of the mouth); glossitis, an inflamed purple tongue; tearing, burning, and itching of the eyes; seborrheic dermatitis, flaking of the skin; normochromic, normocytic anemia, and behavior changes. Riboflavin deficiency may exert some of its effects by reducing the metabolism of other B vitamins, notably folate and vitamin $B_6$. Some children may be marginally deficient as a result of taking antibiotics because antibiotics interfere with absorption and utilization of riboflavin. Phototherapy used to treat hyperbilirubinemia in neonates is also associated with transient deterioration in riboflavin status, although no functional deficits have been described; however, supplements are warranted. Riboflavin deficiency also has been implicated as a risk factor for cancer, although this has not been satisfactorily established.

Riboflavin is unique among the water-soluble vitamins in that milk and dairy products make the greatest contribution to its intake in Western diets. Meat and fish are also good sources of riboflavin, and certain fruits and vegetables, especially dark-green leafy vegetables, contain reasonably high concentrations. Breast milk concentrations of riboflavin are fairly sensitive to maternal riboflavin intake and can be moderately increased by riboflavin supplementation of the mother when natural intake is low. Even in well-nourished communities, concentrations of riboflavin in breast milk are considerably lower than in cow's milk. Infants receiving donor breast milk may be at risk of developing transient riboflavin deficiency because of losses in the milk during collection, storage, and administration.

Intakes of riboflavin in excess of tissue requirements are excreted in the urine as riboflavin or other metabolites. Some urinary metabolites reflect bacterial activity in the gastrointestinal tract as well. Urinary excretion, however, is not a sensitive marker of very low riboflavin intakes (Powers, 2003).

#### Vitamin $B_3$ (Niacin)

There are two forms of niacin, nicotinic acid and niacinamide, which our bodies make from tryptophan. Nicotinic acid and niacinamide have identical vitamin activities but have very different pharmacologic activities. For example, nicotinic acid lowers cholesterol, but niacinamide does not. Niacin is a coenzyme in many important biochemical reactions involved in maintaining healthy skin, properly functioning gastrointestinal tract and central nervous

system, and metabolism of lipids (Hendler & Rorvik, 2008). Because of niacin's role in the metabolism of fats, much of its use involves the treatment of elevated cholesterol. There is strong evidence that the onset of atherosclerosis occurs in childhood. Identifying and treating children and adolescents at risk for hypercholesterolemia should lead to a decrease in adult atherosclerotic disease. Studies show that niacin is effective in decreasing triglycerides (TG) in the blood. Niacin increases high-density lipoproteins (HDL) while decreasing very low density lipoproteins (VLDL) and TG. The mechanisms for correcting dyslipidemia are unknown and continue to be studied. Long-term studies, however, are needed to determine whether delivery of niacin truly provides long-term benefits for those children with hypercholesterolemia (Kronn, Sapru, & Satou, 2000).

Niacin deficiency can occur under certain conditions, which include malabsorption syndromes, cirrhosis, and the provision of total parenteral nutrition with inadequate niacin supplementation. The well-known disorder of niacin deficiency is **pellagra**. Pellagra is a nutritional wasting disease attributable to a combined deficiency of the essential amino acid tryptophan and niacin (nicotinic acid). It is characterized clinically by four classic symptoms, often referred to as the four Ds: dermatitis, diarrhea, dementia, and ultimately death if the diet is not improved. Before the development of these symptoms, other nonspecific symptoms develop gradually and mostly affect dermatologic, neuropsychological, and gastrointestinal systems. Easily observable deficiency generally occurs only in severely malnourished people. A review of literature reveals several case reports describing pellagra in patients with anorexia nervosa, with the most common features being cutaneous manifestations such as erythema on sun-exposed areas, glossitis, and stomatitis. Pellagra may be diagnosed if cutaneous symptoms resolve within 24 to 48 hours of oral niacin administration (Prousky, 2003). Although pellagra was commonly found in the United States through the 1930s, the disorder is rare today in industrialized countries. This is in large part to the result of the enrichment of refined flours with niacin (Hendler & Rorvik, 2008).

**pellagra** A nutritional wasting disease attributable to a combined deficiency of the essential amino acid tryptophan and niacin (nicotinic acid).

Niacin is found naturally in meat, poultry, fish, legumes, fortified cereals and grain, vegetables, and yeast. Consumption of corn-rich diets has resulted in niacin deficiency in certain populations. The reason for this is that niacin contained in corn exists in bound forms, which exhibits little or no nutritional availability. Soaking corn in a lime solution, which releases the niacin, has prevented such a deficiency in parts of the world where corn-based diets are prevalent. Tryptophan can be converted to nicotinic acid mononucleotide in the liver and kidneys but with 1/60th the efficiency of nicotinic acid, so 60 mg of tryptophan can be considered 1 niacin equivalent.

Flushing is the adverse reaction first observed after intake of a large dose of nicotinic acid because it causes vasodilatation of cutaneous blood vessels, which can cause an unpleasant sensation but is not associated with any known tissue injury. Other adverse reactions of nicotinic acid include dizziness, shortness of breath, sweating, nausea, vomiting, insomnia, abdominal pain, and tachycardia. Excessive intake of niacin can also exacerbate gastric ulcers. With liver disorders, high-dose therapeutic supplementation needs to be monitored because of potential increased liver function tests (Hendler & Rorvik, 2008).

### Vitamin $B_6$ (Pyridoxine)

Pyridoxine is one of the most essential and widely used vitamins in the body and participates as a coenzyme in more than 60 enzymatic reactions involved in the metabolism of amino acids and essential fatty acids. Vitamin $B_6$ is needed for proper growth and maintenance of almost all body structures and functions. One of the many systems dependent on vitamin $B_6$ is the nervous system. Pyridoxine is necessary for the production of serotonin and other neurotransmitters in the brain. Infants fed formulas low in vitamin $B_6$ have been reported to suffer from epileptic-like convulsions, weight loss, nervous irritability, and stomach disorders (Libermann & Branning, 2003). Pyridoxine-dependent epilepsy usually presents in the neonatal period or even in utero, is resistant to antiepileptic medications, and is treatable with lifelong administration of pyridoxine. The seizures are typically generalized as tonic-clonic, although myoclonic seizures or infantile spasms have been described (Yoshii, Takeoka, Kelly, & Krishnamoorthy, 2005).

Deficiency can cause microcytic anemia and depression of immune system responses. Symptoms of vitamin $B_6$ deficiency are dermatitis of the nose, eyes, and mouth; acne; cheilosis; stomatitis; and glossitis. Deficiency symptoms also include

alteration of the nervous system, depression, confusion, dizziness, insomnia, irritability, nervousness, and convulsions. Marginal status may increase the risk for chronic diseases including cardiovascular diseases and certain cancers (Erdman et al., 2012).

All foods contain small amounts of vitamin $B_6$, but foods with the highest concentration are eggs, fish, spinach, carrots, peas, beef, pork, organ meats, chicken, brewer's yeast, walnuts, sunflower seeds, whole grains and wheat germ. The amount of vitamin $B_6$ in foods does not necessarily represent the amount that is bioavailable or active in the tissues after ingestion. Studies have shown that bioavailability is quite limited (Libermann & Branning, 2003). The overall bioavailability of vitamin $B_6$ in a mixed diet has been reported to be about 75% (Tan, Tamura, & Stokstad, 1981). It is one of the B vitamins that is not added to enriched flour and other grain products.

Vitamin $B_6$ is relatively nontoxic, but some problems with the nervous system have been reported when consumed in huge doses of 2,000 to 6,000 mg/day, with reversal of side effects when dosage is discontinued. Inconclusive studies have suggested that social isolation, verbal delays, and self-stimulating behaviors of autistic children have been shown to improve when given high doses of vitamin $B_6$ along with magnesium (Libermann & Branning, 2003; Samour & King, 2011). The use of megavitamin intervention began in the 1950s with the treatment of schizophrenic patients. A number of studies attempted to assess the effects of vitamin $B_6$ and magnesium (Yoshii et al., 2005). It is thought that total vitamin $B_6$ is abnormally high in children with autism as a result of impaired conversion of pyridoxine to pyridoxal-5-phosphate. This may explain the published benefits of high-dose vitamin $B_6$ supplementation (Adams, George, & Audhya, 2006). Because of the small number of studies, the methodologic quality of studies, and small sample sizes, no recommendation can be advanced regarding the use of high doses of vitamin $B_6$ as a treatment for autism.

#### Vitamin $B_{12}$ (Cobalamin)

Recent data indicate that cobalamin status undergoes marked changes during childhood, particularly during the first year. During the first year of life, vitamin $B_{12}$ uptake may be limited because of low vitamin $B_{12}$ content in breast milk and immature intrinsic factor system, but the estimated vitamin $B_{12}$ stores in the neonatal liver are assumed to be sufficient for normal growth. Serum cobalamin has been shown to be lower during the first 6 months of life, with total homocysteine and methylmalonic acid particularly high, suggesting impaired cobalamin function. Children have the ability to produce vitamin $B_{12}$ in the intestines, but it is not definite how much can be absorbed by the body. Older children with an omnivorous diet are thought to meet the daily dietary requirement for vitamin $B_{12}$.

Cobalamin is a coenzyme in a methyl transfer reaction that converts homocysteine to methionine, which is later converted to L-methylmalonyl-coenzyme A to succinyl-coenzyme A. This explains why increased total homocysteine and methylmalonic acid in the blood are measures of impaired cobalamin status, which may occur even in the presence of normal serum cobalamin concentrations. The reported occurrence of low serum cobalamin and increased methylmalonic acid in a significant portion of healthy infants, as reported by newborn screening tests, may be more prevalent than once recognized. The increased metabolite concentrations in infants may be a harmless phenomenon related to developmental, physiologic, or nutritional factors or may reflect the common occurrences of impaired cobalamin function in infants. Because impaired cobalamin function may have long-term effects related to psychomotor development, intervention studies with cobalamin supplementation in pregnancy and infancy may be warranted (Monsen, Refsum, Markestad, & Ueland, 2003).

Some causes of deficiency are various forms of intestinal malabsorption, inborn errors of cobalamin metabolism, exposure to nitrous oxide (resulting from inactivation of methionine synthase), infants born to vitamin $B_{12}$–deficient mothers, and children adhering to strict vegetarian diets (Kim et al., 2005; Sander et al., 2005). Vitamin $B_{12}$ is absorbed from the terminal ileum, which is the commonly affected segment of gut in Crohn's disease. Its absorption may be compromised in these children secondary to inflammatory lesions, ileal bacterial overgrowth, and surgical resection. Prolonged depletion of vitamin $B_{12}$ is one of the major causes of megaloblastic anemia and ultimately leads to neuropathy (Ahmed & Jenkins, 2004). An example of such a disorder characterized by megaloblastic anemia resulting from malabsorption of cobalamin is Imerslund-Grasbeck disease (Eitenschenck, Armari-Alla, Plantaz, Pagnier, & Ducros, 2005). Cobalamin deficiency is prevalent in vegetarian

teens and has been associated with increased risk of osteoporosis. In adolescents, signs of impaired cobalamin status, as judged by elevated concentrations of methylmalonic acid, have been associated with low bone mineral density. This is especially true in adolescents fed a macrobiotic diet during the first years of life, where cobalamin deficiency is more prominent (Dhonukshe-Rutten, van Dusseldorp, Schneede, de Groot, & van Staveren, 2005). Childhood pernicious anemia is exceedingly rare; etiology is varied and may result despite adequate dietary intake.

Deficiency in infancy presents as failure to thrive, developmental delay, progressive neurologic disorders, or hematologic changes. Symptoms may be evident as early as 3 to 4 months of age but are often nonspecific and difficult to diagnose. Because the age at onset and the duration of neurologic symptoms may contribute to the development of long-term symptoms, early diagnosis and treatment are important for vitamin $B_{12}$–deficient children (Dror & Allen, 2008; Whitehead, 2006).

The amount of vitamin $B_{12}$ in food sources is small, with good sources including beef, herring, mackerel, egg yolk, milk, cheese, clams, sardines, salmon, crab meat, and oysters. Vegetarian teens should be counseled on the need to identify and regularly consume dietary sources of vitamin $B_{12}$, especially breastfeeding teen girls. Infants of vegan mothers should also receive vitamin $B_{12}$ supplements starting at birth while breastfeeding if the mother's diet is not supplemented.

### Folic Acid (Folate)

One of the most important roles of folate is that it works closely with vitamin $B_{12}$ in metabolism of amino acids, synthesis of proteins, and production of genetic material such as RNA and DNA. Abnormal metabolism of homocysteine and methylmalonic acid, which is directly related to folate metabolism, has been associated with neurologic disorders, such as autism (Miller, 2003; Muntjewerff et al., 2003). The observed imbalance of folate metabolism in autistic children is complex and not easily explained by one single pathway or isolated genetic or nutritional deficiency (James et al., 2004).

**neural tube defects**
Defects in the structure of the embryo that give rise to the brain, spinal cord, and other parts of the central nervous system as a result of inadequate folic acid intake during pregnancy.

The greatest attention received by folic acid is secondary to its ability to prevent **neural tube defects**. The neural tube is the structure of the embryo that gives rise to the brain, spinal cord, and other parts of the central nervous system. The Centers for Disease Control and Prevention estimates the rates for neural tube defects for two of the most common forms in 2005 was 17.96 per 100,000 live births for spina bifida and 11.11 per 100,000 live births for anencephaly (Matthews, 2007). They are the most significant fetal anomalies leading to long-term morbidity. Evidence from a number of studies demonstrated that periconceptional use of vitamin supplements containing folic acid reduces the risk of neural tube defects by at least 60% (Castillo-Lancellotti et al., 2013). Although the mechanism of action of this nutrient in influencing the risk of neural tube defects is poorly understood, the evidence of the benefit of folic acid has led many health organizations to recommend periconceptional supplementation at a level of 400 mg/day. Because of the concern that public education campaigns alone cannot achieve optimal periconceptional intake of folic acid, food fortification has been implemented as a strategy to reach all women of childbearing age. The fortification of grain products with folic acid has been controversial because of the potential to mask vitamin $B_{12}$ deficiency. A study in Newfoundland implemented the food fortification strategy with a marked decrease (78%) in the rate of neural tube defects. No evidence of adverse effect of folic acid fortification on the detection of vitamin $B_{12}$ deficiency was identified (Liu et al., 2004).

Some studies have shown significantly reduced risk of some congenital heart defects with maternal supplementation early in pregnancy (Botto, Mulinare, & Erickson, 2003; Hobbs et al., 2009). Of note is the fact that the development of both the neural tube and cardiac tissue depends on neural crest cells that have a high demand for folate to support cellular differentiation, growth, and migration (Wallis et al., 2009).

Because of folic acid's vital role in healthy cell division, replication, and tissue growth, deficiency of this nutrient is associated with dysplasia, an abnormal growth of tissues that is considered precancerous. Acute lymphoblastic leukemia is the most common childhood cancer in developed countries. Little is known about its causes, although its early age at diagnosis has focused interest on maternal and perinatal factors. The second most common pediatric tumor is neuroblastoma, an embryonic tumor, which is also the most prevalent extracranial solid tumor in children.

Results of previous studies suggested a protective effect of maternal folate supplementation during pregnancy against acute lymphoblastic leukemia and neuroblastoma tumors. Other studies of genes and environment interaction in acute lymphoblastic leukemia and other cancers provided contradictory results, perhaps because of varying definitions of folate exposure (French et al., 2003; Milne et al., 2006). A recent review of childhood cancer trends indicates that since the United States began folic acid food fortification the incidence of Wilm's tumor and possibly primitive neuroectodermal tumors is decreased but no other cancers (Linabery, Johnson, & Ross, 2012).

Like vitamin $B_{12}$, folic acid is needed for formation of red blood cells. Without adequate amounts of folic acid, megaloblastic anemia results, characterized by oversized red blood cells. Symptoms of deficiency are irritability, weakness, sleep difficulty, and pallor. When testing for anemia it is imperative to use diagnostic tests to distinguish vitamin $B_{12}$ anemia from folic acid anemia. If folic acid is supplemented when a child is deficient in vitamin $B_{12}$, severe vitamin $B_{12}$ deficiency will develop because the body needs vitamin $B_{12}$ to use folic acid. The reverse, severe folic acid deficiency, occurs if vitamin $B_{12}$ is given to a child deficient in folic acid (Libermann & Branning, 2003).

Cerebral folate deficiency is defined by low active folate metabolites in the presence of normal folate metabolism. Cerebral folate deficiency could result from either disturbed folate transport or increased folate turnover in the central nervous system. Typical features begin to manifest from age 4 months, starting with irritability, sleep disturbances, psychomotor retardation, cerebellar ataxia, spastic paraplegia, and epilepsy. Children often show signs of decelerated head growth from ages 4 to 6 months. Visual disturbances begin around age 3 years with progressive hearing loss starting at age 6 years. Cerebral folate deficiency has been treated with carefully titrated doses of folinic acid to prevent over- or underdosage of folinic acid. Various conditions such as Rett syndrome, Aicardi-Goutieres syndrome, and Kearns-Sayre syndrome may also be related to cerebral folate deficiency (Ramaekers & Blau, 2004).

Food sources include beef, lamb, pork, chicken liver, deep-green leafy vegetables, asparagus, broccoli, orange juice, peanuts, some legumes, whole wheat, and brewer's yeast. Twenty-five percent to 50% of folic acid in food is bioavailable. As of 1998, all U.S. cereal grain products are enriched with folic acid. In addition to the United States, more than 50 other countries are now mandating folic acid fortification (Berry, Mullinare, & Hamner, 2009).

#### Vitamin C (Ascorbic Acid)

The usefulness of vitamin C in the diet stems from its role as an antioxidant, preventing free radical damage and other antioxidant vitamins from being oxidized and thus keeping them potent. It is also essential for growth and repair of tissues in all parts of the body, such as formation of collagen, bone, and cartilage. The skin may also be protected from free radical damage associated with ultraviolet light with adequate intake of vitamin C. Synthesis of hormones in the adrenal glands requires vitamin C, with vast amounts of this micronutrient depleted during times of stress such as surgery, illness, and infection. Studies have shown that ascorbic acid may help prevent increased blood pressure, atherosclerosis, and decreased serum cholesterol. Vitamin C also plays a role in the bioavailability of other micronutrients, such as converting folic acid to its active form and increasing the body's ability to absorb iron from nonheme sources.

**Vitamin C Research**

Research into the effects of the antioxidant properties of vitamin C and the risk of childhood leukemia, the leading cause of cancer morbidity for those younger than age 15 years, has shown promising results. A two-phase study in California including more than 500 children found that regular consumption (4 to 6 days per week) of oranges, bananas, or orange juice during the first 2 years of life was associated with reduced risk of leukemia in children diagnosed younger than age 15 years. Oxidative damage to DNA may be prevented as a result of the antioxidant properties of vitamin C, thus precluding an initiating event in carcinogenesis (Kwan, Block, Selcin, Month, & Buffler, 2004).

Both cross-sectional and follow-up studies have shown an association between decreased intake of vitamin C–containing fruits during the winter and an increased risk of wheezing symptoms in children. Asthma has been associated with oxidant and antioxidant imbalance. Patients with asthma have been shown to have decreased plasma concentrations of vitamin C and increased oxidative stress. Protective effects of citrus fruit consumption do not seem to follow a dose-related response, with positive effects shown with as little

as fruit once a week. Results, however, of various experimental studies regarding the effect of vitamin C supplementation on lung function and bronchial responsiveness are conflicting (Forastiere et al., 2000).

Vitamin C deficiency in the United States is rare, with the classic disease being **scurvy**. However, more recently pediatric patients have presented with symptoms of respiratory compromise, difficulty weight bearing, and increased irritability and were found to be vitamin C deficient as a result of extremely restricted food intake (Mawson, 2009). In general, vitamin C deficiency symptoms are often subtle and difficult to diagnose, with early signs being listlessness, weakness, irritability, vague muscle and joint pain, and weight loss. Late symptoms of deficiency include bleeding gums, gingivitis, loosening of teeth, and fatigue. Unlike most other animals, humans are incapable of producing vitamin C in their bodies and depend on the daily food supply. The level at which tissue saturation occurs is approximately 1,500 mg/day. Concentrated food sources include broccoli, Brussels sprouts, black currants, collards, guava, horseradish, kale, turnip greens, parsley, and sweet peppers. Interestingly enough, citrus fruits are not the most concentrated sources of vitamin C. The bioflavonoids in the skin are responsible for increased vitamin C absorption from citrus fruits. Few people realize that an orange loses 30% of its vitamin C concentration after being squeezed, with nonfortified commercial juice having almost no natural vitamin C left. No proven toxicity has been identified. However, there is a potential for formation of kidney stones, intestinal gas, and loose stools when extremely large doses are consumed (Taylor, Stampfer, & Curhan, 2004).

**scurvy** Classic vitamin C deficiency in which symptoms are often subtle and difficult to diagnose, with early signs being listlessness, weakness, irritability, vague muscle and joint pain, and weight loss.

## Fat-Soluble Vitamins

### Vitamin A

Vitamin A has essential roles in the proper function of the eye, growth and maintenance of epithelial tissue, protection from cardiovascular disease and cancer, immune system function, and reproduction. Vitamin A is especially useful in the prevention of both infectious and noninfectious diseases of the respiratory system.

#### Vitamin A Research

Chronic infection and illness can decrease the body's level of vitamin A, thereby weakening the mucous membranes and making them more susceptible to viral infection. Randomized controlled trials have shown inconsistent responses of childhood pneumonia with the use of vitamin A as an adjunct to the standard treatment of pneumonia (Huiming, Chaomin, & Meng, 2005; Rodriguez et al., 2005). There is growing evidence that vitamin A and beta-carotene provide some protection against various forms of cancer.

Beta-carotene may also offer hope to children infected with HIV where the infection has a devastating impact on children in developing countries. Poor nutrition and HIV-related adverse health outcomes contribute to a vicious cycle that may be slowed down by using nutritional interventions, including supplemental vitamin A. Periodic supplementation with vitamin A starting at age 6 months has been shown to be beneficial in reducing both mortality and morbidity among HIV-infected children (Fawzi, Msamanga, Spiegelman, & Hunter, 2005).

Vitamin A has been proposed as a nutrient for which infants at risk for chronic pulmonary insufficiency may have special requirements. There is suggestive evidence that high doses when given intramuscularly may reduce the incidence of death from chronic lung disease. Exogenous steroid therapy, which is often used to improve pulmonary compliance in ventilated premature infants, may compromise vitamin A status and induce restricted somatic and bone mineral growth (Atkinson, 2001). Further studies are needed to verify the role that vitamin A may play in preventing severe chronic lung disease in premature infants.

Each function of vitamin A can be satisfied by ingesting the compounds that form vitamin A, which are retinoids (preformed vitamin A) and carotenoids (precursor to vitamin A that the body converts into active vitamin A). More than 400 carotenoids exist, such as beta-carotene, lutein, canthaxanthin, zeaxanthin, lycopene, alpha-carotene, and cryptoxanthin. Although not all have vitamin A activity, many have powerful antioxidant properties. All forms of active vitamin A, both fat and water soluble, are stored in the liver where they can be mobilized for distribution to peripheral tissues. Because active vitamin A and beta-carotene have slightly different functions in the body, a combination should be consumed. Palmitate, a synthetic form of vitamin A, is water miscible and appropriate for children experiencing fat malabsorption. Chain-shortened and oxidized forms of vitamin A are excreted in the urine with intact forms excreted in the bile and lost with the feces (Mahan & Escott-Stump, 2008).

An estimated 250 million children globally are at risk for vitamin A deficiency. Traditionally recognized symptoms of deficiency are night blindness, skin disorders, suboptimal growth, and reproduction failure. In 1991, nearly 14 million preschool children, most from South Asia, had clinical eye disease (xerophthalmia) caused by vitamin A deficiency. Xerophthalmia involves atrophy of the periocular

glands, hyperkeratosis of the conjunctiva, and finally involvement of the cornea, leading to softening of keratomalacia and blindness. Although the condition is rare in the United States, where it is usually associated with malabsorption, it is more common in developing countries where it is a major source of blindness among children. Two-thirds of new patients die within months of going blind as a result of enhanced susceptibility to infections. Even subclinical vitamin A deficiency increases child morbidity and mortality (Mahan & Escott-Stump, 2008).

Measles is also a major cause of childhood morbidity and mortality, with vitamin A deficiency recognized as a risk factor for severe measles infections. The World Health Organization recommends administration of an oral dose of vitamin A (200,000 IU for children or 100,000 IU for infants) each day for 2 days to children with measles when they live in areas where vitamin A deficiency may be present. When comparing several studies, there was no evidence that vitamin A in a single dose was associated with a reduced risk of mortality among children with measles (Huiming et al., 2005). In contrast, Sudfeld, Navar, and Halsey (2010) noted reduced measles mortality with vitamin A supplementation in hospitalized children with measles complications in Africa. WHO recommends two consecutive days' worth of age-appropriate high doses. Vitamin A supplements are also being used in the premature infant at less than 1,000 grams to reduce oxygen requirements and overall mortality (Darlow & Graham, 2007).

Vitamin A deficiency produces characteristic changes in skin texture. These involve follicular hyperkeratosis in which blockage of the hair follicles with plugs of keratin causes "goose flesh" or "toad skin." The skin becomes dry, scaly, and rough. At first the forearms and thighs are involved, but in advanced stages the whole body is affected. Subsequently, deficiency leads to failures in systemic functions characterized by anemia and impaired immunocompetence. A deficiency also leads to the keratinization of the mucous membranes that line the respiratory tract, alimentary canal, urinary tract, skin, and epithelium of the eye. These changes hinder the roles that these membranes play in protecting the body against infections. Clinically, these conditions are manifest as poor growth, blindness, periosteal overgrowth of the cranium, or increased susceptibility to infections. Primary deficiencies result from inadequate intakes of preformed vitamin A and provitamin A carotenoids. Secondary deficiencies can result from malabsorption resulting from insufficient dietary fat, biliary or pancreatic insufficiency, liver disease, protein-energy malnutrition, or zinc deficiency.

Acute vitamin A deficiency is treated with large oral doses of vitamin A. When it is part of concomitant protein-energy malnutrition, correction of this condition is needed to realize benefits of vitamin A treatment. The signs and symptoms of vitamin A deficiency respond to vitamin A supplementation in the same order as they appear: Night blindness resolves quickly, whereas the skin abnormalities may take several weeks to resolve. Treatment with single doses of 200,000 IU of vitamin A has reduced child mortality by 35% to 70%. This approach is very costly, which has stimulated interest in increasing vitamin A content of local food systems as a more sustainable approach to preventing the deficiency (Mahan & Escott-Stump, 2008). In areas at risk of vitamin A deficiency, the recommendation based on studies is to provide supplementation to children younger than 5 years of age. This may be accomplished through fortification, food distribution programs, and horticultural developments. More than 190 million children are vitamin A deficient. Reducing this risk of mortality by 24% could mean a savings of approximately 1 million lives per year (Imdad, Herzer, Mayo-Wilson, Yakoob, & Bhutta, 2011).

Active vitamin A, or retinol, is found only in food sources with preformed vitamin A, which are foods of animal origin, either in storage areas such as the liver or associated with the fat of milk and eggs. Very high concentrations occur in fish liver oil from cod, halibut, salmon, and shark. Provitamin A carotenoids are found in dark green leafy and yellow-orange vegetables and fruit; deeper colors are associated with higher carotenoid levels. In much of the world, carotenoids supply most of the dietary vitamin A.

Persistent large doses of vitamin A, which overcome the capacity of the liver to store the vitamin, can produce intoxication. Congenital birth defects risk is associated with doses in excess of 10,000 IU. The damage occurs within days of conception during the embryonic stage. Specific defects associated with vitamin A excess includes damage to the ocular, pulmonary, cardiovascular, and urogenital systems (Lefebvre et al., 2010). Hypervitaminosis A can be induced by single doses of retinal greater than 330,000 IU in children, potentially resulting in liver disease. Vitamin A toxicity is characterized by changes in the

skin and mucous membranes. Early signs of toxicity include fatigue, nausea, vomiting, muscular incoordination, and dryness of the lips. Dryness of the nasal mucosa and eyes, erythema, scaling and peeling of the skin, hair loss, and nail fragility follow. Chronic hypervitaminosis can result from chronic intakes, usually from misuse of supplements, greater than at least 10 times the DRI of 14,000 IU for an infant (Mahan & Escott-Stump, 2008). Reversal of symptoms occurs when supplementation is discontinued. On the other hand, beta-carotene, a naturally occurring pigment, can be given for long periods of time virtually without risk of toxicity. Carotenemia, a harmless condition, is a sign that the body converted as much beta-carotene to active vitamin A as it can, leaving the excess as an orange-yellow pigment in the skin (Erdman et al., 2012).

### Vitamin D

Vitamin D is not truly a vitamin and can be described as a steroid hormone because it does not need to be supplied from a source outside the body. Recognized as the "sunshine vitamin," it is a hormone produced in the body by the photoconversion of 7-dehydrocholesterol in the skin. With adequate exposure to solar ultraviolet B radiation (290 to 320 nm), theoretically, no dietary supplement is needed (Mughal, 2002). There are two forms of vitamin D: vitamin $D_2$ (ergocalciferol) and vitamin $D_3$ (cholecalciferol), which is the preferred form because of its natural occurrence in the body. Both vitamin $D_2$ and $D_3$ become active hormone vitamin D after passing through the liver and kidneys. The bioactive form of vitamin D is produced not only in the kidneys but also in the placenta during pregnancy. In the active form it is considered to be a hormone because of its roles in calcium and phosphorous homeostasis, cell differentiation, and bone formation and maintenance. As compared with the knowledge of vitamin D in calcium homeostasis and skeletal growth, very little is known about its role in the central nervous system. Vitamin D may also have anticancer properties and play a role in treatment of immunologic disorders, mood behavior, and improvement in muscle strength (Chaudhuri, 2005; Erdman et al., 2012).

Lifestyle, skin pigmentation, clothing, degree of air pollution, and geographic latitude affect the degree of exposure to the sun and therefore the amount of vitamin D internally produced by the body. It is also speculated that iron deficiency may affect vitamin D handling in the skin, gut, or its intermediary metabolism (Wharton & Bishop, 2003). It is difficult to determine amount of sun exposure needed; however, modest exposure to sunlight is sufficient for most children to produce their own vitamin D. Light sun exposure to the hands and face for 20 to 30 minutes two to three times per week has been shown to release approximately 50,000 IU into circulation within 24 hours of exposure (Hollis & Wagner, 2004). Most vitamin D in older children and adolescents is supplied by sunlight exposure. Ethnic populations living in geographic areas with high levels of solar exposure throughout the year have darkly pigmented skin, a feature associated with reduced rate of cutaneous vitamin D. It is also questionable whether any vitamin D is synthesized during the winter months and whether the body's stores of this vitamin are able to meet the daily requirements during this period because the half-life of 25-hydroxyvitamin D is only 19 days (Chaudhuri, 2005; Mughal, 2002). As a population, dermatologists discourage unprotected exposure to sun because of the increased risk of skin cancer and premature aging.

**nutritional rickets** Vitamin D deficiency that was thought to have vanished but is reappearing.

The incidence of vitamin D deficiency, also known as **nutritional rickets**, was thought to have vanished, but it is reappearing. Concerns about vitamin D resurfaced because of the promotion of exclusive breastfeeding for long periods without vitamin D supplementation, particularly for infants whose mother are vitamin D deficient; reduced opportunities for production of the vitamin within the skin because of cultural practices and fear of skin cancer; and the high prevalence of rickets in groups in more temperate regions (Pawley & Bishop, 2004; Wagner & Greer, American Academy of Pediatrics Section on Breastfeeding, & American Academy of Pediatrics Committee on Nutrition, 2008). Rickets has been reported in children with chronic problems of lipid malabsorption and in those receiving long-term anticonvulsant therapies. Rickets can also be secondary to disorders of the gut, pancreas, liver, kidney, or metabolism; however, it is most likely the result of nutrient deprivation of not only vitamin D but also of calcium and phosphorus (Wharton & Bishop, 2003).

Clinical features of rickets vary in severity and age at onset, with symptoms present for months before diagnosis. Juvenile deficiency affects ossification at the growth plates, resulting in deformity and impaired linear growth of long bones

(Pawley & Bishop, 2004). A state of deficiency occurs months before structural abnormalities of the weight-bearing bones and is associated with bone pain, muscular tenderness, and hypocalcemic tetany. Soft, pliable, rachitic bones cannot withstand ordinary stresses, resulting in the appearance of bowlegs, knock-knees, pigeon breast, and frontal bossing of the skull. Improvement in clinical symptoms, such as aches and pains, occurs within 2 weeks of supplementation. Full correction of bowlegs and knock-knees can take up to 2 years. Adolescents are usually left with residual skeletal deformities that require surgical correction (Mughal, 2002).

Regular sources of dietary vitamin D are recommended for those otherwise at risk for deficiency. The American Academy of Pediatrics (Wagner et al., 2008) recommends the regular intake of a multiple vitamin with 400 IU vitamin D per day for all infants who are breastfed unless weaned to 1,000 mL/day of formula, for all nonbreastfeeding infants with less than 1,000 mL/day formula and for children not regularly exposed to sunlight and not ingesting 1,000 mL fortified milk. It is generally accepted that nutritional rickets can be prevented by dietary supplementation of 400 IU/day. In the past, human milk was thought to be an adequate source of vitamin D for neonates and growing infants (Hollis & Wagner, 2004). However, it has been determined that human milk typically contains 25 IU/L or less of vitamin D. Thus, the recommended intake cannot be met with expressed breast milk as a sole source (Wagner et al., 2008). The Institute of Medicine (IOM, 2011) recommends 600 IU per day for children 1 year of age and older. More data are needed to support adequacy of present recommendations and possibly even higher recommendations for vitamin D daily intake.

### Vitamin D Research

Previously, it was hypothesized that vitamin D deficiency in early life may be a risk factor for various neurologic and psychiatric diseases, including multiple sclerosis (MS), which is a demyelinating disease of the central nervous system that runs a chronic course and disables young people. The suggestion that vitamin D may have a protective effect against MS is not new. In areas at high latitudes where MS is common, ultraviolet radiation is too low to produce sufficient vitamin D in the fair-skinned population throughout the year. Vitamin D supplementation may significantly reduce the incidence of MS. The protective effects of sunlight exposure in early life and cutaneous vitamin D synthesis on the lifetime risk of developing MS are probably not without genetic predisposition because the population prevalence of MS does not parallel the prevalence of rickets. Although subclinical vitamin D deficiency is more common in dark-skinned people, MS is more common in fair-skinned people of European descent. Low vitamin D, however, appears to be an important modifiable external risk factor for MS (Chaudhuri, 2005).

Vitamin D deficiency has also been associated with **osteopenia** and osteoporosis in adults with cystic fibrosis and is potentially related to childhood deficiency. One study demonstrated lower serum 25(OH)D levels in children, adolescents, and young adults with cystic fibrosis despite vitamin D supplementation (Rovner et al., 2007). Lower levels in adolescents may be a precursor to low levels in adulthood and do not seem to be simply related to poor compliance with supplementation. Higher doses (more than 800 IU) may be necessary to keep serum levels within the recommended 30–60 ng/mL range.

**osteopenia** Bone mineral density that is lower than normal peak bone mineral density but not low enough to be classified as osteoporosis; often present in preterm infants, resulting in fractures.

Excessive intake of vitamin D can produce intoxication characterized by hypercalcemia and hyperphosphatemia and, ultimately, calcinosis of the kidney, lungs, heart, and even the tympanic membrane of the ear, which can result in deafness. Infants given excessive amounts of vitamin D may have gastrointestinal upsets, bone demineralization, poor appetite, and retarded growth. Nutritional hypervitaminosis results from pharmacologic doses of vitamin D consumed over a long period of time and is defined by a large increase in circulating 25-hydroxyvitamin D concentration. The Tolerable Upper Intake Level has been established at 1,000 IU/day for infants and 2,000 IU/day for children (Holick et al., 2011). Amount consumed to cause toxicity is thought to be 20,000 IU/day. Hypervitaminosis of vitamin D is a serious, albeit, very rare condition with mild cases being treatable (Hollis & Wagner, 2004). Evidence exists that synthetic active forms of vitamin D, available by prescription, may be better in individuals with malabsorption because smaller doses can be used and thus decrease the risk of toxicity. Caution is advised in dosing because some products are per drop versus per milliliter. There are single vitamin D supplement products with 400 IU vitamin D per milliliter or per drop. A recent case publication highlights the potential for vitamin D toxicity caused by dosing error using over-the-counter vitamin D supplements (Rajakumar, Cohen-Reis, & Holick, 2013).

Calcium and magnesium, as well as other vitamins and minerals, should be taken with vitamin D because these nutrients all work together in the body to form and maintain bone mass. Vitamin $D_3$ occurs naturally in animal products, the richest sources being fish liver oils and eggs. However, approximately 98% of all fluid milk sold in the

United States is fortified with vitamin $D_2$ (usually at the level of 400 IU per quart), as is most dried whole milk and evaporated milk as well as some margarines, butter, soy milk, certain cereals, and all infant formulas. A typical American diet does not supply the recommended 400–600 IU/day, requiring supplementation or fortification (Mughal, 2002; Samour & King, 2011). In many European countries, supplemented foodstuffs with vitamin D are limited; therefore, solar exposure is responsible for providing most of the body's vitamin D.

### Vitamin E

Vitamin E is now recognized as having a fundamental role in the normal metabolism of all cells as a powerful antioxidant and anticarcinogen. Benefits may be the result of its ability to protect vitamins A and C from oxidation, thus keeping them potent. In addition, vitamin E helps to increase the body's level of superoxide dismutase, an enzyme that is a powerful free radical scavenger. The antioxidant function of vitamin E can be affected by the nutritional status of a child with respect to one or more other nutrients that, collectively, protect against the potentially damaging effects of oxidative degradation. Therefore, deficiency can affect several different organ systems. The absorption of vitamin E is highly variable, with efficiencies in the range of 20% to 70% (Mahan & Escott-Stump, 2008) and is also dependent on fat intake. Vitamin E actually consists of eight substances, with alpha, beta, delta, and gamma tocopherols being the most active forms. The naturally occurring form is D-alpha-tocopherol, which appears to be the most absorbable.

#### Vitamin E Research

A variety of studies has provided inconclusive, but promising, results identifying additional roles vitamin E may play in childhood. The results of a study in China ($n = 120$) suggest that chronic childhood constipation causes oxidative stress, in which levels of vitamins C and E were both decreased (Wang, Wang, Zhou, & Zhou, 2004). Because of its antioxidant capability, vitamin E also helps protect the body from mercury, lead, carbon tetrachloride, benzene, ozone, and nitrous oxide exposure that brings about harm through the ability of these substances to act as free radicals. The use of vitamin E during cancer treatment to enhance the ability of radiation treatment to shrink cancer cells has been considered. Vitamin E may also play a role in wound healing and reduction of scar formation (Libermann & Branning, 2003).

Deficiencies of vitamin E in childhood present in a variety of ways. Children with progressive neuromuscular diseases are reported to have vitamin E deficiency, which may take 5 to 10 years to develop. Deficiency is manifested clinically as abnormal reflexes, impaired ability to walk, changes in balance and coordination, muscle weakness, and visual disturbances, which are improved after vitamin E supplementation. Additional deficiency symptoms include anemia caused by premature aging and death of red blood cells. These symptoms generally only appear when severe fat malabsorption results from disorders of the pancreas, celiac disease, intestinal failure, and cystic fibrosis. The limited transplacental movement of vitamin E results in newborn infants having low tissue concentrations of vitamin E. Premature infants may therefore be at increased risk of vitamin E deficiency because they typically have limited lipid absorptive capacity for some time (Mahan & Escott-Stump, 2008). Premature infants may also suffer from disorders of the retina, which can lead to blindness, as a result of vitamin E deficiency.

Vitamin E is synthesized only by plants and therefore is found primarily in plant products, the richest sources being oils such as cottonseed, corn, soybean, safflower, and wheat germ. Smaller amounts may be found in whole grains, green leafy vegetables, nuts, and legumes. Animal tissues tend to contain low amounts of vitamin E, the richest source being fatty tissues and tissues of animals fed large amounts of the vitamin.

Vitamin E is one of the least toxic of the fat-soluble vitamins. Humans appear to be able to tolerate relatively high intakes, at least 100 times the nutritional requirement. At very high doses, however, vitamin E can antagonize the utilization of other fat-soluble vitamins (Mahan & Escott-Stump, 2008). It is also not clear whether high-dose vitamin E supplementation is effective in reducing oxidative stress and damage and therefore in reducing chronic diseases (Erdman et al., 2012). Many supplements contain only alpha-tocopherols. However, it is recommended that a mixture of tocopherols be provided because this is how they exist in foods. For children experiencing fat malabsorption, vitamin E succinate is an alternative, synthetic, oil-free powder that is water miscible and well tolerated. With very high doses of more than 1,200 IU/day some adverse effects result, such as nausea, flatulence, diarrhea, heart palpitations, and fainting, which are reversible upon decrease in dose. To avoid adverse side effects, the recommendation is to begin with the provision low dose and increase gradually (Libermann & Branning, 2003).

### Vitamin K

*Vitamin K* is a general term used to describe a group of similar compounds. Vitamin $K_1$ is found in food, vitamin $K_2$ is made by intestinal bacteria, and vitamin $K_3$ is a synthetic form only available by prescription. For this reason vitamin K is not strictly considered a vitamin, with its most important function being the role it plays in production of coagulation factors in the body. Vitamin K is required to make prothrombin, which is converted to thrombin, which converts fibrinogen to fibrin and creates a blood clot. Newborn infants, particularly those who are premature or exclusively breastfed, are susceptible to hypoprothrombinemia during the first few days of life as a result of poor placental transfer of vitamin K and failure to establish a vitamin K–producing intestinal microflora. This is associated with hemorrhagic disease of the newborn, which is treated prophylactically by administering 1 mg vitamin K intramuscularly on delivery, which has been a standard of care since 1961 (Lauer & Spector, 2012; Shearer, Fu, & Booth, 2012).

Vitamin K is also essential for development of normal bone density during childhood and is thought to be important in preventing the development of osteoporosis in later life. Relatively little research emphasizes the effect of vitamin K on bone health during childhood. Better vitamin K status is associated with lower levels of markers of bone resorption and bone formation, suggesting a lower rate of bone turnover. There is a need for randomized supplementation trials to better understand the role of vitamin K in bone acquisition in growing children (Cashman, 2005). A marked reduction in bone mineral density has also been observed in children on long-term use of warfarin, a vitamin K antagonist. The etiology for reduced bone density is likely to be multifactorial; however, screening children on long-term warfarin for reduced bone density should be considered (Newall et al., 2005).

Because vitamin K is easily obtained from the diet and synthesized in the body, deficiencies are rare and usually occur only with lipid malabsorption, destruction of intestinal flora as with chronic antibiotic therapy, and liver disease in which supplementation is recommended. The predominant sign of vitamin K deficiency is hemorrhage, which in severe cases can cause a fatal anemia. The underlying condition is hypoprothrombinemia, which manifests as prolonged clotting time (Mahan & Escott-Stump, 2008).

Vitamin K is found in large amounts in green leafy vegetables, especially broccoli, cabbage, turnip greens, and dark lettuces. Breast milk tends to be low in vitamin K content, providing insufficient amounts of the vitamin for infants younger than 6 months of age. An important source of vitamin K is a result of microbiologic synthesis in the gut, which is affected by long-term antibiotic treatment by altering the gut flora (Samour & King, 2011). The synthetic form, vitamin $K_3$, is known to potentially result in toxicity when consumed in large doses. Major symptoms of overdose include hemolytic anemia, in which red blood cells die more quickly than the body can replace them, hyperbilirubinemia, and kernicterus in amounts greater than 5 mg/day to infants (Erdman et al., 2012).

## Multiple Vitamin Supplementation

Multiple vitamin supplementation is a common practice in the United States, with approximately 54% of preschool children taking a supplement, typically a multiple vitamin and mineral preparation with iron. Use of supplementation generally decreases in older children and adolescents. Children taking supplements do not necessarily represent those who need them the most. Higher rates of use are found in families of higher socioeconomic status and education levels. Supplements may be taken inappropriately when the marginal or deficient nutrient is not supplied. For example, a child may be taking a children's vitamin but may actually need extra calcium, not always provided in a supplement. Except for fluoride supplementation in nonfluoridated areas, the American Academy of Pediatrics (AAP) does not recommend routine supplementation for normal, healthy children. The American Medical Association and the Academy of Nutrition and Dietetics recommend that nutrients for a healthy child come from food, not supplements. Dietary evaluation determines the need for supplementation.

The AAP identifies six groups of children or adolescents at nutrition risk who might benefit from supplementation: (1) those with anorexia, poor appetites, and fad dieting; (2) those with chronic disease states such as cystic fibrosis or irritable bowel syndrome; (3) those from deprived families involving abuse or neglect; (4) those who use diet to manage obesity; (5) those who have an inadequate consumption of dairy products; and (6) those who have limited sun exposure or no consumption of vitamin D–fortified milk. Children with food allergies, those who omit entire food groups, and those

with limited food acceptances may also be likely candidates for supplementation. Megadose levels of nutrients should be discouraged and parents counseled regarding dangers of toxicity, especially with fat-soluble vitamins. The Dietary Reference Intakes include the Tolerable Upper Intake Levels, which can be used to detect excess levels of vitamins and minerals from supplemental sources. Because many children's vitamins look and taste like candy, parents should be educated to keep them out of reach of children. Megavitamin therapy has been promoted, despite the lack of controlled studies, for many disorders, such as attention deficit hyperactivity disorder (ADHD), autism, and various behavioral problems.

**Learning Point** Except for fluoride supplementation in nonfluoridated areas, the AAP does not recommend routine supplementation for normal healthy children. The American Medical Association and the Academy of Nutrition and Dietetics recommend that nutrients for a healthy child come from food, not supplements. Dietary evaluation determines the need for supplementation.

## Minerals

**Minerals** are inorganic elements not produced by plants and animals and are primarily stored in bone and muscle tissues. Like vitamins, many minerals function as coenzymes, enabling chemical reactions to occur throughout the body. Minerals belong to two groups: **macrominerals**, which are needed in large amounts (calcium, magnesium, and phosphorus), and microminerals, or **trace minerals**, which are needed in much smaller amounts (zinc, iron, and potassium). Currently, nine trace minerals are considered to have a nutrient requirement: iron, zinc, copper, selenium, iodine, manganese, molybdenum, chromium, and cobalt. Notable differences between trace elements are the availability of biomarkers such as tissue concentration, mineral homeostasis and metabolism, body stores, functional indices, and response to increased intake.

**minerals** Inorganic elements not produced by plants and animals and primarily stored in bone and muscle tissues.

**macrominerals** Minerals that are needed in large amounts (calcium, magnesium, and phosphorus).

**trace minerals** Minerals that are needed in small amounts (iron, zinc, copper, selenium, iodine, manganese, molybdenum, chromium, and cobalt).

### Calcium

Calcium is an essential mineral with a wide range of biological roles. Apart from being a major constituent of bones and teeth, calcium is crucial for muscle contraction and relaxation, transmission of nerve impulses, the beating of the heart, blood coagulation, glandular secretion, and the maintenance of immune function. Calcium is also used to activate enzymes involved in fat and protein digestion and production of energy. Calcium also aids in the absorption of many nutrients, especially vitamin $B_{12}$. Studies have also shown potential anticarcinogenic, antihypertensive, and hypocholesterolemia activity (Hendler & Rorvik, 2008).

Human bodies contain approximately 2½ pounds of calcium, 99% of which is stored in the bones and teeth with the remaining 1% (13 oz) distributed throughout the body in the bloodstream and fluids surrounding cells. Calcium is primarily found in bone and teeth in the form of the calcium phosphate compound hydroxyapatite (Hendler & Rorvik, 2008). The skeleton functions as a bank from which the body can draw the calcium it needs for various purposes. Therefore, identifying skeletal growth is a dynamic process. Approximately 100 mg of calcium per day is retained as bone during the preschool years. This triples and quadruples for adolescents during peak growth. Normally, sufficient calcium absorption from diet, blood levels, and bone calcium levels remain in balance and fluctuate only slightly. From the body's point of view, however, it is more important to maintain enough calcium in the blood to keep the heart beating regularly than it is to keep the bones strong. Because the body gives top priority to the maintenance of normal calcium levels in the blood, blood tests are an ineffective means of determining calcium levels in either the bone or diet. The concentration of calcium in the blood may be perfectly normal, whereas bone mineral density is suboptimal. If a child's diet is deficient in calcium, the body will always choose to maintain plasma calcium concentration by drawing it out of the bone, often resulting in osteopenia (Erdman et al., 2012; Samour & King, 2011).

Optimal dietary calcium and possibly vitamin D intakes throughout childhood and adolescence may enhance bone mineral accrual and are central to osteoporosis prevention. During adolescence, children often receive less calcium because of rapid growth, dieting, and substitution of carbonated beverages for milk. From early childhood to late adolescence, it has been reported that milk intake decreases by 25%. Mean calcium intake from ages 5 to 9 years is positively related to bone mineral density at 9 years of age and is weakly related to bone mineral content after control for pubertal

development and height at age 9 years. Adolescence is a critical period for optimal calcium retention to achieve peak bone mass, especially for females at risk for osteoporosis in later years. During this period of rapid bone mineral acquisition, adolescent girls' calcium intakes are reported to be 30% to 40% below the 1,300 mg/day recommendation (IOM, 2011; Samour & King, 2011), placing these adolescents at risk for calcium deficiency. Studies have also shown that gender, lifestyle factors, and socioeconomic status are significant predictors of calcium and vitamin D intakes (Salamoun et al., 2005). Children who are vegetarians and do not consume fortified foods may also be at risk for not consuming the recommended calcium requirement. These findings have important implications regarding the institution of public health strategies to promote skeletal health during a critical time for bone mass accrual.

Bone health is now recognized as contributing to overall lifetime management of children and adolescents with disabling conditions, including physical and intellectual disabilities and chronic diseases. Increased skeletal fragility in children with disabilities is well recognized. Strategies to address bone health, including public and medical education concerning consumption of calcium, appropriate selection of vitamin D preparations, and possible use of newer drugs, such as bisphosphonates, are changing the outlook for this large group (Zacharin, 2004).

Nutrition and oral health are also closely related. Micronutrients such as calcium, vitamin D for mineralization, and fluoride for enamel formation are critical to developing and maintaining oral structures. Because of individual variability, children receiving less than the recommended allowance of calcium may not necessarily be at risk.

Clinical signs of calcium depletion from bone gradually develop and are not usually apparent until the symptoms of osteopenia begin to appear. Even X-rays are incapable of picking up bone loss until 30% to 40% of bone has disappeared. Calcium-deficient diets, over long periods of time, result in bones that become porous, brittle, and weak. Children may easily suffer fractures from normal activity such as sneezing, bending over, or receiving a hug. The spinal vertebrae have also been reported to compress or fracture. Inconclusive studies have linked diets high in protein and fat with loss of calcium from the bone. Medications, such as antibiotics, anticonvulsants, and laxatives, can also affect absorption and utilization of calcium (Libermann & Branning, 2003).

Needs are determined by growth velocity, rate of absorption, and phosphorus and vitamin D status. The absorption efficiency of calcium varies throughout the life cycle. It is highest during infancy, when it is about 60%, with a decrease to approximately 28% in prepubescent children. During early puberty, at the time of the growth spurt, it increases to about 34% and then again drops to 25% 2 years later, where it remains for several years. The efficiency of absorption from a calcium supplement is greatest when taken in doses of 500 mg or lower (Hendler & Rorvik, 2008). Genetics and lifestyle also affect bone mineral density and possible calcium requirements. When assessing calcium status, vitamin D intake should be considered because of its major role in calcium metabolism. Concomitant use of vitamin D and calcium may increase absorption of calcium. The ratio of calcium to phosphorus has also been advised by researchers to be at least 1:1 to improve calcium absorption (Hambridge, 2003). Concomitant use of iron and calcium may inhibit the absorption of iron. Similarly, concomitant use of fluoride, magnesium, phosphorus, or zinc and calcium may decrease the absorption of these minerals (Hendler & Rorvik, 2008).

Food sources include dairy, canned salmon or sardines including the bones, green leafy vegetables, clams, oysters, shrimp, broccoli, soybeans, and soy products. Although unfortified plant foods have less calcium than milk does, increased absorption rate makes them good sources of this nutrient overall. Vitamin D–fortified milk is a primary source of this nutrient. It has been estimated that humans actually absorb as little as 20% to 40% of the calcium in food. This may be a result of the presence of oxalic acid, a substance that has been shown to bind with calcium, preventing its absorption in the colon. Phytate is another inhibitor of calcium absorption. Limited known toxic effects have been reported, such as the rare development of kidney stones as a result of extremely large doses (Erdman et al., 2012).

### Iron

Iron is an essential trace mineral that is involved in the entire process of respiration as a result of its role in the synthesis of hemoglobin, which carries oxygen in the blood. In addition to its fundamental roles in energy production, iron is involved in DNA synthesis and may also play roles in normal brain

development and immune function. Iron is also involved in the synthesis of collagen, serotonin, dopamine, and norepinephrine.

Iron is the most notable example of a mineral that is stored by the body, as ferritin, when intake of bioavailable iron is generous and released as required when intake is less adequate. Serum ferritin is highly correlated with total body iron stores, making it the most sensitive index of iron status in healthy children. Requirements are determined by rate of growth, iron stores, increased blood volume, and rate of absorption from food sources. Menstrual loss and rapid growth increase the need in adolescent girls. Reliable biomarkers of the total quantity of dietary iron are unavailable because of the wide variation in bioavailability and especially because of the large difference in absorption of heme iron versus inorganic iron. In contrast, a range of biomarkers is available that in combination allow for reliable assessment of iron status, therefore identifying the adequacy of iron intake, especially in noninfected nonstressed individuals. Biomarkers can distinguish three generally accepted levels of iron deficiency and are also useful in the evaluation of iron overload. The three levels of iron deficiency are depleted iron stores, early functional iron deficiency, and iron-deficiency anemia (Hambridge, 2003).

**Iron deficiency** is the most common nutritional disorder in the world, with approximately 25% of the world's population being deficient. Iron-deficiency anemia is most common in children between ages 1 and 3 years, with a prevalence of approximately 9%. Over the last two decades, there has been an overall decrease in prevalence of iron-deficiency anemia. Factors that have influenced this are prolonged used of iron-fortified infant formulas, increased breastfeeding, iron intake in the form of food sources, and use of the Supplemental Nutrition Assistance Program and Women, Infants, and Children food program. The AAP recommends universal or selective screening for infants between 9 and 12 months of age, with a second screening 6 months later. Universal screening up to age 2 years for communities with significant levels of iron deficiency or infants whose diets put them at risk is also recommended. Selective screening may be based on risk factors such as prematurity, low birth weight, and dietary intake. Routine screening is not recommended for children older than 2 years of age except when risk factors such as poor diet, poverty, and special needs are present (Samour & King, 2011).

**iron deficiency** The most common nutritional disorder in the world, with approximately 25% of the world's population being deficient.

The relationship between iron deficiency and cognition has been debated for many years. Much of the research has been focused on the idea that even in the early stages of iron deficiency, iron-dependent neurotransmitters can be altered, which in turn can impair learning ability and behaviors. It is plausible that some cerebral changes occur soon after the reserve of iron is depleted and there is a decreased activity in nutrient-dependent enzymes. This could be followed by constraint in motor development and physical activity secondary to a drop in the oxygen supply to muscle fibers. Iron deficiency during infancy may have long-term consequences, as demonstrated by impaired cognitive development and impaired learning ability. Anemic children have been reported to be at risk of delayed acquisition of developmentally appropriate emotional regulation (Carter et al., 2010). It is not certain, however, whether iron status is the major factor contributing to poor school performance of children with general lack of nutrition, such as that which often occurs in rural areas. For these children, cumulative overall nutrient intake may affect school performance more significantly than their iron intake does. Despite the improving trends, iron deficiency is especially common among children from low-income families; many of these children have the more severe form of the disorder, iron-deficiency anemia (Kim et al., 2005).

Rapid growth during the first year of life requires an adequate supply of iron for synthesis of blood, muscle, and other tissues. Preterm infants have an increased requirement for dietary iron to facilitate appropriate growth. Most health authorities recommend exclusively breastfeeding for 4 to 6 months. Iron in human milk, however, is low, but it is thought that the high bioavailability partly compensates for decreased concentration and iron stores are typically adequate in a full-term infant for the first 4–6 months of life (Ziegler, Nelson, & Jeter, 2009). Iron absorption is regulated by recent dietary iron intake, independent of the size of iron stores and the rate of erythropoiesis. Observational studies suggest that dietary regulation of iron absorption is immature in an infant younger than 6 months of age and is subject to developmental changes between 6 and 9 months of age. Changes in the regulation of iron absorption between 6 and 9 months of age enhance the infant's ability to

adapt to a low-iron diet and provide a mechanism by which some, but not all, infants avoid iron deficiency despite low iron intake in late infancy. This adaptation may not be sufficient to prevent iron deficiency in exclusively breastfed infants, especially those who were born prematurely. To prevent this, iron supplements are often recommended for breastfed infants after 4 to 6 months if they are not consuming adequate amounts of iron-rich complementary foods. Iron supplementation is also often recommended for older breastfeeding infants who are not consuming iron-rich infant cereals (Erdman et al., 2012).

Different stages of iron deficiency involve different systemic changes, which in turn affect different psychobiological domains. During early iron deficiency, the supply of iron to the bone marrow is marginal, not causing measurable decline in hemoglobin. Iron-deficiency anemia is associated with variation in red blood cell width. Hemoglobin and hematocrit are the main biological screening tests because of the simplicity of measurements. Low values occur only during later stages of deficiency and result in impairment of normal physiology (Hambridge, 2003). Acute blood loss, acute and chronic infection, micronutrient deficiency (vitamin $B_{12}$ or folate), and defects in red blood cell production (sickle cell or thalassemia) may also cause low values. An advantageous side effect of correcting low iron levels with iron supplements has been shown to be improved appetite (Pawley & Bishop, 2004).

Iron absorption from food depends on iron status of the individual (those with low iron stores have increased absorption rate) and increased absorption rate from heme iron versus nonheme sources. It is uncertain whether excessive dietary iron levels result in abnormally high ferritin levels. A typical American mixed diet contains approximately 6 mg iron per 1,000 calories. Adolescents dieting to lose weight will likely have minimal iron intake, especially if animal protein is limited. The best dietary sources of iron are green vegetables, legumes, iron-fortified cereals, and meat. Much of the iron ingested in the American diet in the form of bread and cereals is not well absorbed. Although iron is clearly essential for a wide range of vital biological processes, it is also a potentially toxic substance. Iron overload disorders, which can lead to cirrhosis, coronary heart disease, and congestive heart failure, are also a public health concern (Erdman et al., 2012).

### Magnesium

Magnesium is an essential mineral involved with more than 300 metabolic reactions. It is necessary for every major biological process, including the production of cellular energy and the synthesis of nucleic acids and proteins. It is also important for the electrical stability of cells, the maintenance of membrane integrity, muscle contraction, nerve conduction, and regulation of vascular tone. About 50% to 60% of the body's magnesium content is in the bone, whereas a mere 1% is found extracellularly. Magnesium is the second most abundant intracellular cation, with potassium being the most abundant. There is much about the pharmacokinetics of magnesium that is not known (Erdman et al., 2012).

#### Magnesium and Respiratory Illness: A Connection?

Randomized controlled trials assessing the use of magnesium sulfate in asthmatic children with moderate exacerbation have produced contradictory results. Higher intakes of magnesium are associated with lower incidence of airway reactivity and respiratory symptoms. There is evidence that magnesium functions, in part, by antagonizing calcium in membrane channels of smooth muscle cells, thus decreasing uptake of calcium and leading to smooth muscle relaxation. In addition, magnesium may inhibit release of histamine and other inflammatory mediators. More research is needed to further determine the relative value of supplemental magnesium in the prevention and treatment of asthma and related conditions. There is poor correlation between serum and tissue magnesium levels. Therefore, children who have serum levels falling within a normal range may be total body magnesium deficient. Because magnesium deficiency is difficult to diagnose, one question that has proved difficult to answer is whether all asthmatic patients may potentially benefit from magnesium's physiologic effects.

Magnesium deficiency is associated with the pathogenesis of numerous serious disorders, notably ischemic heart disease, congestive heart failure, sudden cardiac death, diabetes, and hypertension. Treatment with supplemental magnesium is often helpful in these conditions. It may help prevent or reduce the incidence of cerebral palsy and mental retardation in preterm infants. Symptoms and signs of magnesium deficiency include anorexia, nausea and vomiting, diarrhea, generalized muscle spasticity, confusion, seizures, and cardiac arrhythmias. Magnesium deficiency may be found in diabetes mellitus, malabsorption syndromes, and hyperthyroidism. Long-term use of diuretics may also result in magnesium deficiency. Magnesium deficiency itself is an important cause of hypokalemia (Erdman et al., 2012).

Magnesium is associated with a low risk of adverse effects that are most often related to the

rate of administration and the dose given. The most common adverse reaction from the use of magnesium supplements is diarrhea. Other gastrointestinal symptoms that may occur with the use of magnesium supplements are nausea and abdominal cramping. Magnesium, excreted by the kidney, is contraindicated in patients with renal failure. Those with renal failure may develop hypermagnesemia with use of magnesium supplements. Magnesium seems to have a wide therapeutic window in which doses expected to produce clinical benefits should not result in significant adverse effects (Erdman et al., 2012).

Foods rich in magnesium include unpolished grains, nuts, and green leafy vegetables, which are particularly good sources because of their chlorophyll content. Refined and processed foods are generally poor sources of magnesium. The efficiency of absorption of magnesium is inversely proportional to the amount of magnesium ingested.

### Potassium

Potassium is an essential micromineral that is important in the transmission of nerve impulses; contraction of the cardiac, smooth, and skeletal muscles; production of energy; and synthesis of nucleic acids. Its antihypertensive properties were first identified in 1928. Evidence suggests that high intakes of potassium also protect against strokes and cardiovascular disease (Hendler & Rorvik, 2008).

The major cause of potassium depletion is excessive losses through the alimentary tract and kidneys. Depletion typically occurs as a result of chronic diuretic use, from severe diarrhea, or in children receiving long-term total parenteral nutrition inadequately supplemented with potassium. The effects of potassium depletion on the rapidly growing infant have not been well studied. Chronic hypokalemia has been associated with renal hypertrophy, interstitial disease, and hypertension in adults. Additional symptoms of depletion include metabolic alkalosis, anorexia, fatigue, and cardiac arrhythmias (Hendler & Rorvik, 2008). Animal studies, including in young rats, have shown significant growth retardation and increased renin-angiotensin system activity. Potassium-depleted kidneys also showed early fibrosis. Systolic blood pressure was also elevated in potassium-depleted rats, which persisted even after the serum potassium was normalized (Ray, Suga, Liu, Huang, & Johnson, 2001).

Foods that are rich in potassium include meats, fresh fruits, and vegetables. The efficiency of absorption of supplementary potassium is significant at approximately 90% via the gastrointestinal tract. Dietary potassium is absorbed at a similar rate. The most common adverse effects of potassium supplementation include nausea, vomiting, abdominal discomfort, and diarrhea. Oral potassium supplements rarely result in hypokalemia when normal renal function is present.

### Phosphorus

Phosphorus is an essential macromineral that plays a pivotal role in the structure and function of the body. Phosphorus is essential for the process of bone mineralization and makes up the structure of the bone. Phosphorus in the form of phospholipids makes up the structure of cellular membranes. Phosphorus also makes up the structure of nucleic acids and nucleotides, including adenosine triphosphate. During the last trimester of pregnancy, the human fetus accrues approximately 80% of the calcium, phosphorus, and magnesium present at term. Therefore, infants born prematurely require higher intakes of these minerals per kilogram of body weight than do term infants. Providing adequate amounts of these nutrients, particularly phosphorus, to very low birth weight infants during the first few weeks of life is not always possible. As a result, osteopenia is frequent in these infants, with fractures developing in some. Preterm human milk is low in phosphorus and has been associated with impaired bone mineralization and rickets. The addition of human milk fortifiers has improved bone mineralization (Kleinman & American Academy of Pediatrics Committee on Nutrition, 2009).

Phosphorus deficiency states are usually a result of malabsorption syndromes and diseases causing renal tubular losses of phosphorus. In addition, those with malnutrition and critically ill patients, such as those being treated for diabetic ketoacidosis, are at risk for phosphorus deficiency and phosphorus imbalance. The so-called refeeding syndrome can cause hypophosphatemia, which may be life threatening. Phosphorus deficiency can result in anorexia, impaired growth, osteomalacia, skeletal demineralization, proximal muscle atrophy, cardiac arrhythmias, respiratory insufficiency, nervous system disorders, and even death.

Phosphorus supplements, in the form of phosphate salts, are not widely used in the United States to treat phosphorus deficiency. The one exception is calcium phosphate, which is mainly used as a delivery form of calcium. The most common adverse

reaction to use of sodium or potassium phosphate is diarrhea. The salts are less likely to cause diarrhea when they are used by phosphorus-deficient individuals than when used by those with normal phosphorus status. Those with renal failure may develop hyperphosphatemia, which can result in ectopic calcification (Hendler & Rorvik, 2008).

Phosphorus, mainly in the form of phosphates, is widely distributed in the food supply, and phosphorus intake from the normal diet is usually sufficient to meet the body's needs. Meat, milk, and milk products are particularly good sources of phosphorus.

### Copper

Copper essentiality was first demonstrated in the late 1920s when malnourished children with anemia were nonresponsive to iron supplementation. Anemia, neutropenia, and osteopenia are found with frank copper deficiency. Twenty years later the essentiality of copper for fetal development was reestablished when it was shown that deficits of this nutrient during pregnancy can result in gross structural malformations in the fetus and persistent neurologic and immunologic abnormalities. Copper is required for normal infant development, red and white blood cell maturation, iron transport, bone strength, cholesterol metabolism, myocardial contractility, brain development, and immune function. Premature and small-for-gestational-age infants who are born with low copper stores can exhibit signs of copper deficiency, including neutropenia, normocytic hypochromic anemia, osteopenia, pathologic bone fractures, depigmentation of the skin and hair, and distended blood vessels. During the past decade, there has been increasing interest in the concept that marginal deficits of this element can contribute to the development and progression of a number of disease states, including cardiovascular disease and diabetes.

**Menkes disease** X-linked genetic disorder that is responsible for copper deficiency. Symptoms are characterized by hypothermia; neuronal degeneration; mental retardation; abnormalities in hair, skin, and connective tissue; bone fractures; and widespread vascular abnormalities, with death typically occurring by age 4 years.

Several human disorders with genetic mutations in copper transporters have further defined the role of copper in human health. Infants with the X-linked genetic disorder **Menkes disease** have a gene mutation that is responsible for copper deficiency. Symptoms are hypothermia; neuronal degeneration; mental retardation; abnormalities in hair, skin, and connective tissue; bone fractures; and widespread vascular abnormalities, with death typically occurring by age 4 years. Some of the symptoms likely may be secondary to excessive iron accumulation and subsequent oxidative stress (Uriu-Adams & Keen, 2005).

Plasma copper and ceruloplasmin levels are the most frequently used biomarkers of copper status and are depressed in copper deficiency states. Levels plateau when copper intake reaches an adequate level, and these biomarkers do not reflect the magnitude of copper intake beyond this point (Hambridge, 2003). Low intakes of copper in the diet can result in marginal copper deficiency in young children. Although the mean dietary intake of copper in most countries is close to the Dietary Reference Intake, a substantial percentage of individuals have intakes less than the recommendation, fueling the concern that marginal copper deficiency may be a common public health concern. Moreover, high dietary intakes of iron or zinc can adversely affect copper status. For example, infants consuming infant formula with high iron absorb less copper than do those consuming formulas with low iron level. Manifestations of mild copper deficiency may include abnormal glucose intolerance, hypercholesterolemia, myocardial disease, arterial disease, cardiac arrhythmias, loss of pigmentation, and neurologic symptoms.

Although copper is clearly essential for a wide range of biochemical processes, which are necessary for the maintenance of good health, it is also a potentially toxic substance. There are a number of case reports of acute copper toxicity in the literature. Typically, these case reports represent instances when the acute toxicity is caused by ingestion of beverages (including water) that have been contaminated with copper. Acute copper toxicity can result in a number of pathologies and, in severe cases, death. **Wilson disease** is an inherited disorder of copper metabolism characterized by a failure of the liver to excrete copper, leading to its accumulation in the liver, brain, corneas, and kidneys, with resulting chronic degenerative changes. Copper toxicity, typically resulting from genetic disorders, can also be a significant health concern (Uriu-Adams & Keen, 2005).

**Wilson disease** An inherited disorder of copper metabolism characterized by a failure of the liver to excrete copper, leading to its accumulation in the liver, brain, corneas, and kidneys, with resulting chronic degenerative changes.

The richest food sources of copper include nuts, seeds, legumes, liver, kidneys, whole grain products, and shellfish. Absorption efficiency appears to

depend on the level of dietary copper intake with a range of approximately 15% to 97%. As dietary copper increases, the fractional absorption of copper decreases. Excessive intake of nonheme iron, zinc, and vitamin C may decrease copper status (Hendler & Rorvik, 2008).

### Zinc

Zinc is an essential nutrient that because of its fundamental role in many aspects of cellular metabolism is critical for normal immune function and physical growth. Zinc is critically involved in several biological mechanisms related to growth, including protein synthesis, gene expression, and hormonal regulation. Zinc is a trace mineral that is involved with RNA and DNA synthesis and is critical to cellular growth, differentiation, and metabolism. The body also appears to have the ability, though modest, to store zinc. As the global public health importance of zinc deficiency has attracted increasing attention during the past decade, so have the limitations of current biomarkers. Plasma zinc is currently the most widely used and accepted biomarker despite poor sensitivity and imperfect specificity. Investigators often use indirect measures of zinc status because only a small percentage of the body's zinc is in plasma. Most of the body's zinc is located in muscle, bone, and liver, where it turns over slowly (Black, 2003; Hambridge, 2003).

Animal studies have shown that zinc deficiency probably begins to affect the growth process before birth (Merialdi et al., 2004). Zinc deficiency may be particularly relevant to early development because it is an essential trace element that plays fundamental roles in cell division and maturation and in the growth and function of many organ systems, including the neurologic system. Zinc deficiency may also influence child development by altering a child's ability to elicit or use nurturing interactions from caregivers. Low-birth-weight infants have been noted to have low zinc concentrations in cord blood, and zinc deficiency in childhood is associated with reduced immunocompetence and increased infectious disease morbidity (Sazawal et al., 2001). With a small liver and thus very limited hepatic stores of zinc and increased requirements for catch-up growth, low-birth-weight infants are at risk for zinc deficiency. Healthy infants are usually able to get an adequate amount of zinc from breastfeeding during the first 6 months of life, as long as the mother's milk supply is adequate and breast milk is not displaced by complementary foods. However, infants who are small for gestational age and premature infants may benefit from zinc supplementation in addition to the zinc they receive from breastfeeding. During the second 6 months of life when complementary foods are introduced, the risk for zinc deficiency increases because most traditional complementary foods are low in bioavailable zinc (Black, 2003).

#### Zinc Research

Investigations of zinc supplementation on infants' development have yielded inconsistent findings. There are suggestions that perhaps zinc supplementation may promote activity and perhaps motor development in the most vulnerable infants. Although motor development is thought to promote cognitive development by enabling children to be more independent and to explore their surroundings, the only evidence linking zinc supplementation to cognitive development is counterintuitive (Black et al., 2004). Long-term follow-up studies among zinc-supplemented infants are needed to examine whether early supplementation leads to developmental or behavioral changes that have an impact on school performance.

Zinc deficiency is regarded as a major public health problem with multiple health consequences. Zinc deficiency appears to be widespread in low-income countries because of low dietary intake of zinc-rich animal source foods and a high consumption of cereal grains and legumes, which contain inhibitors of zinc. The prevalence of zinc deficiency throughout childhood is estimated to be high, primarily related to the low consumption of foods high in bioavailable zinc. Children in poor countries are also frequently affected by diarrhea, which causes excess fecal losses of zinc (Penny et al., 2004). In such settings, zinc supplementation trials among nutritionally deficient infants have demonstrated beneficial effects on mortality and on health indicators, including growth, diarrhea, and pneumonia morbidity (Brown, Peerson, Rivera, & Allen, 2002; Lazzerini & Ronfani, 2012). Improved zinc status has been associated with improved appetites in young stunted children. Randomized trials are necessary to examine the specificity of zinc deficiency on children's behavior and development, but a recent Cochrane review (Gogia & Sachdev, 2012) noted no convincing difference of zinc supplementation versus placebo in mental and motor development in infants and children younger than 5 years. Most of the research linking zinc to child development has not addressed the possibility of interactions with other micronutrient deficiencies.

The small, rapidly exchangeable pool of zinc is dependent on a steady source of zinc from the diet. The optimal source of zinc is from animal sources, such as liver, beef, lamb, and oysters, which are

also important sources of iron and vitamin $B_{12}$. This suggests that children who are zinc deficient are also likely to be deficient in iron and vitamin $B_{12}$. All three micronutrients have been associated with deficits in cognitive functioning. On the other hand, it is possible that simultaneous administration of multiple other micronutrients could interfere with zinc absorption or utilization. For example, adverse reactions between iron and zinc have been described (Penny et al., 2004). Phytates in plant foods can also reduce zinc absorption, which is important to keep in mind when feeding infants and young children soy protein sources.

### Selenium

Selenium is a trace mineral involved in the regulation of thyroid hormone metabolism, healthy immune function, antioxidant activity, and prevention of coronary artery disease. Recognition of selenium's importance in the diet has long been impeded by fear of its toxicity and potential carcinogenic effects.

The quality of biomarkers of dietary intake of selenium and selenium status is quite favorable relative to that of most trace minerals. These biomarkers are in increasing demand principally because of the antioxidant role of this micromineral. Plasma selenium responds rapidly to selenium supplementation and is regarded as a biomarker of short-term selenium status, although in reasonably stable circumstances it also provides a biomarker of long-term intake. Whole blood selenium is of potential value as a biomarker of relatively long-term intake and status. Hair selenium has also been used to assess selenium status. Selenium homeostasis is regulated by excretion via the kidneys with increased intakes, resulting in increased urinary metabolites (Hendler & Rorvik, 2008).

A limited number of studies identified children with severe neurodevelopmental delays and elevated liver function tests after developing intractable seizures during the first year of life. At the time of seizures, evidence of systemic selenium deficiency was documented. Findings support the hypothesis that the presence of selenium depletion in the brain among patients with epilepsy constitutes an important trigger of intractable seizures and neuronal damage (Ramaekers, Calomme, Berghe, & Makropoulos, 1994). Keshan disease (characterized by cardiomyopathy) and Kashin-Beck disease (characterized by osteoarthropathy, joint destruction, and dwarfism) are both associated with selenium deficiency (Erdman et al., 2012).

The amount of selenium found in foods is based on the concentration of selenium in the soil in which they were grown. Because of the uneven distribution of selenium in the soil throughout the world, disorders of deficiency and excesses have been reported. China, for example, is known to have the lowest and highest concentrations of soil selenium in the world. Reported adverse reactions to toxic levels of selenium are skin rash, brittle hair and nails, garlic-like breath, irritability, nausea, and vomiting (Kleinman et al., 2011).

### Iodine

Iodine is required for the production of thyroid hormones, which are necessary for normal brain development and cognition. Iodine deficiency is the main cause of potentially preventable mental deficit in childhood and causes goiter and hypothyroidism in people of all ages. Environmental iodine deficiency causes a wide spectrum of devastating mental and physical disorders, collectively described as iodine deficiency disorders, which are a major public health problem all over the world (Chandria, Tripathy, Ghosh, Debnath, & Mukhopadhyay, 2005). Although endemic goiter is the most visible consequence of iodine deficiency, the most significant and profound consequences are on the developing brain. Congenital hypothyroidism and endemic iodine deficiency are common causes of mental retardation (Simsek, Karabay, Safak, & Kocabay, 2003). Cretinism is the most severe form of neurologic damage resulting from fetal hypothyroidism. The characteristics of cretinism are gross mental retardation, short stature, deaf mutism, and spasticity (Zimmermann, Jooste, & Pandev, 2008).

Impaired intellectual development of people living in iodine-deficient regions is of particular concern, especially when all the adverse effects can be prevented by long-term sustainable iodine prophylaxis. Information processing, fine motor skills, and visual problem solving are improved by iodine repletion in moderately iodine-deficient school children (Zimmermann et al., 2006). Adverse effects of supplementation are generally minor and transient, including adolescent acne, rashes, arrhythmias, numbness, weakness in the hands, hypothyroidism, hyperthyroidism, parotitis (iodide mumps), and small bowel lesions. Children with cystic fibrosis appear to have an exaggerated susceptibility to the goitrogenic effect of high doses of iodide. High-quality controlled studies investigating long-term outcome measures are needed to address the best form of iodine

supplementation in different population groups and settings (Angermayr & Clar, 2004).

The mean serum thyroid-stimulating hormone is increased in iodine deficiency, although absolute values may remain within the normal range. Thyroid function tests provide useful functional biomarkers of longer term iodine intake and iodide status. Urinary iodine is the standard biochemical indicator used worldwide that shows current state of iodine nutrition. It is also used as a valuable indicator for the assessment of iodine deficiency disorders because 90% of the body's iodine is excreted through the urine (Chandria et al., 2005; Hambridge, 2003). Urine iodine excretion reflects intake within the past few days.

Percutaneous absorption of topically applied substances and the potential for systemic toxicity are important considerations in the child. Most cases of percutaneous drug toxicity have been reported in newborns, although cases in infants and young children have also been noted. Iodine-containing compounds such as povidone-iodine have long been used for topical antisepsis, and it has been suggested they may carry a significant risk to infants. Percutaneous toxicity is of greatest concern in the premature infant, in whom immaturity of the epidermal permeability barrier results in disproportionately increased absorption. Immature drug metabolism capabilities may further contribute to the increased risk in this population. Elevations in both plasma and urinary iodine have been documented in premature infants exposed to these agents. The potential for transient hypothyroxinemia and hypothyroidism has been a concern of many clinicians. This concern stems from the known risks of abnormal thyroid function in the infant, including growth and motor retardation, cognitive delay, and intraventricular hemorrhage. The surface area of the treated skin seems to correlate directly with the risk (Mancini, 2000). Currently, the use of chlorhexidine for skin preparation over povidone-iodine is the more common practice that reduces the risk of percutaneous toxicity with iodine (O'Grady et al., 2011). In addition to iodized salts, rich sources of iodine include fish and sea vegetables. Iodine is also available in animal products such as eggs, milk, meat, and poultry.

### Chromium

Chromium is believed to be an essential trace mineral in human nutrition. Evidence suggests that it plays an important role in normal carbohydrate metabolism. The mechanism of chromium's possible glucose regulatory activity is not well understood. It has also been suggested that chromium may decrease hepatic extraction of insulin and improve glucose tolerance. It has been found that patients receiving long-term total parenteral nutrition without chromium develop glucose intolerance, weight loss, and peripheral neuropathy. These symptoms reverse when given intravenous chromium chloride (Hendler & Rorvik, 2008). Suggestive but poorly substantiated evidence that chromium deficiency may be widespread, especially in individuals with some degree of glucose intolerance, points to a need for reliable biomarkers of dietary chromium and chromium status. It has been concluded that plasma chromium is unlikely to offer a useful indicator in part because normal values are so near the limit of detection. Data conflict on the relationship of urine chromium excretion to dietary intake. Only one study, in which controlled quantities of dietary chromium were given, provided some evidence for deterioration in glucose tolerance with a severely chromium-restricted diet (Hambridge, 2003).

The efficiency of absorption of chromium from inorganic or organic compounds is minimal at approximately 2%. Chromium is distributed to various tissues in the body but appears to have a preference for bone, spleen, liver, and kidney. Most of the ingested chromium is excreted in the feces. Chromium that has been absorbed is excreted mainly in the urine. There is much that remains unknown regarding the pharmacokinetics of chromium. Dietary intake is approximately 25 mg/day, with the recommended adequate daily dietary intake being 20 to 150 mg for children younger than age 7 years (Hendler & Rorvik, 2008).

Good food sources of chromium include liver, whole grains, cereals, mushrooms, and brewer's yeast. Fruits and vegetables are generally poor sources of chromium, as are most refined foods.

## Children with Disabilities

*Harriet H. Cloud, MS, RD, FADA*

Increasing numbers of children with developmental disabilities enter the healthcare system each year. It is reported by the Centers for Disease Control and Prevention (CDC, 2003) that 17% of all children younger than 18 years of age have some type of developmental disability. This is the result of improved care for many disorders that were once fatal and the development of new management

techniques. Many of the changes apparent today began in the 1960s as a result of legislation that brought children with developmental disabilities into the educational and healthcare systems. Nutrition is an important component of the care of these children in treating their chronic diseases but also in the prevention of poor growth, obesity, gastrointestinal disorders, feeding problems, and metabolic problems. In 2010, the Academy of Nutrition and Dietetics (American Dietetic Association [ADA], 2010a) published a position paper stating the need for nutrition services being available for all age groups with developmental disabilities.

## Definitions

The Developmental Disabilities Assistance and Bill of Rights Act defines a developmental disability as a severe chronic disability of a person that is attributable to a mental or physical impairment or combination of mental and physical impairments. Characteristically, a developmental disability

- Manifests before age 22 years
- Is likely to continue indefinitely
- Results in substantial functional limitations in three or more of the following areas of major life activity: self-care, receptive and expressive language, learning, mobility, self-direction, capacity for independent living, and economic self-sufficiency
- Reflects the individual's need for a combination and sequence of special interdisciplinary or generic services, supports, or other assistance that is of lifelong or of extended duration

When applied to the younger population, ages 0 to 5 years, the term means the probability of resulting in developmental disabilities if services are not provided (Developmental Disabilities Assistance and Bill of Rights Act, 2002).

*Intellectual disability* is defined as a substantial limitation in present functioning. It is characterized by significantly subaverage intellectual function existing concurrently with related limitations in two or more areas: communication, self-care, home living, social skills, and community use; self-direction; health and safety; functional academics; leisure; and work. The term *children with special healthcare needs* is used to describe children who have or are at increased risk for a chronic physical, developmental, behavioral, or emotional condition and who require health and related services of a type or amount beyond that required by children generally.

## Etiology and Incidence

The etiologies of developmental disabilities are multifactorial. They can include **chromosomal aberrations** such as **Down syndrome** (trisomy 21), neuro-logic insults in the prenatal period, **congenital anomalies**, prematurity, infectious diseases, untreated inborn errors of metabolism, trauma, neural tube defects (such as spina bifida), and other **syndromes** of lesser incidence. The incidence of the various conditions included under the umbrella of developmental disabilities varies. It is estimated, for example, that Down syndrome has an incidence of 1 in 600–800 live births, whereas spina bifida has an incidence of 1 in 1,000. The CDC (2003) reported that 17% of children younger than 18 years of age have some type of developmental disability. Other surveys reported that 3 million to 4 million Americans have a developmental disability and another 3 million have a milder form of cognitive disability or intellectual disability (ADA, 2010a).

**chromosomal aberration** A change in the makeup of a chromosome, often leading to a developmental disability.

**Down syndrome** An aberration of the 21st chromosome that causes mental retardation, low muscle tone, and other physical abnormalities.

**congenital anomalies** Malformations present at birth that can affect various organs or structures of the body, such as a cleft lip or palate.

**syndromes** A term used to identify a developmental disability with a cluster of distinctive features, such as Down syndrome.

## Nutrition Considerations

The nutritional needs of the child with developmental disabilities are the same as those for a nonaffected child from the standpoint of energy needs, nutrients, and fluids. The needs may vary depending on a particular syndrome involving growth in height, energy needs being lower or greater, and metabolic factors. As a result, there are nutritional risk factors such as growth deficiency, obesity, gastrointestinal disorders, feeding problems, and drug–nutrient interaction problems.

As a result of varying nutritional needs, nutrition considerations include an assessment of growth and energy needs, feeding issues (such as oral-motor problems), developmental delays of feeding skills,

and behavioral problems. Other areas of nutrition consideration include drug–nutrient interaction, constipation, dental caries, urinary tract infections, allergies, and food or nutrition misinformation related to various types of developmental disabilities. These issues are addressed separately under each condition.

### Nutrition Assessment

Assessment of the child with developmental disabilities includes anthropometric measures, biochemical measures when indicated by a particular syndrome, dietary intake, and inclusion of an evaluation of feeding development. Anthropometric measures can be extensive or are limited in most clinical settings to height, weight, head circumference, arm circumference, and triceps skinfold measures. Although a number of growth charts exist for specific conditions and syndromes, the CDC recommends using the current CDC charts. Information for accurately measuring the child with disabilities and the limitations of the specific use of the growth curves can be found on the CDC website (www.cdc.gov).

Obtaining special equipment for determining height and weight is often necessary for accurate assessment. Weight-measuring devices may include chair scales, bucket scales, or wheelchair scales. Obtaining height for the nonambulatory individual requires either a recumbent board or alternative measures such as arm span, knee height, or sitting height.

### Biochemical Assessment

Laboratory assessment of the child with developmental disabilities is generally the same as for the nonaffected child, with the exception of individuals who are receiving medication. Children with seizures or epilepsy receiving anticonvulsant medications are at risk for low blood levels of folic acid, carnitine, ascorbic acid, calcium, vitamin D, alkaline phosphatase, phosphorus, and pyridoxine. Assessment of thyroid status is recommended for children with Down syndrome and a glucose tolerance test for Prader-Willi syndrome (PWS).

### Dietary Information and Feeding Assessment

Dietary information may be difficult to obtain for children with developmental disabilities, just as it is for nonaffected children related to the various environments where children are fed. Diet histories obtained from the parent can be helpful in revealing diet progression and texture issues that may exist. Often, written diaries are required.

Many children with developmental disabilities display feeding problems that decrease their ability to eat an adequate diet. The problems may include oral motor difficulties, positioning problems, sensory issues, tactile resistance post intubation, and conflict in parent–child relationship (Cooper-Brown et al., 2008). Evaluation of the feeding process is an important component of nutrition assessment for children with developmental disabilities because difficulties may result in poor weight gain, decreased immunity, poor growth, anemia, and mineral and vitamin deficiencies. The problems should be assessed with an understanding of the normal development of feeding and the physical makeup of the mouth and pharynx (Cloud, Ekvall, & Hicks, 2005). Collaboration with other disciplines such as occupational therapy, physical therapy, and speech pathology is often indicated.

Once the nutrition assessment has been completed, problems should be identified and priorities set for addressing them. This information should be shared with the parents and other disciplines working with the child before the intervention process begins. For the child in an early intervention program (ages 0 to 3 years), the nutrition intervention plan should become part of the individualized family plan. If the child is of school age, the nutrition component should be a part of the individualized education plan.

Although interventions vary depending on the particular condition, general rules should govern all interventions. These include programs that are comprehensive, family centered, culturally appropriate, and community based. A second consideration is the need for follow-up, either by the dietitian or another healthcare professional, and it is important to clarify information provided and to answer questions. A third consideration involves the cost of the nutrition therapy required and possibly finding resources for special nutrition products if indicated.

## Chromosomal Aberrations

### Down Syndrome

Down syndrome (also called trisomy 21) is a chromosomal aberration involving chromosome 21 that results in the presence of an extra chromosome in

each cell of the body. There is an incidence of 1 in 600–800 live births, and there are three processes by which this anomaly can occur: nondysfunction, translocation, and mosaicism. In nondysfunction, the chromosome (21) fails to separate before conception and the abnormal gamete joins with a normal gamete at conception to form a fertilized egg with three of chromosome 21. Distinguishing features include short stature, congenital heart disease, mental retardation, **hypotonia**, hyperflexibility of the joints, upward slant of the eye, Brushfield spots (speckling of the iris of the eye), epicanthal folds, small oral cavity, and short broad hands with the single palmar crease, and a wide gap between the first and second toes. The National Down Syndrome Congress developed a chart of health concerns that should be addressed by those providing care to children with Down syndrome, and many have nutritional concerns (available at www.ds-health.com/health99.htm).

**hypotonia** Low tone of the muscles frequently found in Down syndrome, Prader-Willi syndrome, and other conditions such as prematurity.

Growth in children with Down syndrome differs markedly from that of nonaffected children. Typically, growth is at the third percentile for height when compared with the general population. Birth weights can be in a normal range of greater than 2,500 g; however, longitudinal follow-up indicates a sloping off of height after the child is 15 months of age. As a result of the differences in height from the general population, special growth curves have been developed. Cronk and associates (1988) developed Down syndrome curves in the 1970s using a population of children with Down syndrome followed over time. Children with heart defects, one of the common findings in Down syndrome, were included. Studies completed in Sweden (Myerlid, Gustafsson, Ollars, & Anneren, 2002) led to the development of growth-specific curves as an aid to diagnosing celiac disease and hypothyroidism. The growth charts were developed from longitudinal and cross-sectional data from 4,532 examinations of 354 males and females from 1970 to 1977. The mean birth length in both sexes was 48 cm, and a final height of 161.5 cm for boys and 147.5 cm for girls ages 15 and 16 years was found. Average height at age 16 years developed by Cronk and colleagues (1988) for girls with Down syndrome was 145 cm and for boys, 155 cm. The Swedish study found that European boys with Down syndrome are taller than corresponding American boys, although European girls with Down syndrome have similar height to corresponding American girls. Specific growth curves have now been developed in the United Kingdom, Saudi Arabia, and Japan. Currently, researchers at the Children's Hospital of Philadelphia are measuring children with Down syndrome from birth to age 21 years to provide updated growth curves (Children's Hospital of Philadelphia, 2010).

Various factors have been listed as contributing factors for less growth in height, including congenital heart disease, hypotonia, hypothyroidism, and hip dysplasia. Growth hormone (GH) therapy has become a clinical option for children with Down syndrome, but research studies are in the beginning stages. Annerén and associates (2000) compared the use of GH therapy between PWS and Down syndrome and found the mean height of Down syndrome children changing from –1.7 to –0.8 standard deviations. The mean height of the control group fell from –1.7 to 2.2 standard deviations. One of the conclusions of this study was that GH is not recommended for children with Down syndrome who have not been diagnosed with GH deficiency. Although height increased in the study, there was no change in head circumference, gross motor development, or mental development. There was some improvement in fine motor development.

### Energy Needs

Children with Down syndrome have a high prevalence of obesity related to their metabolic rate, lowered gross motor activity, low muscle tone, and poor dietary practices. Recent studies on the energy needs of Down syndrome children are limited. Luke, Roizen, Sutton, and Schoeller (1994) investigated the relationship between energy expenditure and obesity in a study exploring body composition, resting metabolic rate, and total energy expenditure for 13 prepubescent children with Down syndrome and in 10 control subjects matched for age, weight, and percentage of fat. Indirect calorimetry and doubly labeled water were used. Measurement of resting metabolic rate was complicated by excessive movement by both the Down syndrome subjects and the control subjects. The investigators calculated a corrected resting metabolic rate and found the value significantly lower in the Down syndrome children than in the control group when expressed as a percentage of the World Health Organization basal metabolic rate: 79.5% ± 10.4% and 96.8% ± 7.8%, respectively. No significant differences were detected in daily energy expenditure or nonresting metabolic rate expenditure between the subject groups.

### Down Syndrome Research

Whitt-Glover, O'Neill, and Stettler (2006) investigated physical activity in Down syndrome children and their siblings between 3 and 10 years of age. Results of this study, which used accelerometers for 7 days, were that the Down syndrome children accumulated less vigorous-intensity activity than did their siblings ($p = .04$) and for shorter periods of time ($p < .01$). Child feeding practices were also evaluated (O'Neill, Shults, Stallings, & Stettler, 2005) to find their relationship to weight status among children with Down syndrome and their unaffected siblings. The study included 36 children with Down syndrome and 36 children without Down syndrome between 3 and 10 years of age. A child feeding questionnaire was completed assessing six aspects of control in feeding. Anthropometric measures were completed with height and weight and calculation of a body mass index (BMI) score and the BMI z-score. Mean BMI z-scores were higher for the children with Down syndrome than for their siblings. Parents' perceptions of feeding included greater use of restriction, greater feelings of responsibility for feeding and concern about child weight status, and lower pressure for the child with Down syndrome to eat than for their siblings. It was concluded that differences in child feeding practices may play a role in the development of obesity in Down syndrome.

In contrast to the work of Luke and colleagues (1994), a more recent study of adults with Down syndrome (Fernhall et al., 2005) comparing the basal metabolic rate of 22 individuals with Down syndrome to 20 nondisabled control subjects of similar age (25.7 and 27.4 years, respectively) showed no differences in basal metabolic rate. One older study was completed to investigate energy intake related to body composition in the Down syndrome population (Luke, Sutton, Schoeller, & Roizen, 1996). The researchers measured nutrient intake and body composition in prepubescent children with Down syndrome and 10 children without Down syndrome. The subjects with Down syndrome were significantly shorter than the control subjects, body composition did not differ between the groups, and reported energy intake was low in subjects with Down syndrome, at less than 80% of the Recommended Dietary Allowance. The investigators concluded that the tendency for obesity in the Down syndrome child should be treated with a balanced diet with no energy restriction and increased physical activity.

#### Nutrient Needs

Numerous studies have shown biochemical and metabolic abnormalities in individuals with Down syndrome; however, many have involved small samples and were difficult to interpret (Capone, Muller, & Ekvall, 2005). Although serum concentrations of albumin have been found to be low, the guidelines from the Down Syndrome Medical Congress do not list serum albumin assessment as routine. Increased glucose levels have been reported, with one study reporting an increased incidence of diabetes mellitus (Van Goor, Massa, & Hirasing, 1997).

A number of studies looked at zinc, copper, and selenium status, with some reporting reduced zinc and selenium levels and conflicting plasma levels of copper (Ani, Grantham-McGregor, & Muller, 2000; Lima, Cardoso, & Cozzolino, 2010). These studies, along with multiple studies involving vitamin A, carotene, and vitamin D, concluded that these deficiencies do not exist. Recent studies in Venezuela found that vitamin A deficiency was a public health issue for children with Down syndrome 2–6 years of age (Chávez et al., 2010). Studies involving vitamin E reported decreased concentrations in Down syndrome (Shah & Johnson, 1989). It is suggested that trisomy 21 predisposes to increased oxidative stress and the increased use of antioxidants protect against the stress, thus the low levels of vitamin E, an antioxidant. This theory of oxidative stress has led parents of individuals with Down syndrome to purchase many vitamin supplements with the expected outcome of improved cognitive ability and growth. At this point, there is no conclusive evidence that supplementation is effective.

#### Dietary Intake

During infancy the food intake of the infant with Down syndrome may differ from that of the normal infant. Although human breast milk is recommended, many infants with Down syndrome are formula-fed. Pisacane and colleagues (2003) found that of 560 children with Down syndrome, 57% were formula-fed versus 24% of nonaffected infants. The main reasons reported by the mothers were infants' illness and admission to the neonatal unit, frustration or depression, perceived milk insufficiency, and difficulty in suckling by the infant. It has been this author's experience that the problems with sucking and latching onto the breast have been the deciding factors in the use of formula rather than breastfeeding, although use of the pump is an option.

Progression to solid food has been found to be delayed in children with Down syndrome, mostly because of delays in feeding and motor development (Hopman et al., 1998). Introduction of solid food may not be offered at 4 to 6 months if the infant has poor head control or is not yet sitting up. Low tone and sucking problems also delay weaning from the breast or bottle to the cup. Early intervention programs include feeding and feeding progression instruction and practice.

### Feeding Skills

Feeding skills are delayed in the infant and child with Down syndrome. Some parents found difficulty in initiating oral motor skills such as suckling and sucking. The infant with Down syndrome often has difficulty in coordinating sucking, swallowing, and breathing, the foundation for early feeding. When the infant has a congenital heart defect, which occurs in 40% to 60% of infants with Down syndrome, sucking is weakened and fatigue interferes with the feeding process. Gastrointestinal anomalies are found in 8% to 12% of infants with Down syndrome, and these infants often require nasogastric or gastrostomy feedings.

#### Special Olympics Serves Diversity

Special Olympics is a program for children and adults with special needs that now reaches 2,256,733 participants across the world. Areas involved include Africa, Asia Pacific, East Asia, Europe/Eurasia, Latin America, Middle East, and North America.

More than 67% of the participants are younger than age 22 years. Sports training and athletic competitions constitute the core of the organization's activities and include athletics, basketball, football, bowling, and aquatics. The global Special Olympics movement began in July 1968 when the first international games were held in Chicago. The concept for Special Olympics began in 1962 through the efforts of Eunice Kennedy Shriver when she started a day camp for people with intellectual disabilities at her home. Through the efforts of the Kennedy Foundation and her leadership, Special Olympics began and grew. Many associations such as the American Dental Association and the Academy of Nutrition and Dietetics are involved in the various competitions, providing evaluations and nutrition education.

Other physical factors that make feeding difficult in the first years of life include midfacial hypoplasia, small oral cavity, small mandible, delayed or abnormal dentition, malocclusion, nasal congestion, small hands, and short fingers (Cooper Brown et al., 2008). Weaning and self-feeding are usually late when compared with the nonaffected infant and frequently do not emerge until 15 to 18 months of age. Infants with Down syndrome strive for independence and autonomy about 6 months later than children without Down syndrome do.

### Nutrition Problems and Intervention Weight Status

The most effective intervention for the overweight child with Down syndrome is to design a well-balanced diet without energy restriction, with vitamin and mineral supplementation, and with increased physical activity (Luke et al., 1996). Dietary management includes assessing the feeding developmental level of the child, working with a physical therapist related to gross motor skills to determine possible activity levels, and making environmental changes. Environmental changes should include following a regular eating schedule of three meals at regular times with the child sitting either in a high chair or at the table and planned low-fat and low-sugar snacks, matching the child's feeding development. Soft drinks should be drastically limited, and milk should be low fat (after age 2 years). Physical activity should be encouraged. Counseling with the parent helps determine a realistic plan that focuses on serving sizes and food preparation and decreases the number of times meals are purchased in fast food restaurants. If the child or adolescent is school age, a special meal at school can be obtained by using the school food service prescription (see the box titled "Community Resources").

#### Community Resources

For many types of nutrition problems and medical nutrition therapy, the school system is an excellent resource through the school lunch and school breakfast programs (Cloud, 2001). Children and adolescents may receive modified meals at school. Child and Adult Care Food Programs must provide meals at no extra cost for children and adolescents with special needs and developmental disabilities. School food service is required to offer special meals at no additional cost to children whose disabilities restrict their diets as defined in the U.S. Department of Agriculture's nondiscrimination regulations. The term *child with a disability* under Part B of the Individuals with Disabilities Education Act (IDEA) refers to a child evaluated in accordance with IDEA as having 1 of the 13 recognized disability categories: (1) autism; (2) deaf–blindness; (3) deafness; (4) mental retardation; (5) orthopaedic impairments; (6) other health impairments caused by chronic or acute health problems such as asthma, nephritis, diabetes, sickle cell anemia, a heart condition, epilepsy, rheumatic fever, hemophilia, leukemia, lead poisoning, or tuberculosis; (7) emotional disturbances; (8) specific learning disabilities; (9) speech or language impairment; (10) traumatic brain injury; (11) visual impairment; (12) multiple disabilities; and (13) developmental delays. Attention deficit disorder may fall under 1 of the 13 categories (USDA Food and Nutrition Service, 2001).

When a referral is made to the school system for a special meal related to a developmental disability, it must be accompanied by a medical statement for children with special dietary needs, for example, children with severe allergies or inborn errors of metabolism. The availability of school food service for children with developmental disabilities is an important resource in the long-term implementation of medical nutrition therapy (McCrary, 2006).

Other community-based programs legally required to serve children with special needs and that include a nutrition services mandate include Part C of IDEA for the Early Intervention (0–3) part of the act, Head Start (required to include 10% of their clients with developmental disabilities), and the Special Supplemental Nutrition Program for Women, Infants, and Children. Funding for nutrition services for this population can be a problem because these services are not funded for individual clients through Medicaid (ADA, 2010b). In 1997, the Children's Health Insurance Program (CHIP) was enacted to provide funding to states to deliver health insurance to children and families with incomes too high to qualify for Medicaid and too low to afford private health insurance. Almost all states now have CHIP programs in operation (ADA, 2010b).

### Feeding Skills

Often, parents erroneously expect different feeding development for the child with Down syndrome. Behavioral problems related to feeding usually develop based on what happens between the parent and child at mealtime. An example of this is the unnecessary delay of weaning to a cup or avoidance of progression of food textures because of inadequate effort or education needed to enable this in children with Down syndrome. During intervention programs, the feeding team can guide the parent in positioning the child and working toward attainable feeding skills related to the developmental level of the child. Close attention should be paid to feeding and the development of self-feeding skills.

### Constipation

This is a frequent problem for children with Down syndrome because of overall low tone, followed by lack of fiber and fluid in the diet. Treatment should involve increasing fiber and fluid, with water consumption emphasized. Fiber content of the diet for children after age 3 years is 5 to 6 g plus year of age per day. For adults the recommendation is 25 to 30 g dietary fiber/day.

## Prader-Willi Syndrome

**Prader-Willi syndrome (PWS)** A genetic condition caused by an absence of material from the 15th chromosome. Characteristics include developmental delays, low motor tone, and an insatiable appetite.

**Prader-Willi syndrome (PWS)** was first described in 1956 by Drs. Prader, Willi, and Lambert. It is a genetic condition caused by the absence of chromosomal material from chromosome 15. PWS occurs with a frequency of 1 in 10,000–25,000 live births. Characteristics of the syndrome include developmental delays, poor muscle tone, short stature, small hands and feet, incomplete sexual development, and unique facial features. Insatiable appetite leading to obesity is the classic feature of PWS; however, in infancy the problem of hypotonia interferes with feeding and leads to failure to thrive (McCune & Driscoll, 2005).

### GH Research

GH therapy was approved by the U.S. Food and Drug Administration in 2000. In a 5-year study in Japan (Obata, Sakazume, Yoshino, Murakami, & Sakuta, 2003), 37 children with PWS from ages 3 to 21 years were evaluated for height velocity, final height, BMI, and Röhrer index. After 1 year of treatment, the mean height velocity improved significantly from 4.32 to 8.69 cm/yr ($p < 0.0001$). After 5 years, the mean standard deviation score increased from −0.99 to + 0.88 ($p = 0.003$). A U.S. study (Carrel et al., 2004) investigated body composition and motor development in 29 infants and toddlers with PWS aged 4 to 37 months who were randomized for GH treatment for 12 months. The GH-treated subjects, compared with control subjects, demonstrated decreased percentage of body fat ($p < 0.001$), increased lean body mass ($p < 0.001$), and increased height velocity z-scores ($p < 0.001$). Children who started GH treatment before 18 months of age showed higher mobility skill acquisition compared with control subjects in the same age range ($p < .05$). These data have encouraged the use of GH in children with PWS.

The genetic basis of PWS is complex. Individuals with PWS have a portion of genetic material deleted from chromosome 15 received from the father. Seventy percent of the cases of PWS are caused from the paternal deletion, occurring in a specific region on the q arm of the chromosome. PWS can also develop if a child receives both chromosome 15s from the mother. This is seen in approximately 25% of the cases of PWS and is called maternal uniparental disomy. Early detection of PWS is now possible with the use of DNA methylation analysis, which can correctly diagnose 99% of the cases (McCune & Driscoll, 2005). This is an important development in the early identification and subsequent treatment of these children to prevent obesity and growth retardation. It is selected for use at birth for the infant born with the features and characteristics described previously (McCune & Driscoll, 2005).

### Metabolic Abnormalities

Short stature in the individual with PWS has been attributed to GH deficiency. In addition to decreased GH release, children have low serum insulin-like growth factor-I, low insulin-like growth factor binding protein-1, and low insulin compared with normal obese children.

In addition to GH deficiency, individuals with PWS have a deficiency in the hypothalamic-pituitary-gonadal axis causing delayed and incomplete sexual development. Finally, there is a decreased insulin response to a glucose load in children with PWS compared with age-matched obese children without PWS (Schuster, Osei, & Zipf, 1996).

### Appetite and Obesity

Appetite control and obesity are common problems with individuals with PWS. After the initial period of failure to thrive, children begin to gain excessively between the ages of 1 and 4 years, and appetite is excessive. This uncontrollable appetite, a classic feature of PWS, when combined with overeating, a low basal metabolic rate, and decreased activity, leads to the characteristic obesity. The cause of the uncontrollable appetite involves the hypothalamus

and the parvocellular oxytocin neurons, which are decreased in the brains of those afflicted with PWS (Swaab, Purba, & Hofman, 1995). Other hormones and peptides related to appetite control in animals were not found to be increased in the hypothalamus of individuals with PWS (Goldstone, Unmehopa, Bloom, & Swaab, 2002). Developmental delays (affecting 50% of the population), learning disabilities, and mental retardation (affecting 10%) are associated with PWS.

Body composition is an important consideration in the evaluation of individuals with PWS. They have abnormal body composition, decreased lean body mass, and increased body fat, even in infancy. In a study of 16 infants (Bekx, Carrel, Shriver, & Allen, 2003), the percentage of body fat was significantly increased ($p < 0.001$) and the percentage of fat-free mass significantly decreased in both male and female subjects ($p \leq 0.001$, $p = 0.04$) with decreased energy expenditure. The conclusion was that the lower energy expenditure is caused by the decreased fat-free mass. Body fat is generally deposited in the thighs, buttocks, and abdominal area. The lowered energy expenditure is also found in young children, adolescents, and adults with PWS. One study found adolescents with PWS having a total energy expenditure 53% of that of nonaffected obese adolescents (McCune & Driscoll, 2005). The low muscle tone contributes greatly to the lack of interest in physical activity.

### Nutrition Assessment

#### *Anthropometrics*

As stated earlier, height measurements tend to be lower in infants and young children with PWS, with the rate of height gain tapering off between the ages of 1 and 4. The usual measurements of length or height, weight, and head circumference should be taken and plotted on the CDC growth curves. Other measures of interest include arm circumference and triceps skinfold measures. BMI may be distorted for the individual with PWS because of short stature; however, plotting the BMI over time is useful in determining unusual changes. It is important that anthropometric measures are done frequently and reported to the parents or caregiver.

#### *Biochemical Measures*

Biochemical studies are generally the same for the PWS individual with the exception of either fasting blood glucose tests or glucose tolerance tests. These are added because of the risk for diabetes mellitus, possibly related to the obesity that usually accompanies PWS.

#### *Dietary Intake*

Dietary information varies for individuals with PWS depending on their age. In infancy, the dietary information should be obtained with a careful dietary history and analyzed for energy and nutrient intake. Infants are commonly difficult to feed because of their hypotonia, poor suck, delayed motor skills, and failure to thrive. Breastfeeding has been limited as a result of their sucking difficulties. Generally, their feeding development is slower than in normal infants, and transitioning to food at 4 to 6 months of age may be difficult. Many of these infants have gastroesophageal reflux that requires medication or thickening of their formula. During the toddler years, weight gain may increase rapidly as intake increases. Appetite may increase around 1 year of age and continue. This requires careful assessment of portion sizes, frequency of feeding, and types of foods served. Although some parents may report that the child with PWS does not eat more than other children in the family, they need to be informed that energy needs are lower because of the reduced lean muscle mass and slow development of motor skills and activity. As the children get older their interest in food increases, and starting around 5 years through 12 years of age they may be hungry all the time and display difficult behaviors such as tantrums, stubbornness, and food stealing. Many parents have found it necessary to lock cabinets, refrigerators, and the kitchen door to control food intake (Dimitropoulos, Feurer, Butler, & Thompson, 2001). Information gathered during the dietary interview should include environmental control techniques.

Whitman, Myers, Carrel, and Allen (2002) investigated the impact of GH therapy on behavior problems that have been attributed to the appetite and compulsive desire for food. Previous studies were reported to improve alertness, activity level, endurance, irritability, tendency to worry, and extroversion, resulting in better personal relationships. This study included 54 children with PWS from ages 4 to 16 years. Children with previous GH therapy were excluded as were children with scoliosis greater than 20 degrees. Behavior was monitored at 6-month intervals using the Oxford Survey Diagnostic Instrument, which is a 165-item behavioral checklist that was modified to include 10 items specifically designed for PWS.

No differences were found between treatment and control groups for attentional symptoms, anxiety, violence, or psychotic symptoms. A significant positive effect (reduction of depressive symptoms) was noted for the treatment group. The study conclusions were that GH therapy contributes to behavioral improvement, an important outcome related to programs designed to prevent or treat obesity in this population.

Determination of energy needs for the infant with PWS is the same as for a normal infant. However, as the child enters the toddler years he or she needs fewer calories to maintain weight gain along the growth curve. This also applies in adulthood, when fewer calories are needed to maintain weight. Energy needs have been calculated according to centimeters of height from 2 years on. The macronutrient intake of the diet should be 25% protein, 50% carbohydrate, and 25% fat.

#### *Feeding Skills*

Infants with PWS often present with weak oral skills and poor sucking skills in the first year of life. As children mature feeding skills are not a problem, but they may be delayed. Chewing and swallowing problems are not widely found, although they may be associated with low muscle tone. Behavioral feeding issues are associated with an insatiable appetite and not being provided with food. As stated earlier, this can bring about tantrums.

#### *Intervention Strategies*

Intervention for PWS involves several age stages: infancy, toddler and preschool age, school age, and adult. In infancy, providing adequate nutrition as established by the AAP related to breastfeeding or formula feeding is recommended. Because feeding may be difficult related to sucking, concentrating the formula or breast milk may be necessary to promote adequate weight gain. Feeding intervention assists in improving the sucking problems caused by hypotonia. As the infant matures, a concentrated formula is not necessary, and foods can be added when head control and trunk stability are achieved, usually around 4 to 6 months.

Most toddler and preschool age children begin to gain excessive weight between 1 and 4 years of age. Beginning a structured dietary protocol for the child and the family is important so the toddler learns that meals are provided at specified times and a pattern of grazing does not develop. Parents should be taught to provide small servings of meats, vegetables, grains, and fruits and limit intake of sweets. Early intervention for these children in the preschool years is very important in working with feeding issues and intake control as they grow older. Weight, height, and nutrient intake should be monitored monthly and energy needs adjusted if weight gain becomes excessive. Concurrently, physical activity must be encouraged as a part of early intervention programs and physical therapy services made available if necessary.

For the school age child, collaboration with the school food service program becomes important. Energy needs should be calculated per centimeter of height and are generally 50% to 75% of the energy needs of unaffected children. This may require using the prescription for special meals through the school food service program. At home, environmental controls may be required with locked cupboards and refrigerators because children and adolescents with PWS have limited satiety and search for food away from mealtime. Some parents say that GH therapy for their child helps, but it does not seem to change the child's lack of satiety. Appetite-suppressing medications have been used but are largely unsuccessful.

Medical nutrition therapy of children and adults with PWS requires follow-up through many healthcare providers and schools. Fortunately, parents of individuals with PWS now have access to a number of support groups and organizations dedicated to education, research, and establishing treatment programs.

## Neurologic Disorders

### Spina Bifida

**Spina bifida** is a neurologic tube defect that presents in a number of ways: meningocele, myelomeningocele, and spina bifida occulta. Myelomeningocele is the most common derangement in the formation of the spinal cord and generally occurs between 26 and 30 days gestation with the date of occurrence affecting the location of the lesion. The lesion may occur in the thoracic, lumbar, or sacral area and influences the amount of paralysis. The higher the lesion, the greater the paralysis. Manifestations range from weakness in the lower extremities to complete paralysis and loss of sensation. Other manifestations include

**spina bifida** A neurologic tube defect caused by a lesion in the spinal cord that occurs during the formation of the spinal cord.

incontinence and hydrocephalus. The incidence of spina bifida is 1 per 1,430 births (CDC, 2010), whereas the incidence of myelomeningocele is 5 per 10,000 live births in the United States. It is found more frequently in whites than in African Americans or Asian Americans and more frequently in girls than in boys, with a ratio of 1.25:1.00 (Ekvall & Cerniglia, 2005). The spinal lesion may be open and can be surgically repaired shortly after birth, usually within 24 hours to prevent infection. Although the spinal opening can be surgically repaired, the nerve damage is permanent, resulting in varying degrees of paralysis of the lower limbs. In addition to physical and mobility issues, most individuals have some form of learning disability.

Prevention of spina bifida is now possible (Stevenson, Allen, & Pai, 2000). In 1983, Smithells and coworkers published results of a multilevel study involving the preconceptional supplementation of mothers with folic acid plus multivitamins. This reduced the risk of a second pregnancy with spina bifida as an outcome. As a result of numerous studies showing folic acid supplementation before conception to be effective, the national recommendation is 400 µg/day for all women of childbearing age. These studies resulted in the addition of folic acid to many flours and other cereal and grain products in the food supply (U.S. Department of Health and Human Services & U.S. Food and Drug Administration, 1996). These public health measures have resulted in increased folic acid blood levels in U.S. women of childbearing age and a decrease of 20% in the national rate of spina bifida (Williams et al., 2005).

### Health Concerns

The spinal lesion affects many systems of the body and can result in weakness in the lower extremities, paralysis and nonambulation, poor skin condition resulting from pressure sores, loss of sensation and bladder incontinence, hydrocephalus, urinary tract infections, constipation, and obesity. Seizures occur in approximately 20% of children with myelomeningocele and require medication. Chronic medication is also required for prevention and treatment of urinary tract infections and for bladder control. The resulting nutrition problems include obesity, feeding problems, constipation, and drug–nutrient interaction problems. Children with spina bifida can become allergic to latex brought about by multiple surgeries. It is recommended that children with spina bifida avoid certain foods, such as bananas, kiwi, and avocados. Mild reactions can occur from apples, carrots, celery, tomatoes, papaya, and melons (Cloud et al., 2005).

### Nutrition Assessment

#### *Anthropometrics*

Infants and children with neural tube defects are usually shorter because of reduced length and atrophy of the lower extremities, although other problems such as hydrocephalus, scoliosis, renal disease, and malnutrition may contribute to it. The level of the lesions can also affect the length and height of the individual.

Obtaining accurate length and height measures can be difficult, especially as the child grows older. An alternate measure for determining height, the arm span to height ratio, is used and modified depending on leg muscle mass. Arm span can be used directly as a height measure (arm span × 1.0) if there is no leg muscle mass loss, as in a sacral lesion. Arm span × 0.95 can be used to determine height if there is partial leg muscle loss, and arm span × 0.90 is used for a height measurement when there is complete leg muscle loss, such as with a thoracic spinal lesion (Ekvall & Cerniglia, 2005).

Weight measures can be obtained for the child unable to stand by using chair scales, bucket scales, and wheelchair scales. In a clinical situation, weight should be obtained in a consistent manner, with the person in light clothing or undressed to obtain an accurate weight. Triceps skinfold measurements can also be used along with subscapular and abdominal and thorax measurements to determine the amount of body fat.

Head circumference should be measured in infants and toddlers up to age 3 years. A high percentage of children with spina bifida have head shunts as a result of their hydrocephalus. Unusual changes in the size of the head may indicate a problem with the shunt.

#### *Biochemical Measures*

Most protocols in the treatment of spina bifida include iron status tests, measurements of vitamin C and zinc levels, and other tests related to the nutritional consequences of medications needed for seizures and urinary tract infection control.

#### *Dietary Intake*

Many children with spina bifida eat a limited variety of foods and are frequently described as "picky eaters" by the parents. When doing a dietary history

it is important to ask about the variety of foods, particularly of high-fiber foods. The school age child may be prone to skipping breakfast because early morning preparations for school require more time than for the nonaffected child.

Energy needs are lower for the child with spina bifida, and calorie requirements must be carefully determined to prevent the obesity to which many are prone. Ekvall and Cerniglia (2005) found that for children 8 years or older with myelomeningocele, the caloric need is 7 kcal/cm of height for weight loss and 9 to 11 kcal/cm of height to maintain weight. It is important to evaluate how the mother or caregiver perceives food for the child because food represents sympathy and love for many parents.

Fluid intake is very important to evaluate because so many children have urinary tract infections and may be drinking inadequate amounts of water and excessive amounts of soft drinks, tea, and so on. Physical activity must also be evaluated and may be found to be very limited, particularly when the child is nonambulatory. Ambulatory individuals with a shunt may be restricted from contact sports but can be involved in walking and running.

Feeding skills need to be evaluated, along with oral motor function in particular. Many children with spina bifida are born with the **Arnold-Chiari malformation of the brain** that affects the brainstem and swallowing. Swallowing may be difficult and contributes to the child avoiding certain foods later in life. Because of this there may be delays in weaning from the breast or bottle to the cup, but there should be no delays in gaining self-feeding skills.

**Arnold-Chiari malformation of the brain** A structural disorder affecting the cerebellum, frequently found in spina bifida; can affect swallowing and gagging.

### *Clinical Evaluation*

This evaluation should include examination of the skin for pressure sores, along with asking about the amount and type of fluids consumed. Cranberry juice has been recommended along with vitamin C to provide a urine pH of 5.5 to 6; however, cranberry juice has been discounted by some literature reviews. Constipation may be caused by the neurogenic bowel and a diet low in fiber and fluids. The evaluation should include a review of food intake, fiber content, and fluids.

### *Intervention Strategies*

From a nutritional standpoint, many children with spina bifida have obesity as the number one problem because of the impact of other physical problems. It usually occurs when ambulation is a problem and there is a lack of awareness of energy needs coupled with a lack of exercise. Other problems include inadequate fluids and fiber and refusal to accept a wide variety of foods. Feeding is frequently a problem and can be both behavioral and oral motor. Early intervention and counseling about introducing foods around age 6 months, limiting the intake of high-sucrose infant jar foods, and training the child in accepting a wide variety of flavors and textures are important.

Obesity prevention should include addressing the problems with limited physical activity and lack of fluids and fiber and should begin with a calculation of the appropriate amount of calories and fluid. If the child is overweight, the food service manager should be provided with a prescription for a low-calorie breakfast and lunch, and weight management should be listed as a part of the individualized education plan. Enrollment in a group weight management program has been used successfully with modification of the accompanying physical exercise. The ideal program uses a team approach with involvement of the physician, dietitian, nurse, occupational therapist, physical therapist, educator, and psychologist.

In many clinics serving the child or adult with spina bifida, clients are seen on a semiannual or annual basis. This frequent follow-up is necessary and should include monitoring of growth, particularly weight; food and fluid intake; and medication use. School programs and early intervention programs are excellent follow-up sites; however, often the school lacks appropriate scales for weighing a nonambulatory student. In this situation, parents should be encouraged to bring the child to the clinic for weight checks or, if distance is a problem, to find a long-term care facility that permits using their scales. Follow-up by phone contact or email can be done for evaluating dietary intake and fluid management.

## Cerebral Palsy

**Cerebral palsy (CP)** is a disorder of motor control or coordination resulting from injury to the brain during its early development. Among the causative agents of CP are prematurity, blood type incompatibility, placental insufficiency, maternal infection that includes German measles or other viral diseases, neonatal

**cerebral palsy (CP)** A disorder of motor control or coordination resulting from injury to the brain during its early development.

jaundice, anoxia at birth, and other bacterial infections of the mother, fetus, or infant that affect the central nervous system. The problem in CP lies in the brain's inability to control the muscles, even though the muscles themselves and the nerves connecting them to the spinal cord are normal. The extent and location of the brain injury determine the type and distribution of CP. The incidence of CP varies with different studies, but the most commonly used rate is 2 to 3 per 1,000 live births. The increasing prevalence of premature births has contributed to an increase in this figure (Winter, Autry, Boyle, & Yeargin-Allsop, 2002).

Various types of CP are classified according to the neurologic signs involving muscle tone, abnormal motor patters, and postures. The diagnosis of CP is generally made between 9 and 12 months of age and as late as 2 years with some types. The various types of CP include spastic, dyskinetic, mixed, and ataxia of CP (Ekvall & Cerniglia, 2005).

### Health Concerns

Poor nutritional status and growth failure, often related to feeding problems, are common in children with CP. Meeting energy needs is particularly difficult in those children and adults with more severe forms of CP, such as **spastic quadriplegia** and athetoid CP. Assessment of the bone mineral density of children and adolescents with moderate to severe CP showed lower scores associated with gross motor function and feeding difficulty (Henderson, Kairalla, Barrington, Abbas, & Stevenson, 2005). One hundred seven participants (aged 2 years, 1 month to 21 years, 1 month) with moderate to severe spastic CP were assessed for anthropometric measures of growth and nutrition and dual energy X-ray absorptiometry measures of bone mineral density. Seventeen participants were ambulatory, and 90 had little or no ambulation. The weight z-score proved to be the best predictor of bone mineral density, an important indicator of a nutritional risk factor. Other factors related to the bone mineral density score included a history of fractures, anticonvulsant medication, and feeding difficulties.

**spastic quadriplegia** Spastic paralysis of the arms and legs.

Constipation is another health problem and may be caused by inactivity, lack of fiber and fluids, and feeding problems. Dental problems occur and are often related to malocclusion, dental irregularities, and fractured teeth. Lengthy and prolonged bottle feedings of milk and juice promote the decay of the primary upper front teeth and molars. Hearing problems and especially visual impairments, mental retardation, respiratory problems, and seizures affect nutritional status. Seizures are controlled with anticonvulsants, and a number of drug–nutrient interaction problems occur.

### Nutrition Assessment

#### *Anthropometrics*

This is an important area of assessment because of the growth failure of the more severely involved child or adult with CP. Children with CP are often shorter, and depending on the level of severity, some children with CP may need to be measured for length using **recumbent length** boards or standing boards even as they grow older. However, some of the measuring devices are inappropriate for the child with contractures and inability to be stretched out full length. Arm span can be used when the individual's arms are stretchable as well as upper arm length and lower leg length. Hogan (1999) and Stevenson (2005) recommended lower leg length or knee height as a possible measure for determining height for both children and adults with lower leg CP. Krick, Murphy-Miller, Zeger, and Wright (1996) developed growth charts for children with CP using weight and length data on 360 children; however, the CDC training module on use of the growth curves for children from birth to 20 years of age recommends using the CDC curves, designed for nonaffected children, and plotting sequentially for indications of malnutrition rather than using the disease-specific curves.

**recumbent length** Measuring the length of an individual lying down.

Stevenson and coworkers (2006) completed a six-site, multicentered, region-based, cross-sectional study of children with moderate or severe CP. There were 273 children enrolled (71% white) and the anthropometric measures included weight, knee height, upper arm length, upper arm muscle area, triceps skinfold, and subscapular skinfold. Growth curves were developed and z-scores published for each of the six measures. Pilot studies will be conducted on the validity of the growth curves before they are distributed. Current results indicated that from the growth data collected, the most positive measures were found in children with the fewest days of healthcare use and fewest days of social participation missed.

Weight measures should be collected over time. Scales may require modifications with positioning devices for the individual with CP who has

developed scoliosis, contractures, and spasticity. Working with a physical therapist to find a positioning device that can be placed in a chair scale or using a bucket scale often works well. Midupper arm circumference and triceps skinfold measures are reported by Samson-Fang and Stevenson (2000) as the recommended way to screen for fat stores in children with CP. Head circumference should be measured regularly from birth to 36 months and plotted on the CDC growth curves.

### *Biochemical Measures*

Although there are no specific laboratory values indicated for the child with CP, a complete blood count, including hemoglobin and hematocrit, should be done when food intake is limited and malnutrition is a possibility. Because bone fractures are a significant problem for many children and adults with spastic quadriplegia, bone mineral density may need to be evaluated. Medications for seizures may be given, and many have nutrition interaction problems. Evaluation of vitamin D, calcium, carnitine, and vitamin K levels may be indicated (Andrew & Sullivan, 2010).

### *Dietary Intake*

Feeding may be an important problem that limits the intake of food and fluid, and caregivers may not provide sufficient food to meet nutritional needs. The energy needs of the individual with CP vary according to the type of CP. Studies show that the resting metabolic rate and total energy expenditure are lower in those with spastic quadriplegic CP than in normal control subjects (Bandini, Schneller, Fukagana, Wykes, & Dietz, 1991; Stallings, Zemel, Davies, Cronk, & Charney, 1996). Bandini, Puelzll-Quinn, Morelli, and Fukagawa (1995) recommended that measures of energy intake should be adjusted for changes in body weight to determine energy requirements for the individual with severe CP. Stallings and associates (1996) found total energy expenditure in the child with spastic quadriplegic CP in a ratio to resting energy expenditure significantly lower in the spastic quadriplegic CP children compared with a control group. Dietary intake was markedly overreported by caregivers. Stallings and associates concluded that growth failure and an abnormal pattern of resting energy expenditure were related to inadequate energy intake.

### *Feeding Problems*

A high percentage of children with CP have feeding problems that are largely caused by oral motor, positioning, and behavioral factors. As infants, they have difficulty swallowing and coordinating swallowing and chewing, so the normal progression to solid foods is difficult. All this may lead to inadequate intake and growth limitations. Early detection of the feeding problem was demonstrated by Motion and colleagues (2001), who investigated the prevalence of feeding difficulties at 4 weeks and 6 months of age in 33 children with CP. Feeding difficulties at 4 weeks of age were associated with a pattern of functional impairment at 4 years of age and at 8 years and being clinically underweight and having speech and swallowing difficulties at 8 years. Sullivan and associates (2002) studied 100 children (mean age, 9 years) with disabilities related to the impact of feeding problems on nutritional status and growth. Ninety children had a diagnosis of CP. Results confirmed the significant impact of neurologic impairment and oral motor problems on energy intake, leading to poor growth and nutritional status.

Increasing numbers of children with CP with severe feeding problems are tube fed. This usually follows swallowing studies such as the modified barium swallow where there is video fluoroscopy indicating aspiration. Sullivan and coworkers (2005) reported a longitudinal, prospective, multicenter, cohort study designed to measure the outcomes of gastrostomy tube feeding in children with CP. The study included 57 children with CP (28 girls and 29 boys; median age 4 years, 4 months; range 5 months to 17 years, 3 months). The children were assessed before gastrostomy placement and at 6 months and 12 months. At baseline, half of the children were more than three standard deviations below the average weight for age and gender when compared with standards for normally developing children. Weight increased over the study period from a median weight z-score of –3 to –2.2 at 6 months and –1.6 at 12 months.

## CRITICAL Thinking

For those infants and children in early intervention programs, the team of dietitian, speech therapist, occupational therapist, and physical therapist should evaluate the feeding problems and work together in planning therapy. Providing an appropriate formula requires the dietitian to evaluate the caloric and nutritional values of the product selected, determine the amount needed, and work with the parent in obtaining insurance funding. Working out an intervention plan is most successful when it involves the parent as part of the team, addresses cultural issues, and recognizes the importance of the feeding problem (Spiker, Hebbeler, Wagner, Cameto, & McKenna, 2004).

Children with CP have complex problems that require follow-up with the family and in the community and take time to correct. Agencies within the state provide tube-feeding formulas, special wheelchairs, and equipment to assist with feeding problems. Early involvement of a skilled multidisciplinary team is essential to reduce the impact of feeding difficulties and improve nutritional intake and growth (Andrew et al., 2012).

## Autism

Autism is one of five disorders under the category of pervasive developmental disorder (PDD). The term *PDD* was first used in the 1980s to describe a class of disorders: autistic disorders, Rett disorder, childhood disintegrative disorder, Asperger disorder, and PDD not otherwise specified (American Psychiatric Association, 1994). In general, children with PDD have a neurologic disorder usually evident by age 3 years characterized by difficulty in talking, playing with other children, and relating to others, including family.

**autism spectrum disorders (ASDs)** A number of disorders that involve poor social interaction, impaired communication skills, a tendency to be repetitive, and sometimes mental retardation; found more in males than in females.

**Autism spectrum disorders (ASDs)** affect 3.4 per 1,000 live births (Yeargin-Allsopp et al., 2003) and are diagnosed by the presence of qualitatively impaired reciprocal social interaction, impaired communication skills, and restricted, repetitive, stereotypical interests and behaviors. Many children with autism also have mental retardation. Autistic disorder is four times more common in boys than in girls. Asperger syndrome is most often used to describe children with the problems of ASDs but who have normal to high cognitive level (Autism Society of America,1995).

ASDs may occur with other developmental or physical disabilities. They have been associated with tuberous sclerosis, maternal rubella, and mental retardation. Macrocephaly has been a common finding in large surveys of individuals with autism and also among their relatives. Overall growth is usually normal and medical problems nonexistent. It is possible that with the limited variety of foods usually eaten by these children vitamin and mineral intake could be inadequate.

Efforts to find the cause of ASDs have led to many studies to look at a possibly toxic environment, toxic food, a nutritionally deficient diet, immune system problems, oxidative stress, and emotional stress as important factors. Other studies have studied neurotransmitters such as elevated serotonin levels and disturbances in gamma-aminobutyric acid receptors, glutamate transmitters, and cholinergic activity.

Research studies are needed to find a major link among heredity and neuropathology and autism. Some treatment and research programs are using genomic panels to identify specific intervention protocols. The genomic panel identifies single nucleotide polymorphisms that are identified from blood samples or cell cultures. This work has revealed that the child with autism may need additional essential fatty acids; nutrients with antioxidant qualities such as vitamins A, C, and E and selenium; mineral supplementation with zinc, calcium, and magnesium; a mercury-free diet; or an allergy elimination diet.

Interest in a neurochemical cause of ASDs was started in 1979 when Jaak Panksepp proposed that ASDs simulated brain opioid dysfunction. Earlier studies discovered a unique urinary peptide pattern in adults with ASDs and hypothesized that brain opioids came from an exogenous source. Gluten and casein were the suspected sources, and in the 1980s researchers found these urinary peptides in the urine and cerebrospinal fluid of autistic individuals (Reichelt & Knivsberg, 2003). The condition of the intestine has played a role in this theory, with constipation and diarrhea common in individuals with ASDs. Intestinal inflammation has been reported in children with ASDs and has reportedly improved with dietary restriction of gluten and casein (Knivsberg et al., 2002).

### Nutrition Assessment

#### *Anthropometrics*

Height and weight are determined for the child and adult with ASDs using the equipment and growth charts for nonaffected individuals. Head circumference should be taken and has been found to be larger than for individuals without ASDs.

#### *Biochemical Measures*

These tests vary depending on the clinic where the child is followed. There is no standard pattern of tests that should be given, other than the regular blood work for health monitoring. However, amino acid screening shortly after birth is indicated along with thyroid testing. For some children allergy testing may be indicated.

#### *Dietary Intake*

Evaluations are sometimes difficult to complete for the child with a very limited intake. An effective measure may be to have parents and caregivers keep a food diary for several days to determine the

macronutrient intake in addition to the vitamin and mineral intake. Obtaining information related to when food is presented and the amounts eaten is important along with information on fluid consumption. Often, excessive fluids are provided to compensate for limited food consumption.

Evaluations should include an observation of the child during mealtime. Some children are slow in arriving at developmental milestones for self-feeding and require feeding. Others finger feed or insist on self-feeding. The texture of the food presented should be recorded because sensory integration is difficult for children with ASDs, and they may be very resistant to texture progression or variety. This is reflected in their fixation on one food, for example, crackers, dry cereal, or chips. Fugassi, Stevens, and Ekvall (2003) found that 70% of 87 children with autism had food jags and were picky eaters. The feeding evaluation should also include a description of the feeding environment, whether there is a high chair or age-appropriate toddler chair, the timing of meals, and the location for meals.

#### *Intervention Strategies*

No one therapy or method works for all individuals with ASDs. Conventional treatments include behavior management and medications, whereas others consist of structured educational approach, speech therapy, and occupational therapy. Popular nutrition interventions include mineral and vitamin therapy, elimination diets such as the gluten-free casein-free diet, allergy identification, adding essential fatty acids, megavitamins (Lucas, 2004), and specific diets. Very little has been published to demonstrate the value of the diets, although there are anecdotal reports of success. The exclusion diets are now used in some treatment centers and are publicized on various websites. It is important for the nutrition professional to understand these various forms of therapy to counsel parents effectively. In addition, with the increasing prevalence of ASDs, research of potential medical nutrition therapy should be promoted based on evidence-based practice.

One of the problems with the gluten-free casein-free diet is cost because special foods needed to provide sufficient food choices are expensive and sometimes difficult to find. When medical nutrition therapy is used, taking a team approach and working with the occupational therapist, speech therapist, and other members of the team are important for success. Parents also should be members of the team and counseled that changes take time.

**Learning Point** Follow-up is an important component of all therapy. From a nutritional standpoint, routine measures of height and weight should be scheduled, and there should be regular evaluation of eating and feeding behavior related to increasing ability to self-feed and to accept new and different foods. Children with autism and PDD are at increased nutritional risk because of the limited variety of foods eaten, mealtime behavior problems, elimination diets, food allergy/sensitivity/intolerance, and chronic gastrointestinal disorders.

### Attention Deficit Hyperactivity Disorder

**Attention deficit hyperactivity disorder (ADHD)** is a neurobehavioral problem seen in children with increasing frequency. It has been associated with learning disorders and inappropriate degrees of impulsiveness, hyperactivity, and attention deficit. Diagnostic criteria developed by the American Psychiatric Association have designated three types: (1) combined type of hyperactivity and attention deficit, (2) predominately inattentive type, and (3) predominately hyperactive-impulse type. ADHD affects the child at home, in school, and in social situations.

**attention deficit hyperactivity disorder (ADHD)** Neurobehavioral problem associated with learning disorders, hyperactivity, attention deficit, and inappropriate degrees of impulsiveness.

#### Nutrition Assessment

Many factors should be considered along with the usual anthropometric measures, particularly when the individual is on medication.

#### *Anthropometric Measures*

Measurements of height and weight should be taken and recorded on a regular basis because the medications used in treatment may cause anorexia if given at inappropriate times, resulting in inadequate energy intake and potential slowing of growth.

#### *Biochemical Measures*

These measurements should include a complete blood count and blood and tissue levels of vitamin and minerals if megavitamin therapy is used.

#### *Dietary Intake*

A detailed dietary history should be taken to include infant feeding history, food likes and dislikes, behavior at mealtimes, snacking behavior, food allergies or food intolerances, and special diets. If the individual is on medications, the time of administration in relation to mealtime is important. Information should be obtained regarding any specific diet for the child or individual and how closely it is being followed.

Feeding evaluations should include observing the individual at mealtime. Generally, the problems

around feeding are behavioral and do not include oral motor or positioning peculiarities. Evaluating the environment around mealtime is important because distractions can be problematic.

*Intervention Strategies*

Current treatment may include psychotropic medications and following consistent behavioral management techniques. The timing and type of medication must be adjusted so there is minimal influence on the child's dietary intake.

### Special Diets

Specific diets have been used for many years, but they are not based on scientific research. For example, parents have been advised to use the Feingold diet (Feingold, 1974), which states that foods containing synthetic food colors and naturally occurring salicylates be removed from the diet because of their neurologic effect. Other recommendations have included the elimination of sugar, the elimination of caffeine, or the addition of large doses of vitamins (megavitamin therapy). A series of well-designed studies to evaluate the effectiveness of these recommendations has generally had negative results, and successful outcomes are largely anecdotal (Lucas, 2004).

For the child or adult who is up and down throughout the meal, behavior modification may be indicated, and it should be a part of the overall behavioral management program. Distractions should be eliminated.

The most effective treatment for the individual with ADHD is a diet based on wholesome foods as outlined in the *Dietary Guidelines for Americans, 2010* or MyPlate. The food should be served at regular times, with small servings followed by refills. This is an important concept because of the tendency of the child or individual to eat very small amounts and leave the table, planning to return or graze throughout the day. Some programs recommend removing the food and returning it only once after explaining why this is being done. The intervention requires that the child or individual sit at the table in the high chair away from television or other distractions. These suggestions are most applicable to children in preschool settings and in the school cafeteria or classroom.

It has been suggested that a lack of essential fatty acids is a possible cause of hyperactivity in children. It is more likely the result of varying biochemical influences. These children have a deficiency of essential fatty acids either because they cannot metabolize linoleic acid normally, cannot absorb essential fatty acids effectively from the intestine, or their essential fatty acid requirements are higher than normal. Older studies showed lower levels of docosahexaenoic acid and arachidonic acid in children with hyperactivity, which have been replicated in more recent studies (Burgess, Stevens, Zhang, & Peck, 2000). Recent studies have shown that omega-3 fatty acid supplementation provides a small but significant benefit in the treatment of ADHD; however, the efficacy of pharmacological agents is greater (Bloch & Qawasmi, 2011).

### Controversial Nutrition Therapies

An important factor in providing medical nutrition therapy for children with developmental disabilities is realizing that counseling may have been inadequate in helping the parent accept the limitations of the disorder. These limitations may include growth, feeding, and cognitive ability. As a result, many parents look for unusual medical or nutritional therapies. A major source of information is often the Internet and parent support groups. Recent media coverage has promoted the use of antioxidant vitamins (A, C, and E) and minerals (zinc, copper, manganese, and selenium) along with the amino acids, glucosamine, tyrosine, and tryptophan. The expected outcome is improved growth; increased cognition, alertness, and attention span; and changed facial features.

There is little scientific information to back these therapies. Research studies have addressed the vitamin needs of children with Down syndrome, spina bifida, fragile X syndrome, and ASDs and findings do not indicate that the vitamin and mineral needs of these child with developmental disabilities are higher than normal (Ani et al., 2000; Salman, 2002). Numerous historical studies (Bennett, McClelland, Kriegsmann, Andrus, & Sells, 1983) have searched for nutritional deficiencies as causative factors in Down syndrome. Recently, Chávez and coworkers (2010) evaluated the vitamin A status of 83 children and identified the prevalence of vitamin A deficiency in 18%. Lima and associates (2010) evaluated zinc status in 35 children with Down syndrome with zinc concentrations in the plasma and urine and found them to be significantly lower than the control group. Both studies were completed in South America. Traditionally, the studies have included looking at numerous vitamins, minerals, fatty acids, digestive enzymes, lipotropic nutrients, and numerous drugs with no definitive results.

The key concept in the proposed nutritional interventions for Down syndrome is metabolic correction of genetic overexpression. It is postulated that presence of the third chromosome 21 causes overproduction of superoxide dismutase and cystathionine beta-synthase, which disrupts active methylation pathways. Vitamin supplements of antioxidants counteract this and are considered key to the treatment. However, these are just theories, and at this point nutritional supplements are considered an expensive questionable approach.

Parents of children with ADHD report that omitting sugar from the children's diets decreases hyperactivity, but there is no scientific evidence to support this (MTA Cooperative Group, 1999). However, it probably is a good idea to eliminate or at least reduce the sugar intake in any child's diet to promote better nutritional intake. Blue-green algae have been promoted for children with Down syndrome and other developmental disabilities, and monitoring is part of the initiation of these treatments. High-dose supplementation of vitamin $B_6$ and magnesium has been proposed for autism to diminish tantrums and self-stimulation activities and to improve attention and speech (Martineau, Barthelemy, Cheliakine, & Lelord, 1988). Another proposed treatment is dimethyl glycine. Limited research is available to substantiate anything other than anecdotal reports of success (Cornish, 2002).

## Summary

Generally, healthy infants and toddlers can achieve recommended levels of vitamins and minerals from food alone. Dietetic professionals should encourage caregivers to use foods rather than supplements as the primary source of nutrients in a child's diet. Vitamin and mineral supplements can help infants and toddlers with special nutrient needs or marginal intakes achieve adequate intakes, but care must be taken to ensure that supplements do not lead to excessive levels. This is especially important for nutrients that are widely used as food fortifiers, including vitamin A, zinc, and folate.

Despite the success of child survival programs, approximately 12 million children younger than 5 years old in developing countries still die of preventable causes, half of them of diarrheal diseases and respiratory infections. It is becoming clear that a large portion of the risk of infectious disease morbidity and mortality attributed to malnutrition may result primarily from deficiencies of a few critical micronutrients. Actions needed to control micronutrient deficiencies include prevention strategies, extensive nutrition, and health education through innovative materials to support program-specific problems and strengthening of various state government programs. Interventions to reduce infant and preschool morbidity are a public health priority globally.

The nutritional needs of individuals with developmental disabilities are unique because of differences in body composition, growth, metabolic functions, physical activity, medications, and behavioral issues. Nutrition is an important component of preventing developmental disabilities from occurring; however, more research is needed to identify the role of nutrition as part of the etiology of developmental disorders along with effective intervention strategies.

## Case Study 1

### A Toddler with Cystic Fibrosis

Alison Mallowe, MA, RD, LDN

Lilly is a 3-year-old who was diagnosed with cystic fibrosis (CF) at 3 weeks of age through newborn screening, positive sweat test, genetic testing (mutations: two copies of *deltaF508*), and a fecal elastase (confirming pancreatic insufficiency). At her last CF Center Clinic visit 3 months ago, she weighed 12 kg and her length was 92 cm. Her weight reflects a loss of 700 g since her last clinic visit. Lilly plots in the 10th percentile weight-for-age and 10th–25th percentile height-for-age. Her BMI is 14.1, which plots in the 5th–10th percentile for age. She is showing signs and symptoms of malabsorption, including the following: loose, greasy stools; more frequent stools than her baseline; and complaints of abdominal pain that correlate with eating. She does take pancreatic replacement enzymes (PERT) with her meals and snacks. Lilly is also complaining of nausea and dizziness after playing outside.

At the visit, the dietitian obtained information about oral intake using a 24-hr recall. Lilly was consuming approximately 1,250 calories, 25 g of protein, 900 mL of noncaffeinated fluids, and not adding salt to her foods. Further discussion revealed that Lilly's caregivers had recently left the bottles of enzymes in the car on an extremely hot day. Lilly mentioned that the enzymes melted together; however, the caregivers peeled them apart and are still using those enzymes.

The registered dietician assessed multiple issues including the following:

- Weight loss and current BMI in the 5th percentile (The Cystic Fibrosis Foundation recommends a BMI percentile greater than or equal to the 50th percentile.)
- Malabsorption
- 24-hr recall (Those affected with CF may need 150–200% more calories than those without CF.)
- Inadequate beverage and sodium intake
- Inappropriate enzyme storage

#### Questions

1. Why would Lilly still be losing weight if her current intake meets the RDA for kilocalories for her age?
2. Is Lilly consuming adequate fluid for her age? Is she consuming enough sodium?
3. Were the enzymes stored appropriately?
4. Why does the Cystic Fibrosis Foundation set the goal for BMI at or above the 50th percentile?

## Case Study 2

### Vitamins and Minerals

Sharon Collier, RD, LD, M Ed

A 17-month-old male presents with wheezing and is transferred from the local hospital to Children's Hospital in status asthmaticus. During the transfer, his heart rate dropped and had a question of a seizure activity. He was born full term, appropriate for gestational age. He has a history of multiple food allergies. He has not been seen by his pediatrician since his 12-month well-baby check-up. He does not walk.

His nutrition history is as follows: He was breastfed for the first year of life. His parents noted a rash over his body after he ate some ice cream. Mom changed her diet slightly to exclude cheese. She ate all varieties of grains, fruits, vegetables, and meats while breastfeeding.

Solids were introduced at 4 months of age with rice cereal but because of constipation was changed to oatmeal with good tolerance; pears and applesauce were added with good tolerance. When bananas were added he had a rash, and with subsequent trials of foods (chicken, squash, and infant soy formula) he reacted with a rash that was localized to his face and particularly his mouth, so his parents wondered whether it was a contact reaction.

He was seen by an allergist once; blood tests were done and his mom was given a can of elemental infant formula. Mom reports he had a rash with multiple trials and also he didn't like the taste. She did not introduce wheat, nuts, or eggs. Mom feels milk is the biggest offender because he reacted with hives, emesis, and wheezing. The patient has not been on any vitamins or minerals.

His current diet consists of the following: rice milk mixed with cereal, boiled rice with chopped carrots; apple juice mixed half and half with water—about 24–32 oz/day.

Labs

| | | | |
|---|---|---|---|
| Zinc | 26 (60–120) | Vit D | 11 (20–57) |
| Hgb | 8.9 | PTH | 100 (10–65) |
| Hct | 25 | $B_{12}$ | 140 (126–717) |
| MCV | 89 | Folate | 38 (59–202) |
| MCHC | 357 | Ferritin | 388 (10–75) |
| Fe | 152 (50–120) | Magnesium | 1.4 (2–2.5) |

Allergy testing showed high reaction to milk, tree nuts, peanuts, wheat, oats, and eggs. Chest X-ray showed rickets, poor mineralization, and flaying of humeral head.

Growth Parameters
Weight: 6.8 kg ≪3 percentile Z-score: −5.14
Length: 74 cm < 3 percentile Z-score: −2.43
Head circumference: 45 cm 3 percentile Z-score: −2.05
Ideal wt for lt: 9.4 kg < 3 percentile Z-score: −5.23
Ideal lt for age: 81 cm
Birth weight: 3.86 kg
Birth length: 53.3 cm
2 months wt: 5.45 kg
9 months wt: 8.1 kg
12 months wt: 8.1 kg

Current growth parameters are indicative of severe wasting, stunting, and micronutrient deficiency. Identification and attention to both clinical and biochemical presentation of micronutrient deficiencies and the manifestations that developed are important in how the patient is treated. It is important to address the acute deficiencies and growth delay; however, potential long-term consequences of his malnourished status must also be considered.

**Questions**

1. Based on his presentation, which vitamins and minerals are deficient?
2. What is your assessment of this toddler's nutritional status?
3. What is your assessment of his growth and development status?
4. Explain how each of the vitamins and mineral deficiencies contributed toward his poor nutritional status and growth status.
5. What potential long-term repercussions can be anticipated as results of these deficiencies?
6. What foods would you recommend to correct the deficiencies that also stay within the restrictions of his multiple food allergies? What supplements, if any?

## Case Study 3

### Developmental Disabilities (Down Syndrome)

Harriet H. Cloud, MS, RD, FADA

MP is a 23-month-old boy with Down syndrome who was seen in an early intervention program. He was referred to a dietitian for feeding problems. His history indicates that he was born at 30 weeks gestation with a birth weight of 3 lb 9 oz and birth length of 15¼ inches. Feeding problems at birth consisted of a very weak suck, severe gastroesophageal reflux, and poor weight gain. A gastrostomy tube was placed at 10 days of age and the baby formula supplemented with iron was given. He was referred to the early intervention program at 4 months of age and services were provided by nutrition, speech therapy, physical therapy, occupational therapy, and special education professionals.

The clinical concerns are as follows:

1. Gastric esophageal reflux was a continuous problem although treated with medication. At 7 months of age fundoplication surgery was completed.
2. Respiratory problems were treated with medications as well.
3. Constipation occurred and was treated with lactulose. Many of MP's problems were related to extreme hypotonia even for Down syndrome. The hypotonia also contributed to delays in gross motor development.
4. MP's growth was typical for children with Down syndrome, and although a major family concern, he more than tripled his birth weight (16 lb). He also grew 10 inches in length during the first year. Tube feeding intake was adequate for 100 kcal/kg of body weight, but at 7 months of age (corrected to 4.5 months for prematurity) no food had been introduced for oral intake, which was permissible because head control was good. At that time, cereal, strained fruit, and oral formula intake was started along with the tube feeding. Consumption of baby food was never greater than one jar per day. At 16 months, 1 kcal/cc became the tube feeding at the rate of 21 oz/day with discontinuation of the pump at night and some by bottle. Table food was introduced, but acceptance was poor.
5. Feeding problems identified were weak suck and swallow, gagging on food before the fundoplication surgery, tube feeding with progression to oral feeding difficult resulting from refusal to drink formula and other fluids, and poor appetite resulting from tube feeding.

**Question**

1. What interventions can the medical team make to improve this situation for MP?

## Case Study 4

### Developmental Disabilities (Prader-Willi Syndrome)

Harriet H. Cloud, MS, RD, FADA

LR is an infant with PWS, diagnosed shortly after birth and first evaluated by a dietitian at age 2 months. He had a birth weight of 7 lb 5 oz and birth length of 20 inches. Typical of infants with PWS, LR was very hypotonic and had a weak suck and micrognathia. These problems interfered with his ability to breastfeed, so his mother pumped her milk and supplemented with formula.

LR's intake at 2 months of age consisted of 9 oz of breast milk and 9 oz of baby formula for a total intake of 360 cal and 9 g of protein. His weight was 8 lb 1.7 oz at the 3rd percentile with his length of 22.5 inches at the 10th percentile. His intake was approximately 100 kcal/kg. Both parents were extremely concerned with the prevention of obesity and not providing too many calories.

Feeding was observed during the first visit, and the weak suck was very apparent along with leakage from the sides of the mouth. Positioning was inappropriate for promoting good sucking and better head and trunk control.

**Question**

1. What recommendations could be made by the medical team?

## Issues to Debate

1. Growth charts exist for a number of syndromes such as Down syndrome, CP, PWS, and many others. It has been the position of the CDC that the CDC curves are more appropriate for everyone to use instead of the special growth charts. How would you debate this issue?
2. Many children with developmental disabilities have feeding problems that affect their intake of an adequate diet. Under the regulations for school food service and IDEA, the feeding problem would be a part of the child's individualized education plan. Debate the issue of where the intervention should take place: in the school lunch room or in the classroom?
3. Discuss the issue of the most appropriate site for physical activity or physical education for children with developmental disabilities. Often, this is not adequately addressed in the school system and as a result children may receive no physical education.

## References


Adams, J. B., George, F., & Audhya, T. (2006). Abnormally high plasma levels of vitamin $B_6$ in children with autism not taking supplements compared to controls not taking supplements. *Journal of Alternative and Complementary Medicine, 12*, 59–63.

Ahmed, M., & Jenkins, H. R. (2004). Vitamin $B_{12}$ in Crohn's disease patients with small bowel surgery. *Archives of Disease in Childhood, 89*, 293.

American Dietetic Association. (2010a). Position of the American Dietetic Association: Providing nutrition services for infants, children, and adults with developmental disabilities and special health care need. *Journal of the American Dietetic Association, 110*, 296–307.

American Dietetic Association. (2010b). Position of the American Dietetic Association: Child and Adolescent Nutrition Assistance Programs. *Journal of the American Dietetic Association, 110*, 791–799.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

Andrew, M. J., & Sullivan, P. B. (2010). Growth in cerebral palsy. *Nutrition in Clinical Practice*, *23*(4), 357–361.

Andrew, M. J., Parr, J. R., & Sullivan, P. B. (2012). Feeding difficulties in children with cerebral palsy. *Archives of Disease in Childhood*, *97*(6), 222.

Angermayr, L., & Clar, C. (2004). Iodine supplementation for preventing iodine deficiency disorders in children. *Cochrane Database of Systematic Reviews*, (2), CD003819.

Ani, C., Grantham-McGregor, S., & Muller, D. (2000). Nutritional supplementation of Down syndrome: Theoretical considerations and current status. *Developmental Medicine and Child Neurology, 42*, 207.

Annerén, G., Tuveno, T., & Gustafsson, J. (2000). Growth hormone therapy in young children with Down syndrome and a clinical comparison of Down and Prader-Willi syndrome. *Growth Hormone and IGF Research, 10*(Suppl. B), S87–S91.

Atkinson, S. A. (2001). Special nutritional needs of infants for prevention of and recovery from bronchopulmonary dysplasia. *Journal of Nutrition, 131*, 942S–946S.

Autism Society of America. (1995). *Asperger's syndrome information package*. Bethesda, MD: Author.

Bandini, L. G., Puelzll-Quinn, H., Morelli, J. A., & Fukagawa, N. K. (1995). Estimation of energy requirements in persons with severe central nervous system impairment. *Journal of Pediatrics, 126*, 828.

Bandini, L. G., Schneller, D. A., Fukagana, N. K., Wykes, L., & Dietz, W. H. (1991). Body composition and energy expenditure in adolescents with cerebral palsy or myelodysplasia. *Pediatric Research, 29*, 70–77.

Barnerias, C., Saudubray, J. M., Torati, G., De Lonlay, P., Dulac, O., Ponsot, G., . . . Desguerre, I. (2010). Pyruvate dehyrogenase complex deficiency: Four neurological phenotypes with differing pathogenesis. *Developmental Medicine and Child Neurology*, *52*, e1–9.

Bekx, M. R., Carrel, A. L., Shriver, T. C., & Allen, J. B. (2003). Decreased energy expenditure is caused by abnormal body

composition in infants with Prader-Willi syndrome. *Journal of Pediatrics, 143*, 372–376.
Bennett, F. C., McClelland, S., Kriegsmann, E., Andrus, L., & Sells, C. (1983). Vitamin and mineral supplementation in Down syndrome. *Pediatrics, 72*, 707–713.
Berry, R., Mullinare, J., & Hamner, H. C. (2009). Folic acid fortification: Neural tube defect risk reduction—global perspective. In L. Bailey (Ed.), *Folate in health and disease* (2nd ed., pp. 179–204). Boca Raton, FL: CRC Press.
Berseth, C. L., Van Aerde, J. E., Gross, S., Stolz, S., Harris, C. L., & Hansen, J. W. (2004). Growth, efficacy and safety of feeding an iron-fortified human milk fortifier. *Pediatrics, 114*, 699–706.
Bidder, R. T., Gray, P., Newcombe, R. G., Evans, B. K., & Hughes, M. (1989). The effects of multivitamins and minerals on children with Down syndrome. *Developmental Medicine and Child Neurology, 31*, 532–537.
Black, M. M. (2003). Evidence linking zinc deficiency with children's cognitive and motor functioning. *Journal of Nutrition, 133*, 1473S–176S.
Black, M. M., Sazawal, S., Black, R. E., Khosla, S., Kumar, J., & Menon, V. (2004). Cognitive and motor development among small-for-gestational-age infants: Impact of zinc supplementation, birth weight, and caregiving practices. *Pediatrics, 113*, 1297–1305.
Bloch, M. H., & Qawasmi, A. (2011). Omega-3 fatty acid supplementation for the treatment of children with attention-deficit hyperactivity disorder symptomatology: Systematic review and meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(10), 991–999.
Botto, L. D., Mulinare, J., & Erickson, J. D. (2003). Do multivitamin or folic acid supplements reduce the risk for congenital heart defects? Evidence and gaps. *American Journal of Medical Genetics*, *121A*, 95–101.
Brown, K. H., Peerson, J. M., Rivera, J., & Allen, L. H. (2002). Effect of supplemental zinc on the growth and serum zinc concentrations of prepubertal children: A meta-analysis of randomized controlled trials. *American Journal of Clinical Nutrition, 75*, 1062–1071.
Burgess, J. R., Stevens, L., Zhang, W., & Peck, L. (2000). Long chain poly unsaturated fatty acids in children with attention-deficit hyperactivity disorder. *American Journal of Clinical Nutrition, 71*(Suppl.), 327–330.
Capone, G., Muller, D., & Ekvall, S. W. (2005). Down syndrome. In S. Ekvall & V. Ekvall (Eds.), *Pediatric nutrition in chronic disease and developmental disorders*. New York, NY: Oxford University Press.
Carrel, A. L., Moerchen, V., Myers, S. E., Bekx, M. T., Whitman, B. Y., & Allen, D. B. (2004). Growth hormone improves mobility and body composition in infants and toddlers with Prader-Willi syndrome. *Journal of Pediatrics, 145*, 744–749.
Carter, R. D., Jacobsen, J. L., Burden, M. J., Armony-Sivan, R., Dodge, N. C., Angelilli, M. L., . . . Jacobson, S. W. (2010). Iron deficiency anemia and cognitive function in infants. *Pediatrics*, *126*, e427–434.
Cashman, K. D. (2005). Vitamin K status may be an important determinant of childhood bone health. *Nutrition Reviews, 63*, 284–289.
Castillo-Lancellotti, C., Tur, J., & Uauy, R. (2013). Impact of folic acid fortification of flour on neural tube defects: a systematic review. *Public Health Nutrition, 16*(5), 901–911.
Centers for Disease Control and Prevention. (2003). Developmental disabilities. Retrieved from www.cdc.gov/ncbddd/developmentaldisabilities/index.html
Centers for Disease Control and Prevention. (2010). Spina bifida. Retrieved from www.cdc.gov/ncbddd/spinabifida/facts.html
Chakravarty, I., & Sinha, R. K. (2002). Prevalence of micronutrient deficiency based on results obtained from the national pilot program on control of micronutrient malnutrition. *Nutrition Reviews, 60*(5 Pt. 2), S53–S58.
Chandria, A. K., Tripathy, S., Ghosh, D., Debnath, A., & Mukhopadhyay, S. (2005). Iodine nutritional status and prevalence of goiter in Sundarban delta of South 24—Parganas, West Bengal. *Indian Journal of Medical Research, 122*, 419–424.
Chaudhuri, A. (2005). Why we should offer routine vitamin D supplementation in pregnancy and childhood to prevent multiple sclerosis. *Medical Hypotheses, 64*, 608–618.
Chávez, C. J., Ortega, P., Leal, J., D'Escrivan, A., González, R., & Miranda, L. E. (2010). Vitamin A deficiency and nutritional status in patients with Down's syndrome. *Anales de pediatría (Barcelona)*, *72*(3), 185–190.
Children's Hospital of Philadelphia. (2010). CHOP nutrition researchers to develop new growth charts for children with Down syndrome. Retrieved from www.chop.edu/news/new-growth-charts-being-developed-for-children-with-trisomy-21.html
Cloud, H. H. (2001). Recent trends in care of children with special needs: Nutrition services for children with developmental disabilities and special health care needs. *Topics in Clinical Nutrition, 16*, 28–40.
Cloud, H. H., Ekvall, S. W., & Hicks, L. (2005). Feeding problems of the child with special health care needs. In S. V. Ekvall & V. K. Ekvall (Eds.), *Pediatric nutrition in chronic disease and developmental disorders* (2nd ed.). New York, NY: Oxford University Press.
Commission on the Nutrition Challenges of the 21st Century. (2000). Ending malnutrition by 2020: An agenda for change in the millennium. *Food and Nutrition Bulletin, 21*, 1–88.
Cooper-Brown, L., Copeland, S., Dailey, S., Downey, D., Petersen, M. C., Simson, C., & Van Dyke, D. C. (2008). Feeding and swallowing dysfunction in genetic syndromes. *Developmental Disabilities Research Reviews*, *14*(2), 147–157.
Cornish, E. (2002). Gluten and casein free diets in autism: A study of the effects on food choice and nutrition. *Journal of Human Nutrition and Dietetics, 15*, 261–269.
Cronk, C., Crocker, A. C., Pueschel, S. M., Shea, A. M., Zackai, E., Pickens, G., & Reed, R. B. (1988). Growth charts for children with Down syndrome: 1 month to 18 years of age. *Pediatrics, 81*,102–110.
Cunningham-Rundles, S., McNeeley, D. F., & Moon, A. (2005). Mechanisms of nutrient modulation of the immune response. *Journal of Allergy Clinical Immunology, 115*, 1119–1128.
Darlow, B. A., & Graham, P. J. (2007). Vitamin A supplementation to prevent mortality and short and long term

morbidity in very low birth weight infants. *Cochrane Database of Systematic Reviews*, (4), CD000501.
Developmental Disabilities Assistance and Bill of Rights Act, 2002, Public Law 106-402.
Dhonukshe-Rutten, R. A., van Dusseldorp, M., Schneede, J., de Groot, L. C., & van Staveren, W. A. (2005). Low bone mineral density and bone mineral content are associated with low cobalamin status in adolescents. *European Journal of Nutrition, 44*, 341–347.
Dimitropoulos, A., Feurer, I. D., Butler, M. G., & Thompson (2001). Emergence of compulsive behavior and tantrums in children with Prader-Willi syndrome. *American Journal of Mental Retardation, 106*, 208–201.
Dror, D. K., & Allen, L. H. (2008). Effect of vitamin B12 deficiency on neurodevelopment in infants: Current knowledge and possible mechanisms. *Nutrition Reviews*, *66*, 250–255.
Dwyer, J. T., Butte, N. F., Deming, D. M., Siega-Riz, A. M., & Reidy, K. C. (2010). Feeding Infants and Toddlers Study 2008: Progress, continuing concerns and implications. *Journal of the American Dietetic Association*, *112*, s60–s67.
Eitenschenck, L., Armari-Alla, C., Plantaz, D., Pagnier, A., & Ducros, V. (2005). Belated decompensation of an Imerslund-Grasbeck disease. *Archives of Pediatrics, 12*, 1729–1731.
Ekvall, S. W., & Cerniglia, F. (2005). Myelomeningocele. In S. V. Ekvall & V. K. Ekvall (Eds.), *Pediatric nutrition in chronic disease and developmental disorders* (2nd ed.). New York, NY: Oxford University Press.
Erdman, J. W., Macdonald, I. A., & Zeisel, S. H. (2012). *Present knowledge in nutrition* (10th ed.). Ames, IA: Wiley-Blackwell.
Fawzi, W., Msamanga, G., Spiegelman, D., & Hunter, D. J. (2005). Studies of vitamins and minerals and HIV transmission and disease progression. *Journal of Nutrition, 135*, 938–944.
Feingold, B. F. (1974). *Why your child is hyperactive*. New York, NY: Random House.
Fernhall, B., Figueroa, A., Collier, S., Goulopoulou, S., Giannopoulou, I., & Baynard, T. (2005). Resting metabolic rate is not reduced in obese adults with Down syndrome. *Mental Retardation, 43*, 391–400.
Fisher, J. O., Mitchell, D. C., Smiciklas-Wright, H., Mannino, M. L., & Birch, L. L. (2004). Meeting calcium recommendations during middle childhood reflects mother–daughter beverage choices and predicts bone mineral status. *American Journal of Clinical Nutrition, 79*, 698–706.
Forastiere, F., Pistelli, R., Sestini, P., Fortes, C., Renzoni, E., Rusconi, F., . . . Bisanti, L. (2000). Consumption of fresh fruit rich in vitamin C and wheezing symptoms in children. SIDRIA Collaborative Group, Italy (Italian Studies on Respiratory Disorders in Children and the Environment). *Thorax, 55*, 283–288.
French, A. E., Grant, R., Weitzman, S., Ray, J. G., Vermeulen, M. J., Sung, L., . . . Koren, G. (2003). Folic acid food fortification is associated with a decline in neuroblastoma. *Clinical Pharmacology and Therapeutics, 74*, 288–294.
Fugassi, P., Stevens, F., & Ekvall, S. (2003). The characteristics of autism and nutrition in children. *American Journal of the College of Nutrition, 22*, 481.
Glenn, C. C., Driscoll, D. J., Yang, T. P., & Nicholls, R. D. (1997). Genomic imprinting: Potential function and mechanism revealed by the Prader-Willi and Angelman syndromes. *Molecular Human Reproduction, 3*, 321.
Gogia, S., & Sachedev, H. S. (2012). Zinc supplementation for mental and motor development in children. *Cochrane Database of Systematic Reviews*, (12), CD007991.
Goldstone, A. P., Unmehopa, U. A., Bloom, S. R., & Swaab, D. F. (2002). Hypothalamic NPY and agouti-related protein are increased in human illness but not in Prader-Willi syndrome and other obese subjects. *Journal of Clinical Endocrinology and Metabolism, 87*, 927.
Green, N. S. (2002). Folic acid supplementation and prevention of birth defects. *Journal of Nutrition, 132*(Suppl.), 2356S–2360S.
Greer, F. R. (2004). Issues in establishing vitamin D requirements for infants and children. *American Journal of Clinical Nutrition, 80*(6 Suppl.), 1759S–1762S.
Hambridge, M. (2000). Human zinc deficiency. *Journal of Nutrition, 130*(Suppl. 5), 1344S–1349S.
Hambridge, M. (2003). Biomarkers of trace mineral intake and status. *Journal of Nutrition, 133*, 948S–955S.
Henderson, R. C., Kairalla, J. A., Barrington, J. W., Abbas, A., & Stevenson, R. D. (2005). Longitudinal changes in bone density in children and adolescents with moderate to severe cerebral palsy. *Journal of Pediatrics, 146*, 769–775.
Hendler, S. S., & Rorvik, D. (2008). *PDR for nutritional supplements* (2nd ed.). Montvale, NJ: PDR Network.
Hobbs, C. A., Shaw, G. M., Werler, M. M., & Mosley, B. (2009). Folate status and birth defect risk. In L. Bailey (Ed.), *Folate in health and disease* (2nd ed., pp. 133–153). Boca Raton, FL: CRC Press.
Hogan, S. E. (1999). Knee height as a predictor of recumbent length for individuals with mobility-impaired cerebral palsy. *Journal of the American College of Nutrition, 18*, 201–205.
Holick, M. F., Binkley, N. C., Bischoff-Ferran, H. A., Gordon, C. M., Hanley, D. A., Heaney, R. P., . . . Weaver, C. M. (2011). Evaluating treatment and prevention of vitamin D deficiency: An Endocrine Society Clinical Practice Guideline. *Journal of Clinical Endocrinology and Metabolism*, *96*, 1–20.
Hollis, B. W., & Wagner, C. L. (2004). Assessment of dietary vitamin D requirements during pregnancy and lactation. *American Journal of Clinical Nutrition, 79*, 717–726.
Hopman, E., Csizmadia, C. G., Bastiani, W. F., Engels, Q. M., de Graaf, E. A., Cessie, S., & Mearin, M. L. (1998). Eating habits of young children with Down syndrome in the Netherlands: Adequate nutrient intakes but delayed introduction of solid food. *Journal of the American Dietetic Association, 98*, 790–794.
Huiming, Y., Chaomin, W., & Meng, M. (2005). Vitamin A treating measles in children. *Cochrane Database of Systematic Reviews, 19*(4), CD001479.
Imdad, A., Herzer, K., Mayo-Wilson, E., Yakoob, M. Y., & Bhutta, Z. A. (2011). Vitamin A supplementation for preventing morbidity and mortality in children from 6 months to 5 years of age. *Cochrane Database of Systematic Reviews*, (12), CD008524.
Institute of Medicine. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel., silicon,*

*vanadium and zinc*. Washington, DC: National Academy Press.

Institute of Medicine. (2011). *Dietary Reference Intakes for calcium and vitamin D*. Washington, DC: National Academy Press. Sazawal S

James, S. J., Cutler, P., Melnyk, S., Jernigan, S., Janak, L., Gaylor, D. W., & Neubrader, J. A. (2004). Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. *American Journal of Clinical Nutrition, 80*, 1611–1617.

Juval, R., Osmamy, M., Black, R. E., Dhingra, U., Sarkar, A., Dhingra, P., . . . Sazawal, S. (2004). Efficacy of micronutrient fortification of milk on morbidity in pre-school children and growth—a double blind randomized controlled trial. *Asia Pacific Journal of Clinical Nutrition, 13*(Suppl.), S44.

Kapil, U., & Bhavna, A. (2002). Adverse effects of poor micronutrient status during childhood and adolescence. *Nutrition Reviews, 60*(5 Pt. 2), S84–S90.

Kim, S., Kim, J., & Keen, C. (2005). Comparison of dietary patterns and nutrient intakes of elementary school children living in remote rural and urban areas in Korea: Their potential impact on school performance. *Nutrition Research, 25*, 349–363.

King, W., Levin, R., Schmidt, R., Oestreich, A., & Heubi, J. E. (2003). Prevalence of reduced bone mass in children with spastic quadriplegia. *Developmental Medicine and Child Neurology, 45*, 12.

Kleinman, A. S. (2011). Overview of Psychodermatology. *Psychopharm Review, 46*(1), 1–8.

Kleinman, R. E., & American Academy of Pediatrics Committee on Nutrition (2009). *Pediatric nutrition handbook* (6th ed.). Elk Grove Village, IL: American Academy of Pediatrics.

Krick, J., Murphy-Miller, P., Zeger, S., & Wright, E. (1996). Pattern of growth in children with cerebral palsy. *Journal of the American Dietetic Association, 97*, 680.

Knivsberg, A., Reichelt, K., Høien, T. Nødland, M. (2002). A Randomised, Controlled Study of Dietary Intervention in Autistic Syndromes. Nutritional Neuroscience, *5* (4), 251–261

Kronn, D. F., Sapru, A., & Satou, G. M. (2000). Management of hypercholesterolemia in childhood and adolescence. *Heart Disorders, 2*, 348–353.

Kwan, M. L., Block, G., Selcin, S., Month, S., & Buffler, P. A. (2004). Food consumption by children and the risk of childhood acute leukemia. *American Journal of Epidemiology, 160*, 1098–1107.

Lauer, B., & Spector, N. (2012). Vitamins. *Pediatrics in Review*, *33*, 339–351.

Lazzerini, M., & Ronfani, L. (2012). Oral zinc for treating diarrhea in children. *Cochrane Database of Systematic Reviews*, (3), CD005436.

Lefebvre, P., Benomar, Y., & Staels, B. (2010). Retinoid X receptors: common heterodimerization partners with distinct functions. *Trends in Endocrinology & Metabolism, 21*(11), 676–683.

Li, M., Eastman, C. J., Waite, K. V., Ma, G., Zacharin, M. R., Topliss, D. J., . . . Doyle, Z. (2006). Are Australian children iodine deficient? Results of the Australian National Iodine Nutrition Study. *Medical Journal of Australia, 184*, 165–169.

Libermann, S., & Branning, N. (2003). *The real vitamins and minerals book* (3rd ed.). New York, NY: Avery.

Lima, A. S., Cardoso, B. R., & Cozzolino, S. F. (2010). Nutritional status of zinc in children with Down syndrome. *Biological Trace Element Research*, *133*(1), 20–28.

Linabery, A. M., Johnson, K. J., & Ross, J. A. (2012). Childhood cancer incidence trends in association with US folic acid fortification (1986–2008). *Pediatrics*, *129*,1125–1133.

Liu, S., West, R., Randell, E., Longerich, L., O'Connor, K. S., & Scott, H. (2004). A comprehensive evaluation of food fortification with folic acid for the primary prevention of neural tube defects. *BMC Pregnancy and Childbirth, 4*, 20.

Lucas, B. (2004). Nutrition in childhood. In K. Mahan & S. E. Stump (Eds.), *Krause's food nutrition and diet therapy* (11th ed.). Philadelphia, PA: Elsevier.

Lucas, B., & Feucht, S. (1998). *The benefits of nutrition services for a case series of children with special health care needs*. Seattle: Washington State University of Human Development and Disability.

Luckasson, R., Borthwick-Duffy, S., Buntinx, W. H. E., Coulter, D., Craig, E. M., Reeve, A., . . . Tasse, M. J. (2002). *Mental retardation: Definition, classification, and systems of supports* (10th ed.). Washington, DC: American Association on Mental Retardation.

Luke, A., Roizen, N. J., Sutton, M., & Schoeller, D. A. (1994). Energy expenditure in children with Down syndrome: Correcting metabolic rate of movement. *Journal of Pediatrics, 125*(5 Pt. 1), 829–838.

Luke, A., Sutton, M., Schoeller, D. A., & Roizen, N. J. (1996). Nutrient intake and obesity in prepubescent children with Down syndrome. *Journal of the American Dietetic Association, 96*, 1262–1267.

Mahan, L. K., & Escott-Stump, S. (2008). *Krause's food, nutrition and diet therapy* (13th ed.). St Louis, MO: Saunders.

Mancini, A. J. (2000). Skin. *Pediatrics, 113*, 1114–1119.

Marcellini, M., Di Ciommo, V., Callea, F., Devito, R., Comparcola, D., Sartorelli, M. R., . . . Nobili, V. (2005). Treatment of Wilson's disease with zinc from the time of diagnosis in pediatric patients: A single-hospital 10-year follow-up study. *Journal of Laboratory and Clinical Medicine, 145*, 139–143.

Martineau, J., Barthelemy, C., Cheliakine, C., & Lelord, G. (1988). Brief report: An open middle-term study of combined vitamin $B_6$–magnesium in a subgroup of autistic children selected on their sensitivity to this treatment. *Journal of Autism and Developmental Disorders, 18*, 435–446.

Matthews, T. J. (2007). *Trends in spinal bifida and anencephaly in the United States, 1991–2005*. Hyattsville, MD: National Center for Health Statistics.

Mawson, A. R. (2009). Bone pain, growth failure, and skin rash after an upper respiratory illness in a boy with altered retinoid metabolism. *Clinical Pediatrics*, *48*, 21–25.

Merialdi, M., Caulfied, L. E., Zavaleta, N., Figueroa, A., Costigan, K. A., Dominici, F., & Dipietro, J. A. (2004). Randomized controlled trial of prenatal zinc supplementation and fetal bone growth. *American Journal of Clinical Nutrition, 79*, 826–830.

McCrary, J. M. (2006). Improving access to school based nutrition services for children with special health care needs. *Journal of the American Dietetic Association, 106*, 133–136.

McCune, H., & Driscoll, D. (2005). Prader-Willi syndrome. In S. W. Ekvall & V. K. Ekvall (Eds.), *Pediatric nutrition in chronic disease and developmental disorders* (2nd ed.). New York, NY: Oxford University Press.

Miller, A. L. (2003). The methionine-homocysteine cycle and its effects on cognitive diseases. *Alternative Medicine Reviews, 8*, 7–19.

Milne, E., de Klerk, N. H., van Bockxmeer, F., Kees, U. R., Thompson, J. R., Baker, D., & Armstrong, B. K. (2006). Is there a folate-regulated gene–environment interaction in the etiology of childhood acute lymphoblastic leukemia? *International Journal of Cancer, 119*, 229–232.

Monsen, A. L., Refsum, H., Markestad, T., & Ueland, P. M. (2003). Cobalamin status and its biochemical markers MMA and homocysteine in different age groups from 4 days to 19 years. *Clinical Chemistry, 49*, 2067–2075.

Morgan, J. (2005). Nutrition for toddlers: The foundation for good health—toddler's nutritional needs: What are they and are they being met? *Journal of Family Health Care, 15*, 56–59.

Motion, S., Northstone, K., & Emond, A. (2001). Persistent early feeding difficulties and subsequent growth and developmental outcomes. *Ambulatory Child Health*. *7*(3/4), 231.

MTA Cooperative Group. (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder: Multimodal treatment study of children with ADHD. *Journal of the American Medical Association Psychiatry, 56*(12), 1073–1086.

Mughal, Z. (2002). Rickets in childhood. *Seminars in Musculoskeletal Radiology, 6*, 183–190.

Muntjewerff, J. W., Van der Put, N., Eskes, T., Ellenbroek, B., Steegers, E., Blom, H., & Zitman, F. (2003). Homocysteine metabolism and B-vitamins in schizophrenic patients: Low plasma folate as a possible independent risk factor for schizophrenia. *Psychiatry Research, 121*, 1–9.

Murphy, S. P., Gewa, C., Liang, L., Grillenberger, M., Bwibo, N. O., & Neumann, C. G. (2003). School snacks containing animal source foods improve dietary quality for children in rural Kenya. *Journal of Nutrition, 133*, 3950S–3956S.

Myerlid, A., Gusfasson, J., Ollars, B., & Anneren, G. (2002). Growth charts for Down syndrome from birth to 18 years of age. *Archives of Disease in Childhood, 86*, 97–103.

Neumann, C. G., Gewa, C., & Bwibo, N. O. (2004). Child nutrition in developing countries. *Pediatric Annals, 33*, 658–674.

Newall, F., Barnes, C., Savoia, H., Campbell, J., & Monagle, P. (2003). Warfarin therapy in children who require long-term total parenteral nutrition. *Pediatrics, 112*(5), 386–388.

Obata, K., Sakazume, S., Yoshino, A., Murakami, N., & Sakuta, R. (2003). Effects of 5 years growth hormone treatment in patients with Prader-Willi syndrome. *Journal of Pediatric Endocrinology & Metabolism, 16*, 155–162.

O'Grady, N. P., Alexander, M., Burns, L. A., Dellinger, E. P., Garland, J., Heard, S. O., . . . Healthcare Infection Control Practices Advisory Committee (HICPAC). (2011). Guidelines for the prevention of intravascular catheter-related infections. *Clinical Infectious Diseases*, *52*(9), e162–193.

O'Neill, K. L., Shults, J., Stallings, V., & Stettler, N. (2005). Child feeding practices in children with Down syndrome and their siblings. *Journal of Pediatrics, 146*, 234–238.

Pawley, N., & Bishop, N. J. (2004). Prenatal and infant predictors of bone health: The influence of vitamin D. *American Journal of Clinical Nutrition, 80*(6 Suppl.), 1748S–1751S.

Penland, J. G. (2000). Behavioral data and methodology issues in studies of zinc nutrition in humans. *Journal of Nutrition, 130*, 147S–153S.

Penny, M. E., Marin, R. M., Duran, A., Peerson, J. M., Lanata, C. F., Lonnerdal, B., . . . Brown, K. H. (2004). Randomized controlled trial of the effect of daily supplementation with zinc or multiple micronutrients on the morbidity, growth and micronutrient status of young Peruvian children. *American Journal of Clinical Nutrition, 79*, 457–465.

Persad, V. L., Van den Hof, M. C., Dube, J. M., & Zimmer, P. (2002). Incidence of open neural tube defects in Nova Scotia after folic acid fortification. *Canadian Medical Association Journal, 167*, 241–245.

Pisacane, A., Toscano, E., Pirri, I., Continisio, P., Zoli, B., Andria, G., Zoli, B., . . . Vicari, S. (2003). Down syndrome and breastfeeding. *Acta Paediatrica, 92*, 1479–1481.

Pollitt, E. (2001). The developmental and probabilistic nature and functional consequences of iron-deficiency anemia in children. *Journal of Nutrition, 131*, 669S–675S.

Powers, H. J. (2003). Riboflavin (vitamin $B_2$) and health. *American Journal of Clinical Nutrition, 77*, 1352–1360.

Prabhu, S. S., & Dalvi, B. V. (2000). Treatable cardiomyopathies. *Indian Journal of Pediatrics, 67*(3 Suppl.), S7–S10.

Prousky, J. E. (2003). Pellegra may be a rare secondary complication of anorexia nervosa: A systematic review of the literature. *Alternative Medicine Reviews, 8*, 180–185.

Rajakumar, K., Cohen-Reis, E., & Holick, M. F. (2013). Dosing error with over the counter vitamin D supplement: A risk for vitamin D toxicity in infants. *Clinical Pediatrics*, *52*, 82–85.

Ramaekers, V. T., & Blau, N. (2004). Cerebral folate deficiency. *Developmental Medicine and Child Neurology, 46*, 843–851.

Ramaekers, V. T., Calomme, M., Berghe, V., & Makropoulos, W. (1994). Selenium deficiency triggering intractable seizures. *Neuropediatrics, 25*, 217–223.

Ray, P. E., Suga, S., Liu, X. H., Huang, X., & Johnson, R. J. (2001). Chronic potassium depletion induces renal injury, salt sensitivity, and hypertension in young rats. *Kidney International, 59*, 1850–1858.

Reichelt, K., & Knivsberg, A. M. (2003). *Why use the gluten-free and casein-free diet? What results have shown so far*. Paper presented at the Autism Research Institute Conference.

Rodriguez, A., Hamer, D. H., Rivera, J., Acosta, M., Salgado, G., Gordillo, M., . . . Sempértegui, F. (2005). Effects of moderate doses of vitamin A as an adjunct to the treatment of pneumonia in underweight and normal-weight children: A randomized, double-blind, placebo-controlled trial. *American Journal of Clinical Nutrition, 82*, 1090–1096.

Rovner, A. J., Stallings, V. A., Schall, J. I., Leonard, M. B., & Zemel, B. S. (2007). Vitamin D insufficiency in children, adolescents and young adults with cystic fibrosis despite routine oral supplementation. *American Journal of Clinical Nutrition*, *86*, 1094–1099.

Salamoun, M. M., Kizirian, A. S., Tannous, R. I., Nabulsi, M. M., Choucair, M. K., Deeb, M. E., & El-Hajj Fuleihan, G. A. (2005).

Low calcium and vitamin D intake in healthy children and adolescents and their correlates. *European Journal of Clinical Nutrition, 59*, 177–184.

Salman, M. (2002). Systematic review of the effect of therapeutic dietary supplements and drugs on cognitive function in subjects with Down syndrome. *European Journal of Paediatric Neurology, 6*, 213.

Samson-Fang, L. J., & Stevenson, R. D. (2000). Identification of malnutrition in children with cerebral palsy: Poor performance of weight for height percentiles. *Developmental Medicine and Child Neurology, 43*, 162–168.

Samour, P. Q., & King, K. (2011). *Handbook of pediatric nutrition* (4th ed.). Sudbury, MA: Jones and Bartlett.

Sazawal, S., Black, R. E., Menon, V. P., Dinghra, P., Caulfield, L. E., Dhingra, U., & Bagati, A. (2001). Zinc supplementation in infants born small for gestational age reduces mortality; a prospective, randomized, controlled trial. *Pediatrics, 108*, 1280–1286.

Sander, J., Sander, S., Steuerwald, U., Janzen, N., Michael, P., Wanders, R., . . . & Das, A.M. (2005). Neonatal screening for defects of the mitochondrial trifunctional protein. *Molecular Genetics & Metabolism, 85*(2), 108–114.

Scarfone, R. J. (1999). Use of magnesium sulfate in the treatment of children with acute asthma. *Clinical Pediatric Emergency Medicine, 1*, 6–12.

Schoeller, D. A., Levitsky, L. L., Bandini, L. G., Dietz, W. W., & Walczak, A. (1988). Energy expenditure and body composition in Prader-Willi syndrome. *Metabolism, 37*, 115.

Schuster, D. P., Osei, K., & Zipf, W. B. (1996). Characterizations of alterations in glucose and insulin metabolism in Prader-Willi subject. *Metabolism, 45*, 1514.

Shah, S., & Johnson, R. (1989). Antioxidant vitamin A and E status in children with Down syndrome subjects. *Nutrition Research, 9*, 709.

Shearer, M. J., Fu, X., & Booth, S. L. (2012). Vitamin K nutrition, metabolism and requirements: Current concepts and future research. *Advances in Nutrition*, *3*, 182–195.

Simsek, E., Karabay, M., Safak, A., & Kocabay, K. (2003). Congenital hypothyroidism and iodine status in Turkey: A comparison between the data obtained from an epidemiological study in school-aged children and neonatal screening for congenital hypothyroidism in Turkey. *Pediatric Endocrinology Reviews: PER, 1*(Suppl. 2), 155–161.

Smithells, R. N., Seller, M. J., Harris, R., Fielding, D. W., Schorah, C. J., & Wild, J. (1983). Further experience of vitamin supplementation for prevention of neural tube defect recurrences. *Lancet, 1*, 1027.

Spiker, D., Hebbeler, K., Wagner, M., Cameto, R., & McKenna, P. (2000). A framework for describing variations in state early intervention systems. *Topics in Early Childhood Special Education, 20*, 195–218.

Stallings, V. A., Zemel, B. S., Davies, J. C., Cronk, C. E., & Charney, E. B. (1996). Energy expenditure of children and adolescents with severe disabilities: A cerebral palsy model. *American Journal of Clinical Nutrition, 64*, 627–634.

Stevenson, R. D. (2005). Use of segmental measures to estimate stature in children with cerebral palsy. *Archives of Pediatrics and Adolescent Medicine, 149*, 658–662.

Stevenson, R. D., Allen, W. P., & Pai, G. S. (2000). Decline in prevalence of neural tube defects in a high risk region of the United States. *Pediatrics, 106*, 677–683.

Stevenson, R. D., Conaway, M., Chumlea, W. C., Rosenbaum, P., Fung, E. B., Henderson, R. C., . . . North American Growth in Cerebral Palsy Study. (2006). Growth and health in children with moderate to severe cerebral palsy. *Pediatrics, 118*, 1010–1018.

Sudfeld, C. R., Navar, A. M., & Halsey, N. A. (2010). Effectiveness of measles vaccination and vitamin A treatment. *International Journal of Epidemiology*, *39*(Suppl. 1), 1148–1155.

Sullivan, P. B., Juszczak, E., Bachlet, A. M., Lambert, B., Vernon-Roberts, A., Grant, H. Q., . . . Thomas, A. G. (2005). Gastrostomy tube feeding in children with cerebral palsy: A prospective longitudinal study. *Developmental Medicine and Child Neurology, 47*, 77–85.

Sullivan, P. B., Juszczak, E., Lambet, B. R., Rose, M., Ford-Adams, M. E., & Johnson, A. (2002). Impact of feeding problems on nutritional intake and growth: Oxford Feeding Study II. *Developmental Medicine and Child Neurology, 44*, 461–467.

Swaab, D. F., Purba, J. S., & Hofman, M. A. (1995). Alterations in the hypothalamic paraventricular nucleus and its oxytocin neurons (putative satiety cells) in Prader-Willi syndrome—a study of five cases. *Journal of Clinical Endocrinology and Metabolism, 80*, 573.

Svahn, J., Schiaffino, M. C., Caruso, U., Calvillo, M., Minniti, G., & Dufour, C. (2003). Severe lactic acidosis due to thiamine deficiency in a patient with B-cell leukemia lymphoma on total parenteral nutrition during high-dose methotrexate therapy. *Journal of Pediatric Hematology/Oncology, 25*, 965–968.

Tan, J. B., Tamura, T., & Stokstad, E. L. R. (1981). Availability of vitamin B6 and pantothenate in an average American diet in man. *American Journal of Clinical Nutrition*, *34*, 1328–1337.

Tanner, E. M., & Finn-Stevenson, M. (2002). Nutrition and brain development: Social policy implications. *American Journal of Orthopsychiatry, 72*, 182–193.

Taylor, E. N., Stampfer, M. J., & Curhan, G. C. (2004). Dietary factors and the risk of incident kidney stones in man: New insights after 14 year of follow up. *Journal of the American Society of Nephrology*, *15*, 3225–3232.

Tobin, S. P., Cheng, V., Schumacher, C., Barsky, D., Greis, S. M., & Siemon, J. (2005). The role of an interdisciplinary feeding team in the assessment and treatment of feeding problems. Building blocks for life. Pediatric Nutrition Practice Group, American Dietetic Association, 28, 3.

Uriu-Adams, J. Y., & Keen, C. L. (2005). Copper, oxidative stress, and human health. *Molecular Aspects of Medicine, 26*, 268–298.

U.S. Department of Agriculture, Food and Nutrition Services. (2001). Accommodating Children with Special Dietary Needs. Retrieved from www.fns.usda.gov/CND/Guidance/specialdietaryneeds.pdf

U.S. Department of Health and Human Services & U.S. Food and Drug Administration. (1996). Food standards amendment of the standards of identity for enriched grain products to require addition of folic acid. *Federal Register, 61*, 8781–8807.

Van Goor, J., Massa, G., & Hirasing, R. (1997). Increased incidence and prevalence of diabetes mellitus in Down syndrome. *Archives of Disease in Childhood, 77*, 186.

Wagner, C. L., Greer, F. R., American Academy of Pediatrics Section on Breastfeeding, & American Academy of Pediatrics Committee on Nutrition. (2008). Prevention of rickets and vitamin D deficiency in infants, children and adolescents. *Pediatrics*, *122*, 1142–1152.

Wallis, D., Ballard, J. L., Shaw, G. M., Lammer, E. J., & Finnell, R. H. (2009). Folate-related birth defects: Embryonic consequences of abnormal folate transport and metabolism. In L. B. Bailey (Ed.), *Folate in health and disease* (2nd ed., pp. 155–178). Boca Raton, FL: CRC Press.

Wang, J. Y., Wang, Y. L., Zhou, S. L., & Zhou, J. F. (2004). May chronic childhood constipation cause oxidative stress and potential free radical damage to children? *Biomedical and Environmental Sciences, 17*, 266–272.

Wharton, B., & Bishop, N. (2003). Rickets. *Lancet, 362*, 1389–1400.

Whitehead, V. M. (2006). Acquired and inherited disorders of cobalamin and folate in children. *British Journal of Haematology*, *134*, 125–136.

Whitman, B. Y., Myers, S., Carrel, A., & Allen, D. (2002). The behavioral impact of growth hormone treatment for children and adolescents with Prader-Will syndrome: A 2 year controlled study. *Pediatrics, 109*, E35.

Whitt-Glover, M. C., O'Neill, K. L., & Stettler, N. (2006). Physical activity patterns in children with and without Down syndrome. *Pediatric Rehabilitation, 9*, 158–164.

Williams, L., Rasmussen, S., Flores, A., Kirby, R., & Edmonds, L. (2005). Decline in the prevalence of spina bifida and anencephaly by race/ethnicity: 1995–2002. *Pediatrics, 116*(3), 580–586.

Winter, S., Autry, A., Boyle, C., & Yeargin-Allsop, M. (2002). Trends in the prevalence of cerebral palsy in a population based study. *Pediatrics, 110*, 1220.

Yeargin-Alsopp, M., Rice, C., Karapurkar, T., Doernberg, N., Boyle, C., & Murphy, C. (2003). Prevalence of autism in a US metropolitan area. *Journal of the American Medical Association, 289*, 49–55.

Yoshii, A., Takeoka, M., Kelly, P. J., & Krishnamoorthy, K. S. (2005). Focal status epilepticus as atypical presentation of pyridoxine-dependent epilepsy. *Journal of Child Neurology, 20*, 696–698.

Zacharin, M. (2004). Current advances in bone health of disabled children. *Current Opinion in Pediatrics, 16*, 545–551.

Ziegler, E. E., Nelson, S. E., & Jeter, J. M. (2009). Iron supplementation of breastfed infants from an early age. *American Journal of Clinical Nutrition*, *89*, 525–532.

Zimmermann, M. B., Connolly, K., Bozo, M., Bridson, J., Rohner, F., & Grimci, L. (2006). Iodine supplementation improves cognition in iodine-deficient schoolchildren in Albania: A randomized, controlled, double-blind study. *American Journal of Clinical Nutrition, 83*, 108–114.

Zimmermann, M. B., Jooste, P. L., & Pandev, C. S. (2008). Iodine deficiency disorders. *Lancet*, *372*, 1251–1262.

Zinc Investigators' Collaborative Group. (2000). Therapeutic effects of oral zinc in acute and persistent diarrhea in children in developing countries: Pooled analysis of randomized controlled trials. *American Journal of Clinical Nutrition, 72*, 1516–1522.

# CHAPTER 7

# Special Topics in School-Aged Nutrition: Pediatric Vegetarianism, Childhood Obesity, and Food Allergies

*Reed Mangels, PhD, RD, LD, Inger Stallmann, MS, RD, LD, Edna Harris-Davis, MS, MPH, RD, LD, Shideh Mofidi, MS, RD, CSP, Anne R. Lee, MS Ed, RD, Stefanie A. Giampa, MS, RD, LDN, and Ashley Smith, DVM, MS, RD, LD*



## CHAPTER OUTLINE

Case Study 2: An Underweight Vegetarian by Reed Mangels, PhD, RD, LD

Case Study 3: Vegetarian Toddler by Julia Driggers, RD, LDN, CNSC

Case Study 4: Infant Obesity by Sari Edelstein, PhD, RD

Case Study 5: School-Aged Child with Nut Allergy by Shideh Mofidi, MS, RD, CSP

Case Study 6: Childhood Food Allergy by Georgianna Walker, MS, RD, LRD

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Describe the similarities and differences between lacto-ovo-vegetarian, lacto-vegetarian, vegan, macrobiotic, fruitarian, and raw foods diets.
2. List potential health and nutritional benefits of vegetarian diets for the pediatric population.
3. Describe possible motivations for following a vegetarian diet.
4. Contrast growth of lacto-ovo-vegetarian, vegan, and nonvegetarian children and adolescents.
5. Identify key nutrients for vegetarian infants, children, and adolescents and suggest acceptable food sources for each nutrient.
6. Identify major nutritional issues for pediatric vegetarians and describe appropriate approaches for working with these issues.
7. Describe specific considerations for counseling vegetarians of various ages and their families.
8. List the growing problems related to obesity in children.
9. Describe the paradigm shift that occurred over the years to cause increased rates of obesity in children.
10. Articulate some steps that families may take to help prevent obesity in children.
11. List existing prevention strategies that could be incorporated into community programs.
12. Realize the difference between food allergy and food intolerances.
13. Comprehend the principles of diagnosis and management of food allergies.
14. Understand the use and limitations of diagnostic tests for food allergy.
15. Recognize various manifestations of food allergic disorders.
16. Understand basic principles of nutritional management of children with food allergies.
17. Discern the impact of and appreciate the role of diet in the treatment of food allergies.
18. Recognize the proper use of elimination diets and oral food challenges in the diagnostic and/or therapeutic management of food allergies.

## *Pediatric Vegetarianism*

*Reed Mangels, PhD, RD, LD*

Today, more than a million school-aged children in the United States consistently follow a vegetarian diet (Stahler, 2010) and say that they never eat meat, fish, or poultry. Many more children and adolescents eat a mostly vegetarian diet that may include limited amounts of animal products (Perry, McGuire, Neumark-Sztainer, & Story, 2002). Attitudes toward vegetarian diets for children have changed markedly. In the 1970s, vegetarian diets were labeled a form of child abuse (Roberts, West, Ogilvie, & Dillon, 1979). Compare this with the 2009 statement by the American Dietetic Association (now known as the Academy of Nutrition and Dietetics) that states, "Appropriately planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets satisfy nutrient needs of infants, children, and adolescents and promote normal growth" (Craig & Mangels, 2009).

## Types of Vegetarian Diets

It is important for clinicians to understand what a vegetarian diet is and to realize there are several different kinds of vegetarians. In the broadest sense, a **vegetarian** is a person who does not eat meat, fish, poultry, or products containing these foods (Craig & Mangels, 2009). **Lacto-ovo-vegetarians** include dairy products and eggs in their diets. **Lacto-vegetarians** include dairy products but not eggs. **Vegans** avoid eating any animal products.

**vegetarian** A person who does not eat meat, fish, or fowl or products containing those foods.

**lacto-ovo-vegetarian** A vegetarian who eats dairy products and eggs.

**lacto-vegetarian** A vegetarian who eats dairy products but not eggs.

**vegan** A vegetarian who avoids all animal products, including dairy products and eggs.

**macrobiotic** A vegetarian or near-vegetarian diet based largely on grains, legumes, and vegetables; may include limited amounts of fish.

**fruitarian** A person following a diet based on fruits, nuts, and seeds that often includes vegetables that are botanically fruits such as avocado and tomatoes.

**sea vegetables** Wild ocean plants, including nori, kelp, hijiki, and dulse, that often are purchased in dried form.

Other types of vegetarian (or near-vegetarian) diets that may be encountered include **macrobiotic**, raw foods, and **fruitarian** diets (Mangels, Messina, & Messina, 2011). Macrobiotic diets are based largely on grains along with vegetables, especially **sea vegetables**; beans; fruits; nuts; soy products; and possibly fish. A number of studies have reported serious nutrient deficiencies with long-term consequences in children following macrobiotic diets (Dagnelie, van Staveren, et al., 1989; Dagnelie et al., 1990; Dhonukshe-Rutten, van Dusseldorp, Schneede, de Groot, & van Staveren, 2005; Louwman et al., 2000). Depending on food and supplement choices, with careful planning, macrobiotic diets can be used by children (Mangels & Messina, 2001; Mangels et al., 2011). Practitioners of a raw foods diet only consume foods in their raw state. Foods used include fruits, vegetables, nuts, seeds, sprouted grains, and sprouted beans; raw dairy products may be used. Fruitarian diets are based on fruits, nuts, and seeds and often include vegetables that are botanically fruits such as avocado and tomatoes. Use of extremely restrictive diets such as raw foods and fruitarian diets has not been studied in infants and children. These diets can be very low in protein, energy, some vitamins, and minerals and cannot be recommended for infants and children (Mangels et al., 2011). Some self-described vegetarians eat fish, chicken, or even meat (Barr & Chapman, 2002; Perry et al., 2002). This can have a significant impact on food choices and nutrient intake, so individual assessment of the diets of vegetarian clients is essential.

### How Many Vegetarians Are There?

Approximately 3% of children and adolescents in the United States consistently follow a vegetarian diet, whereas about 1% consistently follow a vegan diet, and 1% follow a near-vegan diet but do eat honey, according to a Harris Interactive Poll conducted in 2010 (Stahler, 2010). Similar results were obtained in a poll conducted in 2005 (Stahler, 2005).

#### Reasons for Vegetarianism

Children and adolescents have a variety of motivations for following a vegetarian diet. Some are members of vegetarian families. Others cite an assortment of reasons, including health, animal welfare, and environmental concerns (Larsson, Ronnlund, Johansson, & Dahlgren, 2003; Worsley & Skrzypiec, 1998).

## Vegetarianism and Cultural Diversity

### Seventh-Day Adventists

**Did You Know?**

- Seventh-Day Adventists (SDAs) are a conservative religious group with more than 13 million members worldwide.
- SDAs are strongly encouraged to abstain from meat. About 27% are lacto-ovo-vegetarians, 3% are vegans, and 20% eat meat less than once a week.
- They tend to have healthy lifestyles, eating more fruits and vegetables and exercising more frequently than their neighbors.
- Fewer than 2% use any form of tobacco; fewer than 10% use alcohol and those who do generally drink only small amounts.
- SDAs have been studied extensively and have provided a wealth of information about the health advantages of a healthy lifestyle. Significant findings include
  - Lower rates of coronary disease than non-SDAs with even lower rates in vegetarian SDAs
  - Lower incidence of lung, colon, stomach, bladder, and other cancers than in non-SDAs
  - SDAs live longer than non-SDAs do; vegetarian SDAs live longer than nonvegetarian SDAs do.
  - Vegetarian SDAs weigh less than nonvegetarian SDAs.
  - Vegetarian SDAs are less likely to develop diabetes, hypertension, and arthritis than are nonvegetarian SDAs.

Data from Fraser, G. E. (2003). *Diet, life expectancy, and chronic disease: Studies of Seventh-Day Adventists and other vegetarians*. New York, NY: Oxford University Press.

#### Health Benefits of Vegetarian Diets

There are numerous health benefits for adults following a vegetarian diet, including a lower body mass index (BMI), reduced risk of cardiovascular disease, lower blood pressure and lower rates of hypertension, reduced risk of type 2 diabetes, and a lower risk for prostate and colorectal cancers

(Craig & Mangels, 2009). Less is known about benefits for vegetarian children. Vegetarian children do tend to be leaner than nonvegetarian children are (Hebbelinck & Clarys, 2001), and at least one study reported lower serum cholesterol concentrations in vegetarian children (Krajcovicova-Kudlackova, Simonic, Bederova, Grancicova, & Megalova, 1997). Positive food patterns of vegetarian children and adolescents include greater consumption of fruits, vegetables, nuts, and legumes and lower consumption of sweets, fast food, and salty snack foods (Donovan & Gibson, 1996; Larsson & Johansson, 2005; Neumark-Sztainer, Story, Resnick, & Blum, 1997; Perry et al., 2002). In addition, diets of vegetarian children and adolescents tend to be lower in cholesterol, saturated fat, and total fat and higher in fiber than diets of nonvegetarians are (Larsson & Johansson, 2002; Perry et al., 2002; Thane & Bates, 2000).

## Growth and Energy Needs of Vegetarian Children

A limited number of studies have examined growth of vegetarian children in developed countries. Many of these studies were conducted more than 20 years ago when the availability of many foods frequently used by vegetarian families, such as fortified soy milk and veggie burgers, was much less than today. Generally, studies of lacto-ovo-vegetarian school-aged children and adolescents show that their height is similar and their weight is similar to or slightly lower than those of nonvegetarians (Hebbelinck & Clarys, 2001; Nathan et al., 1997; Sabaté, Linsted, Harris, & Johnston, 1990; Sabaté, Linsted, Harris, & Sanchez, 1991). Some studies have suggested that vegetarian girls enter puberty later than nonvegetarians and therefore are shorter than similarly aged nonvegetarian girls (Kissinger & Sanchez, 1987; Sabaté, Llorca, & Sanchez, 1992), whereas other studies have not found this (Hebbelinck & Clarys, 2001). Vegan children tend to be leaner than nonvegetarian children but are typically within normal ranges of standards for height and weight (Sanders, 1988; Sanders & Manning, 1992). Markedly lower heights and weights have been reported in children following macrobiotic diets (Dagnelie, van Staveren, van Klaveren, & Burema, 1988; Dagnelie, van Staveren, Vergote, Burema et al., 1989), with lower heights persisting even after some relaxation of dietary practices (van Dusseldorp et al., 1996).

Lacto-ovo-vegetarian and vegan diets can support appropriate growth and development of children. If a child's growth rate is below what is expected, an increased energy intake may be necessary. This can be accomplished by providing concentrated energy sources, including soy products and other legumes, nuts and nut butters, and oils. In addition, some children may benefit from a somewhat lower fiber intake (Craig & Mangels, 2009).

### Nutritional Considerations

Key nutrients for vegetarian children and adolescents include protein, iron, zinc, calcium, vitamin D, vitamin $B_{12}$, and omega-3 fatty acids. In the nonvegetarian diet, these nutrients are frequently largely obtained from animal products, so questions have been raised about their adequacy in vegetarian or vegan diets. Other nutrients, including folate, vitamin C, and vitamin A (as beta-carotene), are generally not considered problematic for vegetarians because of many vegetarians' higher consumption of fruits and vegetables.

#### Protein

Protein is rarely below recommendations in vegetarian children's diets that contain adequate energy and a variety of plant foods (Dagnelie, van Staveren, Verschuren, & Hautvast, 1989; Leung et al., 2001; Thane & Bates, 2000). The Institute of Medicine (IOM, Food and Nutrition Board, 2002) concluded that the protein requirement for vegetarians who consume a variety of plant proteins is not different from that of nonvegetarians. Plant proteins contain varying amounts of essential amino acids. For example, compared with the Amino Acid Scoring Pattern developed by the Food and Nutrition Board and IOM, wheat is low in lysine (IOM, Food and Nutrition Board, 2002). Chickpeas and other legumes are relatively low in the sulfur amino acids (methionine and cysteine). By eating a variety of protein sources over the course of a day, differences in amino acid content are satisfied (Craig & Mangels, 2009; Young & Pellett, 1994).

#### Iron and Zinc

Iron intakes of vegetarian children and adolescents vary. Some studies reported iron intakes by vegetarian children that are similar to or higher than those of nonvegetarian children (Houghton et al., 1997; Larsson & Johansson, 2002; Perry et al., 2002; Thane & Bates, 2000), whereas others identified

lower iron intakes in vegetarians (Donovan & Gibson, 1995, 1996). Lower hemoglobin and ferritin levels have been reported in vegetarian children (Donovan & Gibson, 1995, 1996; Nathan, Hackett, & Kirby, 1996; Thane & Bates, 2000; Thane, Bates, & Prentice, 2003), suggesting that iron is not as well absorbed from a vegetarian diet.

**nonheme iron** The form of iron found in plants and the portion of iron from animal foods that is not part of hemoglobin or myoglobin.

**phytate** A phosphorus-containing compound found in whole grains and dried beans that binds with minerals, particularly iron and zinc, and interferes with their absorption.

**Nonheme iron** is the only form of iron found in vegetarian diets. Inhibitors and enhancers affect the absorption of nonheme iron. **Phytate**, found in whole grains, legumes, and to a lesser extent vegetables (Hallberg & Hulthen, 2000), is the main inhibitor of iron absorption in vegetarian diets. Although no information is available with regard to the phytate content of pediatric vegetarian diets, adult vegan diets may contain two to three times as much phytate as nonvegetarian diets; lacto-ovo-vegetarian diets have a phytate content intermediate between vegan and nonvegetarian diets (Ellis et al., 1987; Hunt, Matthys, & Johnson, 1998).

**bioavailability** A measure of how available to the body a nutrient is after it is ingested.

Because of the lower **bioavailability** of iron from vegetarian diets, iron requirements for vegetarians are 1.8 times higher than for nonvegetarians (IOM, Food and Nutrition Board, 2001). Adjustments do not need to be made to recommendations for infants from birth to 12 months (IOM, Food and Nutrition Board, 2001) because recommendations for infants from birth to 6 months are based on the iron content of breast milk. The Recommended Dietary Allowance (RDA) for infants ages 7 to 12 months is based on a bioavailability of 10% rather than the higher bioavailability (18%) that is used for other age groups (IOM, Food and Nutrition Board, 2001). Iron requirements using a factor of 1.8 times the nonvegetarian RDA for older vegetarian children can be found in **Table 7.1**.

Iron absorption from a vegetarian diet can be enhanced even in the presence of phytate by consuming a vitamin C source at the same time as the iron source (Hallberg & Hulthen, 2000; Sandstrom, 2001). Leavening of bread reduces its phytate content and enhances iron absorption (Hunt, 2002). Iron sources for vegetarian children include iron-fortified breakfast cereals, soy foods, dried beans, and whole grains. **Table 7.2** provides more information about vegetarian iron sources, and **Table 7.3** shows a sample menu that would meet a vegetarian adolescent girl's high iron requirements. Laboratory assessment of iron status is appropriate if a child's diet is low in iron and iron supplements are not regularly used.

**TABLE 7.1** **Iron Recommendations for Vegetarian Children and Adolescents**

| Age, Gender | Iron Recommendations (mg) |
|---|---|
| 1–3 yr, M/F | 12.6 |
| 4–8 yr, M/F | 18 |
| 9–13 yr, M/F | 14.4 |
| 14–18 yr, M | 19.8 |
| 14–18 yr, F | 27 |

*Note:* Values are calculated based on 1.8 times the RDA.

Data from Institute of Medicine, Food and Nutrition Board. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc.* Washington, DC: National Academies Press.

Limited information suggests that the zinc content of diets of vegetarian children is similar to that of nonvegetarian children (Sanders, 1995; Thane & Bates, 2000). However, because phytate also inhibits zinc absorption, vegetarian children are likely to have higher requirements for zinc than are nonvegetarian children. The IOM has not specified a zinc RDA for vegetarians but suggests that the dietary requirement for zinc may be as much as 50% greater for vegetarians, especially for those relying mainly on grains and legumes (IOM, Food and Nutrition Board, 2001). Techniques that can enhance zinc absorption include using yeast-leavened breads, using fermented soy products such as tempeh and miso, and soaking dried beans and discarding the soaking water before cooking the beans (Gibson, Yeudall, Drost, Mtitmunit, & Cullinan, 1998). Zinc supplements should be considered for children on vegan diets, especially those based on high-phytate cereals and legumes (Allen, 1998).

### Calcium and Vitamin D

Children following lacto-ovo-vegetarian diets tend to have adequate intakes of calcium and vitamin D because of their use of dairy products (Perry et al., 2002; Thane & Bates, 2000). Limited data suggest that calcium intakes of vegan children are lower than recommendations (Mangels et al., 2011). Rickets, caused by a deficiency of

**TABLE 7.2** **Food Sources of Iron Ranked by Milligrams of Iron per Standard Amount; Also Calories in the Standard Amount**

| Food, Standard Amount | Iron (mg) | Calories |
|---|---|---|
| Fortified ready-to-eat cereals (various), ~1 oz | 1.8–18 | 54–127 |
| Fortified instant cooked cereals (various), 1 packet | 4.9–8.2 | Varies |
| Soybeans, mature, cooked, ½ cup | 4.4 | 149 |
| White beans, canned, ½ cup | 3.9 | 153 |
| Blackstrap molasses, 1 Tbs. | 3.6 | 47 |
| Lentils, cooked, ½ cup | 3.3 | 115 |
| Spinach, cooked from fresh, ½ cup | 3.2 | 21 |
| Pumpkin and squash seed kernels, roasted, 2 Tbs. | 2.5 | 148 |
| Chickpeas, cooked, ½ cup | 2.4 | 134 |
| Navy beans, cooked, ½ cup | 2.3 | 127 |
| Tomato puree, ½ cup | 2.2 | 48 |
| Lima beans, cooked, ½ cup | 2.2 | 108 |
| Soybeans, green, cooked, ½ cup | 2.2 | 127 |
| Refried beans, ½ cup | 2.1 | 118 |
| Vegetarian "meats," fortified, 1 oz | 0.8–2.1 | Varies |
| Kidney beans, cooked, ½ cup | 2.0 | 112 |
| Tomato paste, ¼ cup | 2.0 | 54 |

Food sources of iron are ranked by milligrams of iron per standard amount; also calories in the standard amount.

Data from Nutrient values from U.S. Department of Agriculture, Agricultural Research Service (2009). *USDA National Nutrient Database for Standard Reference, Release 22* and manufacturers' information.

vitamin D, has been seen in some children following macrobiotic diets (Dagnelie et al., 1990) and in infants and toddlers fed unfortified soy or rice milk (Carvalho, Kenney, Carrington, & Hall, 2001; Imataka, Mikami, Yamanouchi, Kano, & Eguchi, 2004). Adequate calcium and vitamin D is important for all children to promote bone growth and to reduce risk of fracture (Black, Williams, Jones, & Goulding, 2002; Kalkwarf, Khoury, & Lanphear, 2003). There is no evidence that vegan children need less calcium than nonvegetarians, and increased calcium intakes (342 vs. 1,056 mg/day) have been shown to improve bone mineral status of children on near-vegan diets (Dibba, Prentice, & Ceesay, 2000). The Dietary Reference Intake (DRI) for calcium is 700 mg/day for 1- to 3-year-olds, 1,000 mg/day for 4- to 8-year-olds, and 1,300 mg/day for 9- to 18-year-olds (IOM, Food and Nutrition Board, 2011).

There are a number of reliable sources of calcium for children and adolescents who limit or avoid dairy products. These include soy milks and juices that have been fortified with calcium (Heaney, Dowell, Rafferty, & Bierman, 2000; Heaney, Rafferty, & Bierman, 2005). Low-oxalate vegetables, such as kale, broccoli, and collard greens, contain generous amounts of well-absorbed calcium (Weaver & Plawecki, 1994), and although these foods may not be the major source of calcium in children's diets, they can significantly increase calcium intake. Tofu processed with calcium sulfate, almonds, almond butter, and dried figs are other sources of calcium (Mangels et al., 2011). Vegan products that

**TABLE 7.3** Example of an Iron-Rich Menu

| Menu Item | Iron (mg) |
|---|---|
| 1 cup oatmeal | 2.1 |
| ¼ cup raisins | 0.8 |
| 8 oz fortified soy milk | 1.1 |
| 6 oz orange juice | 0.2 |
| 2 slices whole wheat toast | 1.8 |
| Bean burrito: | |
| 1 flour tortilla | 1.1 |
| ½ cup kidney beans | 2.0 |
| 2 Tbs. salsa | 0.0 |
| 1 cup watermelon chunks | 0.4 |
| Stir-fry: | |
| ¾ cup tofu | 10.0 |
| 1 cup cooked broccoli | 1.0 |
| 1 cup brown rice | 0.8 |
| ½ cup cooked carrots | 0.3 |
| ¾ cup strawberries | 0.4 |
| 1 cup fortified soy milk | 1.1 |
| Trail mix: | |
| ¼ cup pumpkin seeds | 5.0 |
| 2 Tbs sunflower seeds | 0.6 |
| ¼ cup dried apricots | 0.9 |
| Total | 29.6 |

Data from U.S. Department of Agriculture, Agricultural Research Service. (2009). USDA Nutrient Database for Standard Reference, release 22.

have been developed to resemble dairy products, such as soy yogurt and soy cheese, do not always contain the same amount of calcium as their dairy counterparts. Label reading is essential to determine the nutrient content of these products. **Table 7.4** provides more information about nondairy sources of calcium for vegetarians.

Fortified soy milks and juices also frequently contain vitamin D. Some breakfast cereals are fortified with vitamin D as well. Although cutaneous synthesis can be an important vitamin D source, factors such as age, limited sunlight exposure, skin tone, season, and sunscreen use can lead to concerns about adequate vitamin D production (Holick, 2004). Vitamin D supplements can also be used to ensure adequate vitamin D intakes.

### Vitamin $B_{12}$

Vitamin $B_{12}$ is only found in significant amounts in foods derived from animals or in fortified foods. Lacto-ovo-vegetarians who consume an adequate amount of cow's milk, yogurt, or eggs regularly can meet recommendations for vitamin $B_{12}$. Vegans must obtain their vitamin $B_{12}$ from fortified foods (some brands of soy milk, breakfast cereals, meat analogues, and nutritional yeast) or from vitamin $B_{12}$ supplements. **Table 7.5** provides information about recommendations, food sources, and serving sizes of vitamin $B_{12}$–rich foods. Foods such as sea vegetables, fermented soy products, and Spirulina are sometimes promoted as sources of vitamin $B_{12}$; these cannot be counted on as reliable sources because they have been shown to contain analogues of vitamin $B_{12}$ that can interfere with the absorption of active vitamin $B_{12}$ (Dagnelie, van Staveren, & van den Berg, 1991; van den Berg, Dagnelie, & van Staveren, 1988; Watanabe et al., 1999).

Adequate vitamin $B_{12}$ is especially important during pregnancy and lactation because the maternal diet during these times is the major influence on the infant's vitamin $B_{12}$ status (IOM, Food and Nutrition Board, 1998). Breastfed infants whose mothers do not consume dairy products, vitamin $B_{12}$–fortified foods, or vitamin $B_{12}$ supplements regularly need vitamin $B_{12}$ supplements at the Adequate Intake level from birth (IOM, Food and Nutrition Board, 1998).

Although the RDA for vitamin $B_{12}$ is extremely low, failure to achieve an adequate intake can lead to serious consequences. Marginal vitamin $B_{12}$ status in adolescents has been associated with impaired cognitive function (Louwman et al., 2000) and gait disturbances (Licht, Berry, Brooks, & Younkin, 2001), whereas vitamin $B_{12}$ deficiency in infants and children has led to failure to thrive, developmental delay, and seizures (Centers for Disease Control and Prevention [CDC], 2003).

Vegetarians eating few or no animal foods and not using vitamin $B_{12}$–fortified foods or vitamin $B_{12}$ supplements should have their **cobalamin** status assessed (CDC, 2003). Serum or urine methylmalonic acid is one test for cobalamin deficiency; holotranscobalamin II, total homocysteine, and serum $B_{12}$ also can be used (Hermann & Geisel, 2002).

**cobalamin** Vitamin $B_{12}$.

**TABLE 7.4** Nondairy Food Sources of Calcium for Vegetarians

| Food, Standard Amount | Calcium (mg) | Calories |
|---|---|---|
| Fortified ready-to-eat cereals (various), 1 oz | 250–1,000 | 100–210 |
| Orange juice, calcium fortified, 1 cup | 500 | 117 |
| Tofu, regular, prepared with calcium sulfate, ½ cup | 434 | 94 |
| Soy milk, calcium fortified, 1 cup | 299 | 104 |
| Collards, cooked from frozen, ½ cup | 178 | 31 |
| Molasses, blackstrap, 1 Tbs | 172 | 47 |
| Spinach, cooked from frozen, ½ cup | 145 | 32 |
| Soybeans, green, cooked, ½ cup | 130 | 127 |
| Turnip greens, cooked from frozen, ½ cup | 125 | 24 |
| Oatmeal, plain and flavored, instant, fortified, 1 packet prepared | 99–110 | 97–157 |
| Cowpeas, cooked, ½ cup | 106 | 80 |
| White beans, canned, ½ cup | 96 | 149 |
| Kale, cooked from frozen, ½ cup | 90 | 20 |
| Soybeans, mature, cooked, ½ cup | 88 | 149 |
| Pak-choi, Chinese cabbage, cooked from fresh, ½ cup | 79 | 10 |
| Dandelion greens, cooked from fresh, ½ cup | 74 | 17 |
| Okra, cooked from frozen, ½ cup | 68 | 27 |

Nondairy food sources of calcium are ranked by milligrams of calcium per standard amount; also calories are in the standard amount. The bioavailability may vary. Both calcium content and bioavailability should be considered when selecting dietary sources of calcium. Some plant foods have calcium that is well absorbed, but the large quantity of plant foods that would be needed to provide as much calcium as in a glass of milk may be unachievable for many. Spinach, Swiss chard, beet greens, and rhubarb are high-oxalate vegetables, and the calcium in these foods is largely unavailable. Many other calcium-fortified foods are available, but the percentage of calcium that can be absorbed is unavailable for many of them.

Data from U.S. Department of Agriculture & U.S. Department of Health and Human Services. *Dietary Guidelines for Americans, 2010*. (7th Edition), Washington, DC: U.S. Government Printing Office.; U.S. Department of Agriculture, Agricultural Research Service. (2009). *USDA Nutrient Database for Standard Reference, Release 22*, and manufacturers' information.

**TABLE 7.5** Recommendations, Food Sources, and Serving Sizes of Vitamin $B_{12}$–Rich Foods

| Age Group | Vitamin $B_{12}$ RDA (mcg/day) | Servings of Vitamin $B_{12}$–Rich Foods |
|---|---|---|
| 0–6 mo | 0.4 (AI) | Vitamin $B_{12}$ should be obtained from breast milk, infant formula, or supplements |
| 7–12 mo | 0.5 (AI) | Vitamin $B_{12}$ should be obtained from breast milk, infant formula, or supplements |
| 1–3 yr | 0.9 | 1 |
| 4–8 yr | 1.2 | 1.5 |
| 9–13 yr | 1.8 | 2 |
| 14–18 yr | 2.4 | 3 |

AI, adequate intake.

Data from Institute of Medicine, Food and Nutrition Board. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Washington, DC: National Academies Press.

### Omega-3 Fatty Acids

**alpha-linolenic acid** An essential 18-carbon omega-3 fatty acid.

**eicosapentaenoic acid (EPA)** A 20-carbon omega-3 fatty acid found in fish oil.

**docosahexaenoic acid (DHA)** A 22-carbon omega-3 fatty acid found in fish oil.

Vegetarian diets are generally low in **alpha-linolenic acid**, an omega-3 fatty acid, whose essential role appears to be as a precursor for the synthesis of the long-chain omega-3 fatty acids, **eicosapentaenoic acid (EPA)** and **docosahexaenoic acid (DHA)**. DHA is found in high concentrations in the membrane lipids of the brain and the retina (IOM, Food and Nutrition Board, 2002) and appears to play a role in cognitive and visual performance (Birch, Garfield, Hoffman, Uauy, & Birch, 2000; SanGiovanni, Parra-Cabrera, Colditz, Berkey, & Dwyer, 2000), although results of supplementation studies are mixed (McCann & Ames, 2005). Unless vegetarians eat eggs or generous amounts of sea vegetables, their diets will also lack direct sources of EPA and DHA. Requirements for alpha-linolenic acid may be higher for many vegetarians than for nonvegetarians because vegetarians must rely on conversion of alpha-linolenic acid to EPA and DHA rather than obtaining these **n-3 fatty acids** from their diet (Davis & Kris-Etherton, 2003). The conversion of alpha-linolenic acid to EPA and subsequently to DHA is very limited (Davis & Kris-Etherton, 2003; Francois, Connor, Bolewicz, & Connor, 2003).

**n-3 fatty acid** A polyunsaturated fatty acid in which the first double bond is three carbons from the methyl end of the carbon chain. Important sources in the U.S. diet include certain fish tissues, canola and soybean oils, and seeds and nuts such as flax seeds and walnuts.

Vegetarians' lower dietary intakes of EPA and DHA are reflected in blood and breast milk concentrations. Adult lacto-ovo-vegetarians and vegans have lower blood concentrations of EPA and DHA than do nonvegetarians (Geppert, Kraft, Demmelmair, & Koletzko, 2005; Krajcovicova-Kudlackova, Simoncic, Babinska, & Bederova, 1995; Rosell et al., 2005), and adolescent vegans have lower concentrations than do lacto-ovo-vegetarians or nonvegetarians (Krajcovicova-Kudlackova, Simoncic, Bederova, & Klvanova, 1997). Breast milk concentrations of alpha-linolenic acid, EPA, and DHA reflect the amounts present in the mother's diet and are lower in breast milk of vegetarian and vegan women (Sanders & Reddy, 1992; Uauy et al., 1996). Breastfed infants of vegetarian and vegan women have lower plasma and red blood cell DHA concentrations than do breastfed infants of nonvegetarian women (Sanders & Reddy, 1992).

DHA supplements derived from microalgae are one option for vegetarians (Conquer & Holub, 1996; Davis & Kris-Etherton, 2003; Geppert et al., 2005). Because some EPA can be synthesized from alpha-linolenic acid, good sources of alpha-linolenic acid such as flaxseed and flaxseed oil, canola oil, soybean oil, walnuts, and soy products should be promoted (Craig & Mangels, 2009). The conversion of alpha-linolenic acid to EPA can be enhanced by limiting use of oils high in linoleic acid and trans fats (Davis & Kris-Etherton, 2003).

#### Critical Thinking About a Vegetarian Diet for a Child

- What tools and techniques would you use to assess a 3-year-old vegan's nutritional status?
- What are some red flags for an eating disorder in a vegetarian adolescent? What would you do if you suspected that a client had an eating disorder?
- Why is it important to ask a vegetarian client to tell you specifically about foods eaten and avoided rather than relying on the person's characterization of his or her diet as "vegetarian"?
- When would you recommend supplements for a vegetarian child? Which supplements and what amounts? Would your recommendations differ for lacto-ovo-vegetarian and vegan children?

### Nutritional Counseling of Vegetarian Infants, Children, and Adolescents and Their Families

The "Position of the American Dietetic Association: Vegetarian Diets" states:

> Food and nutrition professionals have an important role in providing assistance in the planning of healthful vegetarian diets for those who express an interest in adopting vegetarian diets or who already eat a vegetarian diet, and they should be able to give current accurate information about vegetarian nutrition. (Craig & Mangels, 2009, p. 1277).

Families with vegetarian infants, children, and adolescents seek nutrition counseling for a variety of reasons. Perhaps a child or adolescent has decided to become vegetarian but the rest of the family wants to continue eating meat. Perhaps a healthcare provider has referred the family because of concerns about dietary adequacy. Perhaps a family that is already following a vegetarian diet is seeking advice on dietary modifications to improve their diet or to cope with a condition such as renal disease or diabetes. In any case, dietetics professionals need to be able to provide information about

**TABLE 7.6** Tips for Effective Counseling of Vegetarian Clients

- Develop vegetarian-specific counseling materials. These might include handouts on good sources of iron for vegetarians and good sources of calcium and vitamin D for vegans.
- Respect the client's food preferences. Suggesting that a vegetarian include fish or fish oil or that a vegan use some dairy products can alienate clients and compromise the practitioner's credibility.
- Be aware of current research. Many vegetarians have a strong interest in nutrition and may ask detailed questions regarding studies they have heard or read about.
- Create lists of resources for additional information: family-friendly cookbooks, credible websites, and vegetarian organizations.
- If you believe you are not familiar enough with vegetarian nutrition to counsel a vegetarian client, it is your responsibility as a professional to assist this client in finding another registered dietitian with expertise in vegetarian nutrition or to inform the client about reliable vegetarian nutrition resources.

Reproduced from Craig, W. J., & Mangels, A. R. (2009). Position of the American Dietetic Association: Vegetarian diets. *Journal of the American Dietetic Association*, *109*(7), 1277.

food sources of key nutrients for infants, children, and adolescents; to adapt guidelines for individuals with allergies, chronic diseases, or other factors necessitating dietary modifications; to assist with meal planning, food purchases, and food preparation decisions; and to give current information about vegetarian nutrition for the pediatric age group (Craig & Mangels, 2009). **Table 7.6** lists some tips for effective counseling of vegetarian clients. Although key nutrients are important at every age, there are some considerations that vary by age group.

**Learning Point** If there is any question about adequate vitamin $B_{12}$ in a lactating woman's diet, a vitamin $B_{12}$ supplement is recommended for her infant.

## Infants and Toddlers

In early infancy, vegetarian infants do not differ from nonvegetarian infants in terms of feeding practices. Exclusive breastfeeding for 6 months and breastfeeding with complementary foods until at least 12 months of age are the ideal feeding patterns for infants (American Academy of Pediatrics Section on Breastfeeding, 2012).

Commercial infant formula is recommended for infants who are not breastfed or who are weaned before 1 year of age. Lacto-ovo-vegetarian families who use infant formula can use a cow milk–based formula, whereas for vegan families who use infant formula, soy formulas are the only option. Commercial soy beverages do not provide adequate nutrition for infants and should not be used, except for small amounts in cooking, during the first year (Mangels et al., 2011). Other formulas such as those based on rice, nut, or seed milks; nondairy creamer; cereal gruels; or mixtures of fruit or vegetable juices should not be used to replace breast milk or commercial infant formula (Mangels & Messina, 2001). After the first year, either fortified full-fat soy milk or whole cow's milk can be used as the primary beverage for a child who is growing normally and eating a variety of foods (Mangels & Messina, 2001).

### A Word on Vegetarianism and Adolescents

Vegetarian diets are somewhat more common among adolescents with eating disorders (Neumark-Sztainer et al., 1997; Perry, McGuire, Newmark-Sztainer, & Story, 2001). Possibly, adolescents who have issues with food and body weight choose vegetarian or partial vegetarian diets as a socially acceptable way to restrict their food intake (Barr, 1999; Janelle & Barr, 1995; Martins, Pliner, & O'Connor, 1999). The use of a vegetarian diet does not appear to increase the risk of developing an eating disorder (Barr, 1999; Janelle & Barr, 1995). Dietetics professionals should be aware of vegetarian clients who exhibit symptoms of an eating disorder.

Adolescents (and some children) may choose to follow a vegetarian diet even though their family is not vegetarian. Depending on the age and ability of the child or adolescent, parental participation may be needed in the areas of menu planning, food purchases, and food preparation (Messina & Messina, 2010). Dietetics professionals can help families to identify vegetarian meals that the whole family enjoys, to plan meals that can be served with vegetarian options, and to find foods that the child or adolescent can prepare independently.

Solid foods can be introduced to vegetarian infants in the same order that they would be introduced to nonvegetarian infants (Scott, 2003). At around 7 to 8 months when higher protein

foods would typically be introduced, vegetarian infants can start to eat cooked and puréed legumes, well-mashed tofu, or soy yogurt. Infants in lacto-ovo-vegetarian families can also have puréed cottage cheese, yogurt mixed with mashed fruit, or egg yolks. Foods that will eventually play a significant role in the diets of vegetarian children (e.g., legumes, tofu, leafy green vegetables) should be introduced in infancy so the child becomes familiar with the flavor of these foods.

As toddlers are weaned from breast milk or infant formula, energy intake may decrease. Foods that are both nutrient and energy dense, such as soy products, bean spreads, and avocado, are often recommended to maintain energy intake during weaning.

## Meal Planning Guidelines

A variety of meal planning guidelines has been developed for vegetarian children and adolescents (Mangels et al., 2011; Messina & Mangels, 2001; Stepaniak & Melina, 2003; Truesdell & Acosta, 1985; U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). The ideal plan should achieve the following goals (Mangels et al., 2011):

- Meet the needs of different types of vegetarian diets
- Help vegetarians meet the most recent nutrient recommendations
- Focus on specific nutrients identified as being of special importance to vegetarians
- Include a wide variety of foods
- Meet the needs of different age groups

# *Childhood Obesity*

*Inger Stallmann, MS, RD, LD, Edna Harris-Davis, MS, MPH, RD, LD, and Ashley Smith, DVM, MS, RD, LD*

In the twenty-first century, the obesity epidemic has spread worldwide. Pediatric obesity is highly complex and multifaceted. As a result, healthcare practitioners are faced with a significant health problem that has no easy solution. Aside from the health issues associated with pediatric obesity, there are social and environmental issues that also must be considered when addressing the problem both at the individual and community levels. This section is designed to give entry-level practitioners an overview of pediatric obesity, potential influences, and basic information on existing programs. Healthcare practitioners are encouraged to use the following material as a stepping stone to create and develop new programs and community policies to solve this issue.

## Assessment of Overweight Children

In the pediatric population, BMI is used as a screening tool and is not a diagnosis for overweight children. The CDC growth charts, for children ages 2 to 20 years, assess body composition based on BMI-for-age and BMI-for-sex percentiles. The BMI-for-age nutrition status indicators for overweight and obese children are greater than or equal to the 85th percentile to less than the 95th percentile and greater than or equal to the 95th percentile, respectively, indicating a change in terminology from previous National Health and Nutrition Examination Survey (NHANES) reports (CDC, 2012). For children younger than the age of 2 years, it is recommended that practitioners use the World Health Organization (WHO) guidelines that assess weight for height and head circumference (see www.cdc.gov/growthcharts/who_charts.htm). Based on the data, children are classified as obese if they measure above the 98th percentile (Lake, 2012). There is increasing evidence that many overweight children will become obese adults; however, the BMI-for-age assessment should be applied carefully so as to not label children as obese in error (Freedman, Khan, Dietz, Srinivasan, & Berenson, 2001; Serdula et al., 1993). It is important to determine whether a child indeed has extra fat mass and not extra muscle mass, particularly across genders and ethnicity (Ellis, Abrams, & Wong, 1999; Taylor, Jones, Williams, & Goulding, 2002).

### Obesity Rates in the United States

For adults, there has been a rapid increase in our nation's prevalence rate of obesity over the past two decades. Recent data from the 2009–2010 NHANES found 66.3% of the U.S. adult population to be overweight or obese as defined by a BMI of greater than 25 kg/m$^2$ (Ogden, Carroll, Kit, & Flegal, 2012). This represents an increase of 1.8% from the rates in 1999–2000 and more than 10% since the NHANES III (1988–1994). Currently, obesity is estimated to affect 17% of children aged

2 to 19 years. Between NHANES I (1971–1974) and NHANES II (1976–1980), the prevalence of excessive weight among children was unchanged overall, but between NHANES II and NHANES III (1988–1994), the prevalence rose within all age and gender groups (Ogden, Flegal, Carroll, & Johnson, 2002).

The rising trend of obesity in children has since continued through NHANES 2009–2010. Compared with 1976–1980 data, obesity rates in NHANES 2007–2008 increased from 5.0% to 18.1% for 12- to 19-year-olds, 6.5% to 19.6% for 6- to 11-year-olds, and 5.0% to 10.4% for 2- to 5-year-olds. The increase in obesity rates has not occurred to the same extent among all age, race, or socioeconomic groups. Mexican American boys ages 12–19 years (46.1%) and ages 6 to 11 years (44.0%) have the highest prevalence of increased BMI followed by followed by non-Hispanic African American girls ages 12 to 19 years (46.3%) and ages 6 to 11 years (38.9%). According to the Pediatric Nutrition Surveillance system, in 2009 one-third low-income children ages 2–4 years were overweight or obese. American Indian and Alaska Native children had the highest prevalence of obesity in this age group at 20.7% and were the only groups to have a significant rise in obesity levels since 2003. The trend for increasing overweight prevalence among children is illustrated in **Figures 7.1**, **7.2** and **7.3**. **Figure 7.4** illustrates a normal growth curve.

### Global Rise in Obesity

The United States is not alone in facing this serious health problem. The World Health Organization declared obesity one of the top 10 health risk conditions worldwide and in the top 5 in the developed world. In 2008, the WHO reported that more than 1.4 billion adults were overweight and more than 500 million were obese. As a consequence, the WHO estimates that more than 2 million people die each year as a result of being overweight or obese. The United Kingdom has also seen a dramatic rise in the prevalence of pediatric obesity in the last 25 years. From 1995 to 2008, the obesity rate in boys and girls ages 2 to 15 years in the United Kingdom rose from 11.1% to 16.8% and 12.2% to 15.2%, respectively (Bogle & Sykes, 2011).

### Health Effects of Obesity

The recent trends of obesity in children are especially disturbing because of the health consequences of being overweight/obese both short and long term. Many obese children now face physical issues once seen predominantly in adults such as hypertension, type 2 diabetes, dyslipidemia, insulin resistance, sleep apnea, asthma, and nonalcoholic fatty liver disease (Baumann & Brown, 2006; Schwimmer, Burwinkle, & Varni, 2003). However, the effects of being overweight are more than physical; these children may suffer from psychological problems, such as depression, anxiety, lowered self-esteem, and sometimes eating disorders (Erickson,

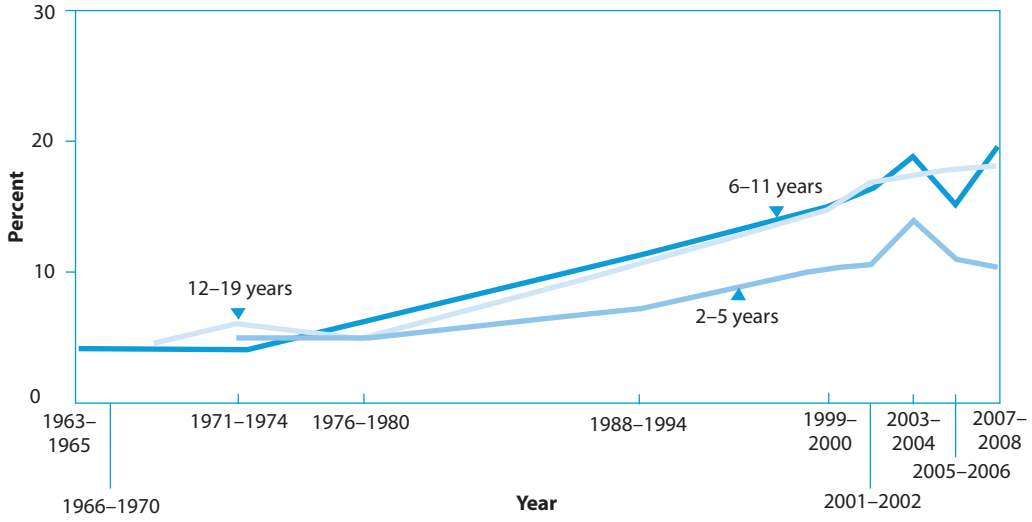



**FIGURE 7.1** Trends in obesity among children and adolescents: United States, 1963–2008.

Courtesy of CDC.

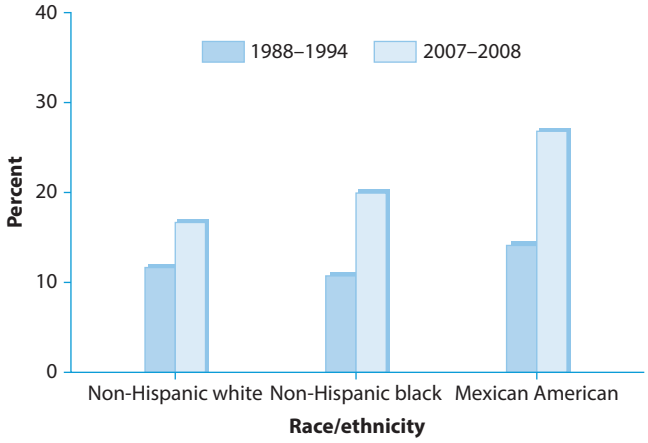



**FIGURE 7.2** Prevalence of obesity among boys aged 12–19 years, by race/ethnicity: United States: 1988–1994 and 2007–2008.

Courtesy of CDC.

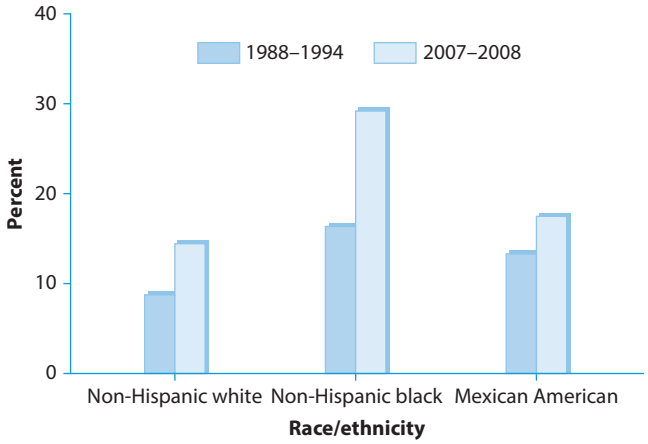



**FIGURE 7.3** Prevalence of obesity among girls aged 12–19 years, by race/ethnicity: United States: 1988–1994 and 2007–2008.

Courtesy of CDC.

Robinson, Haydel, & Killen, 2000; Hassink, 2003; Raman, 2002; Reilly et al., 2003). As the prevalence of obesity in children increases, there is also an increasing trend that they will remain obese as adults (Freedman et al., 2001; Reilly et al., 2003). Freedman and associates (2001) studied the longitudinal relationship between children's BMI and their adult levels of lipids, insulin, and blood pressure. The mean time interval between first and follow-up measurements was 17 years. Over this time period, 77% of overweight children remained so as adults. The likelihood of childhood obesity

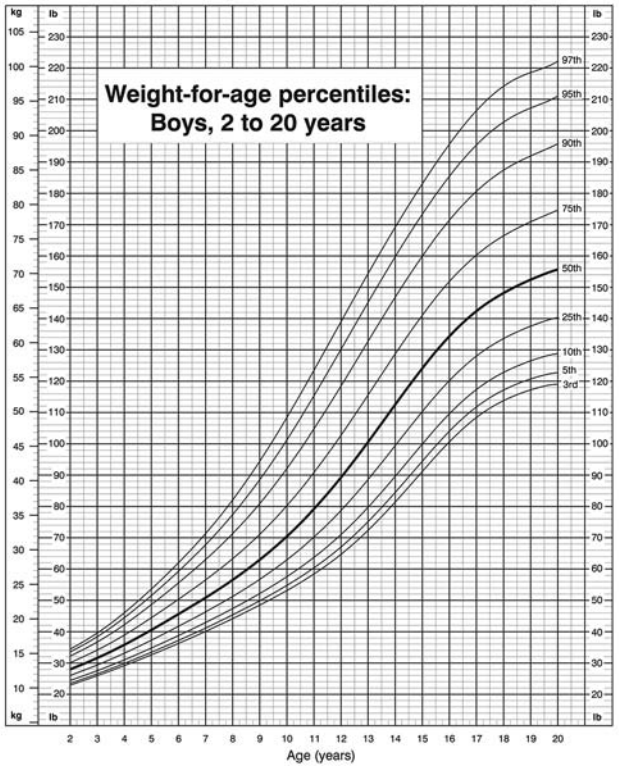



**FIGURE 7.4** Sample of growth chart for ages 2–20 years from the Centers for Disease Control and Prevention.

Courtesy of CDC.

persisting to adulthood was higher if at least one parent is overweight or obese (Reilly et al., 2003; Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). The persistence of excessive weight, once it occurs, is of great concern because being overweight is more strongly linked to chronic disease than is living in poverty, smoking, or drinking. The impact of obesity on overall health has been likened to aging by 20 years (Hill, Wyatt, Reed, & Peters, 2003).

**● Learning Point** Even if overweight status does not track into adulthood, there may be long-lasting medical and psychosocial effects in overweight children (Hill & Trowbridge, 1998).

### Health Disparities in Cultural Diversity

Ethnic, cultural, gender, genetic, socioeconomic, and regional differences exist that influence which children are at greater risk for being overweight. Especially boys of Hispanic American origin, African Americans, and those residing in the South experience higher rates of being overweight (Nelson, Chiasson, & Ford, 2004; Strauss & Pollack, 2003). The ethnic differences in the degree to which certain population groups are affected by overweight prevalence may exacerbate long-term health outcomes and economic disparities that already exist in the United States (Strauss & Pollack, 2003).

## Environmental Influences on Obese Children

### Societal Factors Influencing Overweight Children

Society's perception of overweight or obese people historically has garnered negative attitudes resulting in judgmental behaviors. Overweight and obese people are often perceived as lazy or lacking in willpower to lose the excess weight. However, children's perception of what constitutes overweight or obese status has been shown to be very different from adult perceptions. In one study of overweight and obese Hispanic boys, the children were unable to accurately recognize overweight or obese. Whereas unable to accurately assess themselves, they had negative perceptions of other children they viewed as overweight or obese. Although many had limited knowledge or understanding of what "being healthy" meant, most of the boys modeled their ideas of health after family members they perceived as healthy or based on media messages (Skelton, Irby, Guzman, & Beech, 2012). One of the main limitations of this study was the small size; however, it confirmed several existing theories about the contributors of obesity. The authors concluded limited family mealtime, increased number of meals away from home, and unsafe neighborhoods limiting physical activity all contributed to obesity in the participants. Today, it is widely recognized that the microenvironment (home and family setting) and the macroenvironment in which it is imbedded either promote healthy weight or overweight. Thus, altering both the micro- and macroenvironments is vital to prevention and treatment of overweight in children. The dynamics among individuals and their near, intermediate, and distal environments influence their food choices and physical activity levels, both of which play important roles in the development of overweight (Booth et al., 2001).

**2005 Dietary Guidelines for Physical Exercise**

The 2005 Dietary Guidelines Advisory Committee recommends that children engage in at least 60 minutes of physical activity on most (preferably all) days of the week. Physical activity may include short bouts (i.e., 10 minutes each) of moderately intense activity. In this way, exercise can be accumulated through three to six bouts over the course of the day. The accumulated total of physical activity is what is important for health.

### Obesity and Energy Balance

The environmental factors underlying the recent increase in obesity rates in the United States are multiple; however, diet and physical activity play a crucial role. Basic to this concept is the fact that to maintain a stable weight, energy intake and output must be balanced. Thus, when physical activity level is low, weight status can be maintained with a diet appropriately lower in calories. However, if an adjustment is not made to keep energy intake and expenditure in balance, through increased physical activity or decreased energy intake or a combined approach, excessive weight gain is the result. The alarming rise in obesity among U.S. children begs the question: What changes have occurred in our lifestyles that could have contributed to this problem?

### Dietary Trends Affecting Obese Children

#### Historical Perspective

Today's lifestyles are very different from the lifestyles of our hunter–gatherer ancestors. Genetically, humans evolved during the Paleolithic period 2.6 million to 10,000 years ago and were biologically adapted to survive under the prevailing conditions of that time. Today's Western-style diet is in sharp contrast to the diet of the hunter–gatherers. Yet our human genome has changed little since that period to help us adapt to our present-day lifestyles of "mechanized urban settings, leading sedentary lives and eating a highly processed, synthetic diet" (O'Keefe & Cordain, 2004). The differences in today's diets from that of the hunter–gatherers are that they consumed foods higher in fiber content and lower in energy density and with a different diet composition of long-chain fatty acids. The long-chain fatty acids of the n-6 and n-3 families were consumed in a ratio of approximately 1:1. Today's Western-type diet can provide less of the omega-3 fatty acids with an n-6 to n-3 ratio of 1:15–17 (Simopoulos, 2002).

The diet of the hunter–gatherers was rich in plant foods, supplemented by fish and lean meats and nuts. Indications are that these early ancestors did not experience obesity. Changes in agricultural practices, as well as food processing and manufacturing, have resulted in a vastly different food supply that has evolved over a relatively short time, historically. The development of agricultural societies led to diets based on grains. With this change from a plant-based to a grain-based diet, vitamin and mineral deficiencies began to appear as evidenced by studying bones and teeth. Our natural preference for foods that are calorie dense is rooted in the conditions of our ancestors where obtaining food for survival through hunting, fishing, and gathering

food required high levels of physical activity and a correspondingly high expenditure of energy.

In contrast, obtaining food today means a car ride to the grocery store and little connection between energy intake and expenditure (O'Keefe & Cordain, 2004). The typical American diet today is lower in fiber and plant foods, higher in meat, and high in highly refined carbohydrates and possibly contains a much higher ratio of the n-6 relative to the n-3 long-chain fatty acids. Researchers contend that these dietary changes, combined with sedentary lifestyles, play a significant role in the etiology of chronic diseases affecting developed countries: heart disease, diabetes, cancer, and obesity (Conner, 2000; Simopoulos, 1999). See **Figure 7.5**, MyPlate, which provides healthy meal plans and can be accessed online.

### Changes in the Food Environment

Over the past few decades, significant changes in our food environment have occurred. One of the more notable changes is the rapid increase in the number and types of restaurants found throughout our communities including schools and hospitals. This dramatic growth has been fueled by effective marketing and advertising aimed at the different age groups. Restaurants initially fueled their growth with menu options that included large portions high in calories, fat, salt, and sugars and low in fiber content (Bowman, Gortmaker, Ebbeling, Pereira, & Ludwig, 2004). As competition among restaurants increased, newer marketing tactics arose including "supersized" options and value menus. This allowed consumers to get more for less, making meals faster and more convenient. These factors appeal to working and single parents, and the percentage of children eating fast food meals will likely increase (Bowman et al., 2004). One consequence to this lifestyle of convenience is an inverse relationship between out-of-home meal consumption and diet quality and BMI (American Dietetic Association [ADA], 2002; McCrory et al., 1999; Nicklas, Baranowski, Cullen, & Berenson, 2001).

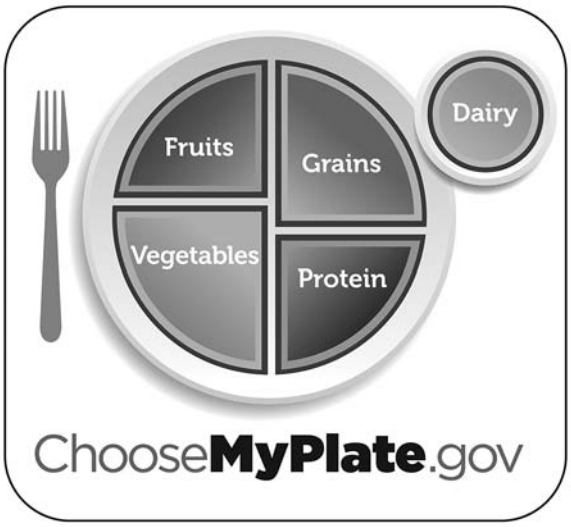



**FIGURE 7.5** MyPlate.gov.

Courtesy of USDA Center for Nutrition Policy and Promotion.

During this same time an economic relationship was noted between the energy cost and the energy density of foods (Drewnowski & Specter, 2004). Healthier options such as lean meat, fruits, and vegetables were found to be more expensive than items such as added fats, sugars, and certain grains. As the obesity epidemic grew, consumers became more aware of healthful options and the restaurant industry responded by offering what consumers perceived as healthier options including salads, whole grains, and alternative beverages. However, the more healthful selections are generally not subject to the low price offers that might otherwise encourage customers to buy them and may not be as healthy as consumers believe.

Recent estimates indicate the amount of food dollars spent outside the home has increased significantly to now comprise approximately 46% of total household food budgets. In 2009, a marked change was noted in consumer spending with regard to food and household income. Households with higher incomes spent approximately half of their income on food at home and half away from home, whereas households in the lowest income bracket spent approximately 70% of their income on meals consumed in the home. This change may be attributable to the recent food crisis and the economic downturn. In addition to the economic changes, the restaurant industry is also facing pressure from the government to make changes.

In 2010, Congress passed the Patient Protection and Affordable Care Act. As part of the act, restaurants with more than 20 locations in a specific area are required to list the caloric count of food items on the menu. Following implementation, there is conflicting information on the impact of the new regulations. One study indicated improvement in the nutritional content of menu items, but most menu items still exceeded recommended daily allowances in sodium, energy, and saturated fat (Bruemmer, Krieger, Saelens, & Chan, 2012).

Another study indicated that adolescents were more likely to reduce their sugar-sweetened beverage intake if caloric information was linked to a physical activity equivalent (Bleich et al., 2012).

The food environment has also been impacted by product innovations and changes during the past two decades. Many of these newer products were designed to lower the fat content of popular foods to address consumers' concerns about dietary fat and unwanted weight gain. One such change seen within the commercial food industry is the ban on trans fats. Despite an explosive growth in low-fat or trans-fat-free food products in many food categories, these food supply changes seemingly have not had the desired effect on our obesity rates. Many food manufacturers offset the lower fat content of the reformulated foods by increasing sugar and carbohydrate contents or substituting unsaturated fats for trans fats to maintain product acceptance by consumers. In the end, the caloric content of the lower-fat product is often similar to the original version. There is also concern that so-called low-fat foods may encourage the consumer to actually eat more calories overall because the designation as a low-fat food provides a "license to eat more" (Kurtzweil, 1996).

Despite the new products, children's overall energy intakes have remained fairly stable since 1971–1974 (NHANES I), except for increases for children ages 1 to 2 years and for adolescent girls ages 12 to 19 years. The macronutrient composition of children's diets has changed, reflecting changes in the food supply. Fat intake as a percentage of energy intake fell from 38% to 33% since 1973, whereas carbohydrate and protein intakes increased. Still, approximately 75% of children did not meet the fat intake recommendations in 1994 (Munoz, Krebs-Smith, Ballard-Barbash, & Cleveland, 1997; Nicklas et al., 2001).

In addition to the change in macronutrient intake, sugar-sweetened beverage (SSB) consumption in adolescents has increased dramatically in the past few decades with the rise of vending machines and fast food restaurants. From 1988 to 2004, sugar-sweetened beverage and 100% fruit juice intake increased from 242 kcal/day to 270 kcal/day in children and adolescents aged 2–19 years (Wang, Bleich, & Gortmaker, 2008). The rise in SSB intake is linked with an overall increase in fast food consumption thanks to targeted marketing by restaurants. Twice as many children and adolescents drank carbonated soft drinks if they had consumed fast foods on one of two survey days than if they did not (Paeratakul, Ferdinand, Champagne, Ryan, & Bray, 2003).

Consumption of SSB among children and adolescents has been shown to be associated with higher energy intake and may account for up to 15% of their caloric intake in a day (Harnack, Stang, & Story, 1999; Wang et al., 2008). Whereas individual studies and review of meta-analysis data have reported an association between school children's sugar-sweetened drink consumption and their BMI, there is limited long-term data, which is important in a growing population (Malik, Willett, & Hu, 2009). After controlling for anthropometric, demographic, dietary, and lifestyle variables, one study conducted prospectively over 19 months showed that for each additional serving of sugar-sweetened beverage consumed, both BMI and propensity to become obese increased (Ludwig, Peterson, & Gortmaker, 2001).

Since 1977, the average serving size of a given sugar-sweetened beverage has increased by 46%, which has been seen as a contributing factor in the rise of obesity (Wang et al., 2008). This increase coincides with the replacement of the 12-fluid-ounce can by the 20-fluid-ounce bottle sold in convenience stores and in vending machines, encouraging increased consumption of these drinks. Additionally, sports drinks offer a 32-fluid-ounce version that is packaged in a bottle shape that is easy to tote, thereby encouraging frequent and increased consumption. Whereas older adolescents obtain the majority of their SSB calories from soda, younger children are more likely to obtain additional calories from 100% fruit juice sources.

In addition to increase in beverage size, the general consensus is that portion sizes have increased in the last few decades; however, limited clinical data actually support this theory. To substantiate this theory, researchers evaluated the average portion size of specific food items consumed from 1977 through 1998 based on the data collected during the Nationwide Food Consumption Survey and Continuing Survey of Food Intake by Individuals (Nielsen & Popkin, 2003). The results concluded that there was an overall increase in portion sizes and energy intakes of specific food groups during this time period. Researchers have since expanded

the impact of portion size on energy intake in a variety of clinical studies. Wansink and Kim (2005) evaluated the influence of portion size on the intake of food even if less palatable. Moviegoers were randomly selected to receive either a medium or large bucket of popcorn that was fresh or stale. Whereas the moviegoers given large containers of fresh popcorn consumed more overall, those given large tubs of stale popcorn consumed more than those given medium-sized portions of stale popcorn (Wansink & Kim, 2005). When evaluating patrons' intake in a restaurant setting, studies indicate that larger portion sizes lead to increased energy intake (Diliberti, Bordi, Conklin, Roe, & Rolls, 2004; Rolls, Roe, & Meengs, 2007). **Figure 7.6** shows food expenditures away from home.

### Regulation of Energy Intake

Although very young children possess innate abilities to regulate energy intake based on their needs, children tend to lose some of this ability as they age and become more responsive to environmental influences and increasingly take their behavioral cues from their surroundings. Children vary in their ability to self-regulate energy intakes (Johnson, 2000), but children who have experienced so-called restrictive feeding practices appear to be less able to respond appropriately to internal signs of hunger and satiety and may be more susceptible to overeating (Birch & Fischer, 1998). Also, as children learn from their environment, food portion sizes influence their intake. Thus, children may overeat when presented with excessive food and beverage portions, risking excessive weight gain over time.

### Family Eating Environment

Parents who are struggling with personal weight issues may hamper their children's ability to self-regulate energy intake. Because many adults suffer from overweight or obesity (National Center for Health Statistics, 1999), some parents are understandably concerned about their children's risk of also becoming overweight or obese. Aiming to avoid overweight in their children, parents may impose greater restrictions on their children's intake (Hood et al., 2000). If these same parents exhibit uninhibited eating styles themselves, they serve as poor role models. These behaviors in combination can cause the transfer of eating styles that pose an increased risk for the development of an overweight child (Zocca et al., 2011). As a result, well-intentioned parents may actually produce the very outcome they seek to avoid (Hood et al., 2000; Johnson, 2000). A common strategy used to control children's intake is to restrict their access to high-calorie foods or to use favorite foods as a reward to shape behavior (Haire-Jooshu & Nanney, 2002). At first, these behaviors may seem reasonable enough; however, restricting certain foods has been shown to increase the child's

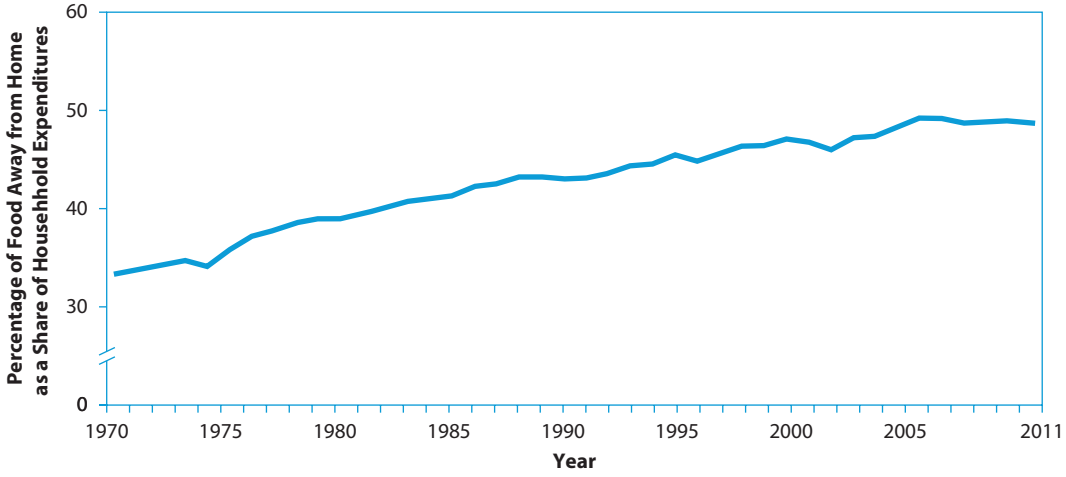



**FIGURE 7.6** Food away from home continues to increase as a share of household expenditures.

Data from Economic Research Service. *Food Expenditures, 2012*. U.S. Department of Agriculture.

desire to consume these foods in greater amounts (Campbell, Crawford, & Ball, 2006). Again, such family dynamics around food may set up patterns of poor self-control of energy intake in children (Campbell et al., 2006; Fischer & Birch, 1999; Haire-Joshu & Nanney, 2002).

Regular family mealtime is another area that contributes a significant amount of influence to the development of adolescent eating patterns (Neumark-Sztainer, Hannan, et al., 2003; Sweetman, McGowan, Croker, & Cooke, 2011). Researchers have demonstrated that the frequency of family mealtime varies with age but also plays a role in dietary intake (Neumark-Sztainer, Hannan, et al., 2003). Children who participated in family mealtime more frequently had increased intake of fruits and vegetables, decreased soda intake, and increased intake of nutrients such as calcium, fiber, and iron (Neumark-Sztainer, Story, et al., 2003; Sweetman et al., 2011). An optimal environment for children to develop self-control of energy intake is when parents provide nutritious food and allow children to determine if and how much to eat. These principles of division of responsibility are discussed extensively elsewhere (Johnson & Birch, 1994; Satter, 1987, 1999, 2000).

#### Household Food Insecurity

Although seemingly paradoxical, low-income compared with higher-income households appear to experience increased rates of overweight individuals. Proposed mechanisms for this effect include higher intakes of cheaper more calorie-dense foods that may lead to excessive energy intakes (Alaimo, Olson, & Frongillo, 2001; ADA, 2010; Drewnowski & Specter, 2004). It is important to note that not all members of a household may experience food insecurity to the same extent (ADA, 2010). Casey and colleagues (2004) found distinct demographic factors associated with food insecurity that puts white girls 12–17 years in households with income less than 100% and more than four times the federal poverty level at highest risk for obesity. This finding is in contrast to children from low-income households who do not experience food insecurity (Alaimo et al., 2001). One study looked at children younger than 5 years of age enrolled in the Women, Infants, and Children (WIC) program and noted a significant relationship between gender, age, and food insecurity (Metallinos-Katsaras, Sherry, & Kallio, 2009). For girls younger than 2 years of age in the program, food insecurity was unlikely to correspond to the child being overweight. For girls ages 2–5 years in food-insecure households, there was a strong correlation between hunger and being overweight (Metallinos-Katsaras et al., 2009). However, these findings did not correlate to boys within the study.

As the children reached school age, participation in programs, such as the National School Breakfast and Lunch Programs and in the Food Stamp Program, may be protective for these girls. Girls who participated in all three programs were at a 68% reduced odds of becoming overweight when compared with nonparticipants from families experiencing food insecurity (Jones, Jahns, Laraia, & Haughton, 2003). On the other hand, a more recent study found a 42.8% increase for young girls and a 28.8% decrease for young boys in the predicted probability of obesity with participation in the Food Stamp Program for the previous 5 years. This study did not control for food insecurity within households, which may help to explain the differing results (Gibson, 2004). Additional retrospective longitudinal studies that included assessment of food security and hunger status of each household member might help explain why some children are affected more than others. **Figure 7.7** illustrates prevalence of obesity by state income levels.

#### Prenatal Influence

There is increasing evidence that the intrauterine environment may play a role in the development of obesity. The developmental origins of adult disease hypothesis suggests that many of the diseases that

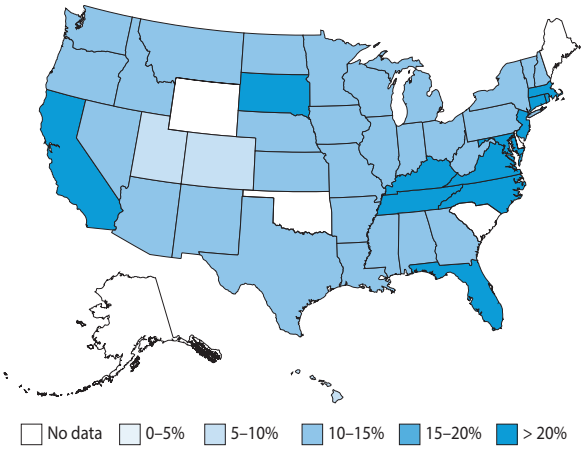



**FIGURE 7.7** 2011 State obesity prevalence among low-income children aged 2 to 4 years.

Courtesy of CDC.

people develop later in life are a result of growth restriction in the intrauterine environment (de Boo & Harding, 2006). Although this theory developed in response to the rise in coronary artery disease–related deaths in England, the effects have been replicated in studies throughout the world (de Boo & Harding, 2006). In 2006, Roseboom, de Rooij, and Painter published one of the most comprehensive studies evaluating the effect of acute undernutrition on the intrauterine environment in a previously well-nourished population. The Dutch Famine Studies concluded that there is a significant correlation between the development of cardiovascular disease and obesity in individuals exposed to famine in utero during the first trimester versus individuals exposed in later stages of the pregnancy. The initial cohort was divided into three sections: early, middle, and late gestation. Based on the evaluation of the cohorts as adults, researchers found significant differences in the rates and severity of clinical disease. Individuals exposed early in their gestation to undernutrition had higher rates of cardiovascular disease, glucose intolerance, and breast cancer as adults (Roseboom et al., 2006).

#### Breastfeeding

New mothers returning to work may decide against breastfeeding or may nurse their infants for only a very limited time because of the real and/or perceived obstacles to breastfeeding while working outside the home. This is unfortunate because breast milk is the ideal food for infants. Evidence is emerging that breastfeeding may offer some protection against obesity (Armstrong & Reilly, 2002). Clinical studies of low-income children followed from birth to 4 years of age indicated that the protective effect of breastfeeding against obesity pertained only to non-Hispanic white children (Bogen, Hanusa, & Whitaker, 2004; Grummer-Strawn, Mei, & Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System, 2004). Proposed mechanisms for this effect include more normal growth patterns (lower early weight gain), possibly associated with lower basal insulin levels in the breastfed versus the formula-fed infant, and the inherent control of food intake maintained by the breastfed infant (Dietz, 2001).

Breastfeeding on demand is thought to teach infants to regulate their intakes appropriately, based on internal cues for hunger and satiety and in response to their individual growth needs. Formula-fed infants may have fewer opportunities to develop this important skill. Reasons for this may be that parents or caregivers may want to follow a set feeding schedule or may encourage the infant to consume a certain amount of formula. Both of these feeding behaviors override the infant's internal cues and may result in overfeeding and lessen the infant's ability to self-regulate energy intake (Birch & Fischer, 1998).

### Physical Inactivity Affecting Obesity in Children

Physical inactivity among children is one of the major health concerns regarding obese children. Children learn physical inactivity from their adult role models and environment through limited access to recreational activities in their community, lack of safe neighborhoods, and, perhaps, unlimited access to television viewing and other media outlets, including computers. Newly developed communities that lack "built-in" access to physical activity are poorly planned. Often, children are bused to school because walking to school is not an option as a result of safety issues and distance. In many towns and cities, sidewalks were once common, but now some urban and suburban developments have shaped many residential areas into "unwalkable" communities. Although further research is needed for a better understanding of the influence of the human-built environment on physical activity, it appears clear that urban sprawl has caused some relationship among land use, transportation, and health, particularly children's health (Frumkin, 2002).

### School Physical Education

At school, children are challenged to find opportunities to be physically active on a daily basis. Often, school academic curricula and programs take priority over physical education and recess time. Physical education curricula and recess have slowly diminished over the last few years (Burgenson, Wechsler, Brener, Young, & Spain, 2001).

Data from the Youth Risk Behavior Surveillance System show that 51.77% of students nationwide were enrolled in physical education classes 1 or more days in an average week, whereas only 31.5% of students nationwide attended physical education classes 5 days in an average week. Improvements in physical education teaching practices are also needed to decrease use of inappropriate techniques, such as using physical activity as a form of punishment or using physical activity games that may cause embarrassment or aggressive behavior (i.e.,

dodge ball) (Burgenson et al., 2001). A nationwide effort to establish laws or policies to increase student enrollment and participation in daily physical education is desirable, especially in the older student population. The goal is for children to establish healthy lifestyle habits that can carry over into adulthood. Advocacy for increasing student participation in daily recess and physical activity should be a top priority of schools and health and community leaders to help prevent overweight children.

#### Television Viewing

To an extent television viewing and other media outlets have displaced a more natural environment of family interactions for growing children, such as learning and playing. Children who spend significant time in sedentary activities tend to weigh more than optimal and to be at greater risk of being obese. Several studies conclude that children who watch 4 or more hours of television are more likely to be overweight than those who watch less television (Andersen, Crespo, Bartlett, Cheskin, & Pratt, 1998; Dowda, Ainsworth, Addy, Saunders, & Riner, 2001; Gortmaker et al., 1996).

Over the past several years, the amount of time adolescents devote to daily media use has risen dramatically. From 2004 to 2009, the average amount of time devoted to media use in teens increased from 6.21 hours to 7.38 hours per day (Kaiser Family Foundation [KFF], 2010). The increase in media usage is spread across a variety of devices including televisions, videos/DVDs, computers, video games, audio/iPods, and cellular phones. Similar to previous studies there is considerable difference in media usage when race and ethnicity are factored in the equation. In 2004, the difference in daily media use between black and white youth was 2 hours; the time difference doubled to 4 hours by 2009. In addition, African American and Hispanic children spent 12:59 and 13:00 hours, respectively, on media usage each day compared to 7:38 hours spent by white children (KFF, 2010).

The results of recent epidemiological studies provide inconclusive links between television viewing and obesity (Jordan & Robinson, 2008). One cross-sectional study failed to find a relationship between television viewing and overweight status in adolescent girls; however, other studies have found a weak association between the two (DuRant, Baranowski, Johnson, & Thompson, 1994; Neumark-Sztainer, Hannan, et al., 2003; Robinson et al., 1993). The trend toward increasingly sedentary lifestyles is obvious to many adults, as they compare this generation's children's activity level with that of children two or three generations earlier. Learning, playing, and physical activity were an integral part of family life, and television viewing was a special occasion. One study indicated that U.S. children overall are less active than children in other countries and cultures (Crawford et al., 2004). Today, television viewing is an integral part of family life because it is an inexpensive form of entertainment; however, it is a sedentary behavior (Hardy et al., 2006).

Excessive television viewing leaves less time for positive family interactions and activities. Although there are many educational and worthwhile programs to watch on television, some programs and advertisements may be questionable for young viewers. The Institute of Medicine recommends that screen time be limited to less than 2 hours per day for children ages 2–5 years in an effort to limit exposure to food and beverage marketing. One major challenge is the multi-million-dollar advertising industry that promotes products between and during television programming (Borzekowski & Robinson, 2001). Appealing and enticing advertising segments are targeted at children to increase their desire to choose unhealthy foods. Studies indicate that 83% of the commercials aired during programming geared toward children younger than 11 years were for snacks, fast foods, and/or sweets (Harrison & Marske, 2005). The young consumers predictably request these products (or may even purchase these themselves) during trips to the grocery store because they recognize their favorite cartoon characters on the colorful packages strategically placed at their eye level. This has resulted in a dramatic increase in sales of candy, snack foods, soft drinks, fast foods, and cereals. It is estimated that food and beverage manufacturers spend in excess of $10 billion per year on marketing directed at the adolescent population (Linn & Novosat, 2008). Excessive television viewing by children fosters a lifestyle of physical inactivity and high calorie consumption as a result of the influence of television food advertising; this combination of factors adds up to a perfect formula for fueling the rising trend of obesity in children.

#### Community Environment

An important part of treating overweight children is to decrease sedentary behavior and increase physical activity (Steinbeck, 2001). However, children

are still challenged with real barriers to being physically active in their existing physical environment. Although research on the impact of the built environment on childhood obesity is still in the early stages, studies have identified several contributing factors. Galvez, Pearl, and Yen (2010) used the Ecological Systems theory to evaluate the impact of the built environment on factors such as diet, physical activity, neighborhood safety, and obesity. The review, although limited, highlighted the fact that many of the problems are directly related to the socioeconomic status and level of urban areas. Areas of lower socioeconomic status are more likely to have fewer parks and green spaces, higher rates of crime, and limited access to healthy foods (Lindholm, 2011).

## Opportunities to Intervene and Prevent Childhood Obesity

Within the last few years, the number of programs available to healthcare practitioners designed to address the problem of childhood obesity has risen dramatically. New and existing programs focus on population-based approaches to prevention and treatment because traditional and clinic-based services alone clearly have failed to address the obesity epidemic adequately. Students in one clinical study indicated that they would be willing to make lifestyle changes such as increasing their physical activity level and eating more fruits and vegetables (Wilson, 2007). However, this same group of students were not willing to reduce their sugar-sweetened beverage intake or give up their computers or video games as part of their lifestyle changes. Thus, healthcare practitioners should examine new and creative avenues to help their patients within existing local, state, and national nutrition and social programs.

## Nutrition Programs

### Special Supplemental Program for Women, Infants, and Children

Researchers have concluded that maternal obesity prior to or during pregnancy plays a significant role in the development of obesity in offspring (Gibson et al., 2007). Developing policies and conducting outreach activities to recruit pregnant women into prenatal care during the first trimester promote early intervention and nutrition care for mothers-to-be. Improving maternal nutrition intake improves nutrition delivery to the unborn child and helps to avoid under- or overnutrition, thereby lowering the risk of future obesity in the child (Dietz, 2004). Some clinics conduct grocery store tours to teach shopping skills to WIC program participants to help them establish healthy eating patterns.

Within this population, it is important to identify participant children older than 2 years of age who exhibit rapid weight gain and to target efforts to ensure that they receive culturally appropriate interventions and guidance to lower their risk of becoming overweight. Training clinic healthcare practitioners in culturally appropriate methods of counseling enables the team to provide infant/child feeding and physical activity advice consistent with evidence-based obesity prevention. The clinic environment should convey clear and consistent messages to encourage healthy eating and physical activity for both program participants and staff.

### Breastfeeding

Breastfeeding may serve as an early intervention to prevent childhood obesity, and breastfeeding promotion can occur in various settings. The promotion of breastfeeding should begin in the hospital as healthcare practitioners work to create a baby-friendly environment according to the guidelines set forth by the World Health Organization and UNICEF in the Baby-Friendly Hospital Initiative (WHO & UNICEF, 2009). This 10-step guide lays the groundwork for practitioners to expand their knowledge into the community by helping to develop breastfeeding policies in work sites, hospitals, and community sites. As part of this program, healthcare practitioners can train nutritionists, nurses, doctors, and other medical professionals on breastfeeding management and supportive ways to increase breastfeeding initiation and duration rates. The collaboration of local, state, and national organizations to increase breastfeeding awareness and education can help the nation reach the Healthy People 2020 objective of having 60% of babies born breastfed for at least 6 months (U.S. Department of Health and Human Services, 2010).

## Social Programs

### Preschool and Day Care

In 2012, the American Academy of Pediatrics, American Public Health Association, and National Resource Center for Health and Safety in Child Care and Early Education published guidelines that child care and early education centers can use to develop policies to support healthy eating, appropriate physical activity, and limited screen

time (National Resource Center for Health and Safety in Child Care and Early Education [NRC], 2011). The evidence-based guidelines set forth standards in a variety of areas starting with general nutrition requirements such as feeding plans and meal planning as well as caring for children with food allergies. The guidelines also encompass areas of nutrition education, physical activity, and screen time exposure from infancy to preschool-aged children. The panel recognizes that feeding should occur within a positive eating environment and that physical activity is necessary for healthy physical, social, and emotional development (NRC, 2011). These guidelines encourage families, caregivers, teachers, healthcare professionals, and policy to promote a healthy and balanced lifestyle for young children to prevent childhood obesity.

#### School Systems

Although the obesity epidemic affects all states at different levels, the scarcity of financial resources and magnitude of the problem often hamper a state's abilities to implement effective programs. In response to the varying needs, the CDC's Division of Nutrition, Physical Activity, and Obesity (DNPAO) works with state programs to improve nutrition and physical activity, thereby reducing obesity levels at the state level. The state-based Nutrition and Physical Activity Program to Prevent Obesity and Other Chronic Diseases currently works with approximately 25 states in a variety of settings. The goal is to implement programs and policies shown to be effective. As a result, the program targeted six strategic areas in which to focus (CDC, 2011):

1. Increase fruit and vegetable consumption
2. Increase physical activity
3. Increase breastfeeding initiation, duration, and exclusivity
4. Decrease consumption of sugary drinks
5. Reduce the consumption of high-energy-dense foods
6. Decrease television viewing

From elementary to high school, the school environment encompasses a large portion of children's day. The focus of school policy development should be on providing access to healthy foods and increasing daily physical activity. These goals can be accomplished through existing local, state, and national programs. Schools should include policies that limit competitive foods and beverages and mandate the use of nutrition standards for food and beverages sold from vending machines and school stores. School systems can also participate in the National Farm to School Program to connect local farms and schools. This program provides free training, assistance, and guidance for schools while promoting use of locally grown products in the schools to increase students' daily fruit and vegetable consumption and support local agriculture (see http://www.farmtoschool.org). Promotion of school breakfast and lunch program participation can help to ensure students within the school receive appropriate nutrition, whereas nutrition education curricula from prekindergarten through 12th grade (50 hours a year minimum has been suggested) can influence student eating behaviors outside the school as well (American Dietetic Association, Society for Nutrition Education, & the American School Food Service Association, 2003).

A campus that is friendly to physical activity with walkways, bicycle racks, and safe outdoor play equipment can help to increase the daily physical activity level of students as well as promote active transport to school. This program was designed in cooperation with the CDC and state organizations to encourage children to seek active methods of getting to school such as a walking school bus or a safe route to school program (CDC, 2011). Although grants came from national sources, each state relied on local participants and leaders to make the programs work within their community (CDC, 2011). Physical activity should not end at the school's front door, but should be incorporated into the school day with increased physical education classes and recess to help students meet the *Physical Activity Guidelines for Americans* goal of 60 minutes of physical activity per day for adolescents ages 6–17 years (U.S. DHHS, 2008). School and community leaders should seek funding from their states to promote a more active lifestyle in adolescents. For example, Georgia and New York provided training for educators prior to implementing the programs and continues to provide ongoing information to participants and states about the program results. Systems to assess children's health and behaviors (i.e., height and weight, BMI, blood pressure, fruit and vegetable consumption, television viewing, transportation modes to school, and actual physical activity during recess and physical education classes) are important to monitor progress and regress in these health promotion efforts and target areas for change.

#### Community-wide Venues

Communities that want to improve the nutritional intake of their residents may need to take a closer look at what is in their community. In the past few years, the CDC Division of Nutrition Physical Activity, and Obesity teamed up with communities throughout the country to provide "policy and guidelines development, surveillance, epidemiological and behavioral research, intervention development, and technical assistance" in an effort to combat the problem of obesity (CDC, 2011). Several unique programs have resulted including one that encourages community leaders to evaluate accessibility of healthy foods within the community and to find viable solutions when good food is not available. Such evaluations enable communities to target areas with higher densities of fast food restaurants or areas that lack a supermarket; identifying such areas could provide incentives to existing supermarkets and farmers markets to establish retail outlets there (CDC, 2011). State and local parks and recreation departments should also routinely assess the safety and accessibility of parks and green spaces throughout the community to make sure that children have a safe place to play. Advocating for a healthy environment in various settings for children, such as Boys and Girls Clubs, YMCA/YWCA, and summer camps, can help children establish and maintain healthy lifestyles year-round.

## Programs and Resources That Support Evidence-Based Practices in Preventing Childhood Obesity

### Let's Move!

In 2010, Let's Move! (www.letsmove.gov) was launched as a nationwide program designed to reduce childhood obesity within a generation. As part of this program, a national task force on childhood obesity was established to review all existing programs in an effort to create a comprehensive national program that encompassed proper nutrition and physical activity. There are five basic tenets of the campaign that include creating a healthy start for children, empowering parents and caregivers, providing healthy food in schools, improving access to healthy and affordable foods, and increasing physical activity. One of the unique aspects of the program is that it challenges parents, kids, schools, chefs, community leaders, politicians, and healthcare providers to work together to find the resources and solutions to end this epidemic.

The Healthy Hunger Free Kids Act passed in 2010 was designed to improve the nutritional quality of school lunches. As part of the new standards, school lunches must include more whole grains, fruits, and vegetables; low-fat dairy products; and less sodium and fat. They also should embody the theory behind the MyPlate program by preparing menus for specific grades, K–5, 6–8, and 9–12, and demonstrating correct portion sizes. Additional funding will be made available to schools that meet the new standards. Schools will be reimbursed an additional 6 cents for each lunch they serve in accordance with the new standards.

### 5 A Day

The Produce for Better Health Foundation is a consumer education foundation that chairs the national 5 A Day for Better Health Program (see http://www.fruitsandveggiesmorematters.org). The 5 A Day for Better Health Program is one of the nation's largest public–private nutrition education initiatives. The 5 A Day initiative encourages people to consume five to nine servings of colorful fruits and vegetables daily. Researchers have studied the relationship between fruit and vegetable intake and weight maintenance and loss, especially in the pediatric population. For example, one study found that families who increased fruit and vegetable intake tended to consume fewer calories and were better able to manage their weight than those families who focused on decreasing fat and sugar intake in their diet. In addition, 5 A Day campaigns are timely and useful because they have adaptable toolkits and other promotional items that fit various settings.

### MyPlate.gov

In 2005, the U.S. Department of Agriculture released its newest tool geared toward healthy eating, ChooseMyPlate.gov. Gone were the hidden messages of the previous food guide pyramid in favor of an easier to understand plate icon designed to show people the proper portion sizes. The initial food guide programs were designed to help people build a foundation for a healthy diet based on the *Dietary Guidelines*, but they often failed to properly educate on portion size (Britten, Marcoe, Yamini, & Davis, 2006). The program website is tailored to be an interactive resource for consumers about healthy eating while focusing on selected messages (see http://www.choosemyplate.gov). As part of the Let's Move! and 5 A Day campaigns, it aims to improve the nutritional status of the average American by eliminating a lot of the guesswork involved with eating.

The programs and resources discussed here are only a few avenues to explore and recommend to partners and perhaps focus groups. To combat the current epidemic, healthcare practitioners should be prepared to engage a variety of resources within the community and beyond. Successful outcomes depend on designing interventions to target the audience and enable collaborative efforts between practitioners and participants. Healthcare practitioners must be creative and timely in sharing their vision and potential solutions to address childhood obesity. Healthcare practitioners and students can research resources and participate in Internet discussion lists to learn of best practices, current research, and new findings in preventing and controlling overweight status among children.

## Food Allergies

*Shideh Mofidi, MS, RD, CSP and Stefanie A. Giampa, MS, RD, LDN*

Over the past 20 years, the prevalence of **food allergy** has almost doubled, resulting in increased awareness among the general public and the scientific community. This has led to significant advances not only in the diagnostic evaluation of food allergy but also in the development of therapeutic modalities for the treatment of food allergy. Although 30% of the general public believes they have food allergies, studies have shown that 3% to 4% of adults and approximately 5% of children have food allergies (Chafen et al., 2010; Sicherer & Sampson, 2010). Any food can provoke a reaction, although relatively few foods are responsible for most food-induced allergic reactions. Currently, management of food allergies is educating the food-allergic individual to avoid the identified food allergen completely and to initiate therapy in case of an accidental ingestion.

**food allergy** An immune-mediated abnormal response to food (specifically food proteins) triggered by the body's immune system with involvement of multiple systems in the body, including the skin, respiratory system, cardiovascular system, and/or the gastrointestinal tract.

### Definitions

*Adverse food reaction* is a broad term defined as any abnormal reaction resulting from the ingestion of a food that might be the result of **food intolerance** (nonallergic food hypersensitivities) or food hypersensitivity (food allergy) (Sampson, 2004a; Sicherer & Teuber, 2004). Food intolerances do not involve the immune system and are usually limited to the gastrointestinal system. In contrast, food allergy is an immune-mediated abnormal response to food with involvement of multiple systems in the body.

**food intolerance** Any abnormal reaction to food that does not involve the immune system and is usually limited to the gastrointestinal system, such as lactose intolerance.

To correctly diagnose food allergies, it is critical to be aware of and able to differentiate the symptoms of food allergy from other adverse reactions to foods. Adverse food reactions can be divided into those that are toxic and those that are nontoxic. Toxic reactions do not rely on individual sensitivity and can occur in virtually anyone who ingests sufficient quantities of a tainted food. Examples of toxic reactions include bacterial food poisoning resulting in diarrhea and vomiting; pharmacologic effects such as jitteriness from caffeine or headaches from tyramine-containing foods; or scombroid fish poisoning whereupon ingestion of sufficient quantities of the fish, the histidine in the spoiled fish is metabolized to histamine, resulting in itching, flushing, and angioedema.

In contrast, nontoxic adverse reactions to foods depend on individual sensitivity and can be further divided to non-immune-mediated (food intolerance) and immune-mediated (food allergy). Food intolerance includes conditions such as lactose intolerance, in which sufficient lactase enzyme is not present and nausea, abdominal cramps, bloating, gas, and diarrhea result when lactose, the major sugar found in milk, is ingested. Other conditions categorized as food intolerance are metabolic disorders such as galactosemia, in which deficiency of an enzyme that converts galactose to glucose results in a myriad of significant complications; pancreatic insufficiency such as in cystic fibrosis; reaction to food additives typically used to enhance taste, add color, or protect against the growth of microbes; and neuronally mediated illness such as in gustatory rhinitis-rhinorrhea from spicy or hot foods or auriculotemporal syndrome where there is facial flushing after ingestion of tart foods (Sampson, 2004b; Sicherer & Sampson, 2010).

Food allergy is different from food intolerance in that the reactions are usually specifically related to food proteins, not to fat or carbohydrate. The offending allergens are typically small glycoproteins that are heat resistant and acid

### Food Additives

Monosodium glutamate (MSG) is a flavor enhancer, and when taken in large amounts, it can cause some of the following that occur rapidly:

- Flushing
- Sensations of warmth
- Headache
- Chest discomfort

Sulfites:

- Are naturally occurring in food typically to increase crispness or prevent mold growth.
- They have been generated during the winemaking process.
- Can cause breathing problems in people with asthma.
- Have been banned by the Food and Drug Administration (FDA) as spray-on preservatives for fresh fruits and vegetables.
- When present in foods, they are listed on ingredient labels.

Courtesy of the National Institute of Allergy and Infectious Diseases.

stable (Sampson, 2004b). Food hypersensitivity can be further classified based on the role of IgE antibody as **IgE-mediated, non-IgE-mediated food allergies** (cell-mediated), and mixed IgE- and cell-mediated (see **Table 7.7**). Typically, the IgE-mediated food allergies are characterized by an acute onset of symptoms that typically involve the skin, respiratory and cardiovascular systems, and/or the gastrointestinal tract (see **Table 7.8**) (Sampson, 2004b). When a food protein is ingested, the IgE recognizes it on the surface of these cells; mediators such as histamine are released, and symptoms occur. These symptoms usually occur within minutes and up to 2 hours after the ingestion of the food. The severity of the reaction depends on many factors, including the disease pathophysiology, host factors, the quantity of allergen ingested, and other ancillary factors such as exercise and intake of other foods and/or alcohol (Sicherer & Sampson, 2010; Sicherer & Teuber, 2004). The non-IgE-mediated food allergies are generally slower in onset and primarily produce gastrointestinal reactions but can affect the same organ systems that the IgE form affects.

**IgE- and non-IgE-mediated food allergies** IgE-mediated allergies are characterized by an acute onset of symptoms that typically involve the skin, respiratory system, cardiovascular system, and/or the gastrointestinal tract. Non-IgE-mediated or cell-mediated allergies are slower in onset and primarily are gastrointestinal reactions.

A single food is composed of many proteins, and these proteins have several areas to which the immune system can respond. These areas, called epitopes, can behave differently in response to a

**TABLE 7.7 Spectrum of Food Allergy Disorders**

**IgE-Mediated Reactions**
Oral allergy syndrome
Anaphylaxis
Urticaria

**Mixed IgE- and Non-IgE (Cell)-Mediated Reactions**
Allergic eosinophilic esophagitis
Allergic eosinophilic gastroenteritis
Atopic dermatitis
Asthma

**Non-IgE-Mediated Reactions**
Protein-induced enterocolitis
Protein-induced enteropathy
Eosinophilic proctocolitis
Contact dermatitis
Dermatitis herpetiformis

**TABLE 7.8 Symptoms of an Allergic Reaction**

**Cutaneous (skin symptoms)**
Itchy rash
Urticaria (hives)
Erythema (redness)
Angioedema (swelling of the face, lips, and tongue)
Atopic dermatitis (eczema)

**Gastrointestinal**
Itching of the lips, tongue, or mouth
Nausea and vomiting
Abdominal cramping and pain
Diarrhea

**Respiratory**
Watery and/or itchy eyes
Rhinitis (nasal congestion)
Sneezing
Dry cough
Itching or tightness in the throat
Tightness in the chest or shortness of breath
Wheezing

**Cardiovascular**
Hypotension (low blood pressure)
Syncope
Shock
Arrhythmia

food protein depending on the way the protein is folded. Recent studies have shown mapping of these epitopes to play a major role in determining the difference in outcome and outgrowing of food allergies. Although epitope sequencing looks promising for the future of food allergy diagnostics, further investigations using larger sample sizes are required before the technique should be applied in practice (Beyer et al., 2003, 2005; Lin & Sampson, 2009).

## Prevalence

It is difficult to determine prevalence of food allergy for several reasons, not the least of which is inconsistencies or deficiencies in study designs. For example, more than 170 foods have been reported to cause IgE-mediated reactions, but most studies focus on only the most common of these foods. Current consensus posits that in North America food allergy affects about 5% of infants and young children and approximately 3% to 4% of adults (Sicherer & Sampson, 2010). Prevalence of IgE-mediated food allergy in children with moderate to severe atopic dermatitis is higher and has been reported at about 35% (Eigenmann, Sicherer, Borowski, Cohen, & Sampson, 1998). Adverse reactions to food additives (nonprotein colors and preservatives) are rare, approximately less than 1% (Sicherer & Teuber, 2004).

The prevalence of an allergy to a specific food depends on societal eating patterns such as increased incidence of fish allergy in Scandinavian countries. In the United States, 2.5% of infants have milk allergy and 2.7% of the general population has peanut or tree nut allergy (Sicherer & Sampson, 2010). The specific food and food proteins in question and the mechanism of reactivity determine the clinical course and natural history of food allergy.

### Allergy Incidence and Immediate Treatment

Although about 30% of the general public believe they have food allergies, studies have shown that 3.5% to 4% of adults and approximately 5% of children have food allergies. Virtually any food protein can cause a reaction; however, only a small number of foods account for most adverse food reactions. The foods that most commonly cause allergic reactions in children are egg, milk, soy, wheat, peanuts, tree nuts, and fish. Adults most often react to peanuts, tree nuts, fish, and shellfish. The drug of choice for the treatment of severe or potentially severe food allergic reactions is epinephrine.

The most common food allergens in the pediatric population are milk, egg, wheat, soy, peanuts, tree nuts, fish, and shellfish, whereas peanuts, tree nuts, fish, and shellfish predominate in adults (Sicherer & Sampson, 2010). An 11-year follow-up study of peanut and tree nut allergy using a random-digit dial telephone survey found a tripling of prevalence of peanut allergy in American children aged 5 years and younger from 0.4% in 1997 to 1.4% in 2008 (Sicherer, Muñoz-Furlong, Godbold, & Sampson, 2010). Most childhood food allergies to cow's milk, egg, wheat, and soy are typically outgrown by 10 years of age and often as early as 5 years of age; however, allergy to peanut, tree nuts, and seafood is not commonly outgrown (Sicherer & Sampson, 2010).

## Other Reactions to Food Proteins

Several other types of reactions to food proteins might or might not be IgE-mediated. In this section, anaphylaxis, oral allergy syndrome, celiac disease, and allergic eosinophilic esophagitis/gastroenteritis are briefly described as a representative of these other allergic disorders.

### Food-Induced Anaphylaxis

**Anaphylaxis** is a rapid multisystem IgE-mediated food allergic reaction that potentially can be fatal. Fatal food-induced anaphylaxis in the United Stated has a frequency of about 200 per year and accounts for approximately 30,000 emergency room visits every year (Sampson, 2004b; Sampson et al., 2006). Any food protein can in essence cause anaphylaxis; however, the foods responsible for 80% to 90% of life-threatening anaphylactic reactions are peanuts, tree nuts (almond, brazil nuts, cashew, hazelnut, pecan, and walnuts), and seafood (Sampson, 2004b; Sampson et al., 2006). Individuals are at higher risk for fatal anaphylaxis when they delay treatment with epinephrine, have asthma, have experienced prior severe reactions, or deny ongoing symptoms (Sicherer & Teuber, 2004). In up to 20% of food-induced anaphylactic episodes, symptoms recur within 2 to 4 hours (Sampson et al., 2006).

**anaphylaxis** Sudden, severe, potentially fatal systemic allergic reaction that can involve various areas of the body, such as the skin, respiratory tract, gastrointestinal tract, and the cardiovascular system.

Food-associated exercise-induced anaphylaxis is a disorder in which eating a particular food (such as celery) before exercising results in anaphylaxis.

Less common is when anaphylaxis occurs after the ingestion of any food. Individuals with this disorder are able to eat any food, even the offending food, and exercise as long as each one is done separately and not in combination (Sampson, 2004b).

### Oral Allergy Syndrome

Oral allergy syndrome is an IgE-mediated condition that is characterized by pruritus (itching) and edema of the oral mucosa occurring after the ingestion of certain fresh fruits and vegetables (Ortolani, Ispano, Pastorello, Ansaloni, & Magri, 1989; Ortolani, Ispano, Pastorello, Bigi, & Ansaloni, 1988). The symptoms typically do not progress beyond the mouth. Individuals with sensitivity to pollens are susceptible because proteins found in pollens and in the fruits and vegetables are similar. For example, individuals with birch pollen allergy may experience symptoms after the ingestion of apple, peach, plum, nectarine, cherry, almond, hazelnut, carrots, and celery, whereas individuals with ragweed allergy may experience symptoms with consumption of banana, melons, and tomato (Ortolani et al., 1988, 1993). These proteins are heat labile; hence, cooked versions of these foods are typically tolerated. The cooking process denatures the responsible cross-reacting proteins in the foods and there is no reaction. Differentiation of symptoms of oral allergy syndrome from early symptoms of a systemic reaction to food (anaphylaxis) is critical.

### Celiac Disease or Gluten-Sensitive Enteropathy

Celiac disease is a non-IgE-mediated immunologic reaction to foods caused by hypersensitivity to gluten typically but not always presenting with flatulence, steatorrhea, and weight loss. Diarrhea, anemia, or metabolic bone disease can also be presenting symptoms of celiac disease. Hypersensitivity to gluten causes the disease, and the characteristic diagnostic feature is flattening of the intestinal villi in a biopsy of the jejunal mucosa upon gluten challenge. Improvement of symptoms upon initiation of a gluten-free diet has been noted in 2 to 3 weeks, although histologic improvements, if any, may take up 2 to 3 months (Freeman, Chopra, Clandinin, & Thomson, 2011; Jones, Robins, & Howdle, 2006).

### Allergic Eosinophilic Esophagitis/Gastroenteritis

These are a group of disorders of the gut characterized by eosinophilic inflammation and infiltration of the esophagus, stomach, and/or the small intestine. Symptoms in allergic eosinophilic esophagitis/gastroenteritis do overlap with other gastrointestinal disorders and may include dysphagia, vomiting, diarrhea, obstruction, and malabsorption (Kelly, 2000; Kelly et al., 1995; Liacouras, 2003, 2006; Lucendo & Arias, 2012; Orenstein et al., 2000; Rothenberg, 2004; Rothenberg, Mishra, Collins, & Putnam, 2001; Sampson & Anderson, 2000; Sampson, Sicherer, & Birnbaum, 2001). However, they are unresponsive to antireflux medications and most often have normal pH probe studies (Furuta et al., 2007; Justinich, 2000; Liacouras, 2006). A subset of these patients does not exhibit specific IgE antibody to foods (Spergel, Andrews, Brown-Whitehorn, Beausoleil, & Liacouras, 2005). Histopathologic confirmation of significant eosinophilic infiltration of the esophagus, stomach, or small intestine via an endoscopy and biopsy is needed, although it does not identify the responsible allergen (Liacouras et al., 2005; Lucendo & Arias, 2012; Sampson et al., 2001). Though medical treatment has proven somewhat successful using corticosteroids, current management through diet includes eliminating all incriminating foods, although the number of foods involved may prohibit the long-term use of such a diet. Use of an elemental formula has been shown to be efficacious in some patients (Justinich et al., 1996; Kelly et al., 1995; Liacouras, 2006; Markowitz, Spergel, Ruchelli, & Liacorous, 2003).

## Diagnostic Evaluation

Once food allergy is identified as a likely cause of symptoms, confirmation of the diagnosis and identification of the implicated food(s) can proceed. Diagnosis of food allergy requires obtaining a careful history (including a thorough medical and reaction history), physical examination, a food and symptom diary, allergy testing (skin prick and food-[serum-]specific IgE antibody testing or serum immunoasssays), and food challenges to confirm the identified or suspected offending allergens (Bock, 2000; Sampson, 2004b; Sicherer, 2002; Sicherer & Teuber, 2004; Wang & Sampson, 2011). In cases of acute reactions, as in urticaria or anaphylaxis, the history is very important and may clearly identify the causal food. In chronic disorders, such as asthma or atopic dermatitis, identification of a particular food may be more difficult. In cases of gastrointestinal reactions, similar problems exist

because most often delayed reactions after ingestion of causal foods are noted, making the identification of the food somewhat difficult.

### History and Physical Examination

Diagnosis is a team effort, and involvement of the child, the child's family, the dietitian, and the physician is instrumental in correctly identifying the allergenic foods. The physician should be able to conclude from the history and physical examination whether an allergy or food intolerance is part of the differential diagnosis. The history should provide a description of symptoms, the length of time between ingestion and the development of symptoms, whether ingesting the suspected food produces similar symptoms on different occasions, quantity of food required to produce symptoms, whether other factors such as exercise or alcohol ingestion are necessary to induce symptoms, and the length of time since the last reaction occurred (Bock, 2000; Sampson, 1999, 2004b). The physical examination focuses on the exclusion of nonallergic causes of food-induced symptoms in addition to evaluating disease severity. Also, in performing the physical examination, the overall nutritional status of the child should be assessed, particularly if the child has undergone a prolonged overly restrictive diet.

### Diet and Symptom Diaries

Diet diaries are an important component of the diagnostic process. Besides providing the ability to determine adequacy of the diet, they serve as another form of recall of foods eaten and timing of symptoms provoked. Details such as time of the meal or snack, brand name if commercially prepared and/or specific ingredients if homemade, amount consumed, and also symptoms noted are recorded in the diary. If the parent can provide the actual labels from the foods consumed during the time the diary was kept, it would further enhance the evaluation. Reviewing the diet diaries can be a starting point for the dietitian and the physician to identify a particular food that is common to different products and is repeatedly causing similar symptoms. It can also help reveal hidden sources of the food allergen or unknown sources of contamination and provide a listing of foods that can help facilitate directive counseling. Utilizing alternative foods similar to what is recorded in the diet diary can enhance compliance to the restricted diet significantly.

### Laboratory Studies

Food-specific IgE antibody testing can help provide more information regarding either identifying or excluding IgE-mediated food allergies. Skin prick tests (SPTs) and serum immunoassays (formerly called radioallergosorbent tests or RASTs) are useful standardized screening tools that establish presence of foodallergen-specific IgE (Bock, 2000; Sampson & Ho, 1997; Sicherer & Sampson, 2010; Sicherer & Teuber, 2004). It is important to stress that these tests do not indicate symptomatic clinical reactivity. These test results must be interpreted in combination with information from the child's history, physical examination, and diet/symptom diaries.

In SPTs, commercial food antigen extracts (foods being tested) and positive (histamine) and negative (saline) controls are applied by the prick or puncture method using a bifurcated needle or multipurpose plastic device. In a sensitized individual, food allergen binds to specific IgE antibody present on the surface of the mast cells in the skin and causes degranulation of mast cells. Histamine released from mast cells leads to a flare or erythema and wheal response within 10 to 15 minutes. Some studies have attempted to identify parameters by which wheal size can confirm food allergy, as in the case of Sporik, Hill, and Hosking (2000), whose study posited that infants and young children with a wheal size greater than 8 to 10 mm were associated with a greater than 95% likelihood of clinical reactivity to cow's milk, egg, and peanuts. These studies, however, are limited in the foods and populations chosen and in the techniques used. Currently, additional studies are needed for the further development of wheal-size diagnostic parameters (Sicherer & Sampson, 2010).

There is a high false-positive rate associated with skin tests (about 50%), although negative predictive accuracy is greater than 95% (Sampson, 2004b). Hence, a negative SPT to a particular food excludes an IgE-mediated reaction and a positive SPT in isolation cannot be proof of clinical reactivity. However, a positive SPT in an individual with a history of systemic anaphylactic reactions after ingestion of the isolated food may be considered diagnostic (Sampson, 2004b; Sicherer & Teuber, 2004). Commercial fruit and vegetable extracts may lack the proteins responsible for IgE-mediated sensitivity because they are susceptible to protein degradation (Ortolani et al., 1989; Sampson, 2004b). Fresh food extracts can be especially useful in detecting sensitivity to fruits and vegetables. The prick–prick method, which involves pricking the fresh food and then pricking the skin of the child, is most beneficial in situations where raw fruits and/or vegetables are symptomatic but cooked versions are tolerated without any problems.

The child undergoing skin testing (SPT) should be off antihistamines; otherwise, it may result in a false-negative test to the particular food tested. Histamine wheals less than 3 to 5 mm in diameter may indicate the presence of an interfering antihistamine.

Food-specific IgE antibody serum immunoassays also measure the presence of food-specific IgE antibodies, though they have been regarded in the past as less sensitive than SPTs. A negative test (food-specific IgE antibody <0.35 kIU/L) has a greater than 95% predictive value; however, a positive blood test has low specificity (Sampson, 2004b; Sicherer & Teuber, 2004). The advantage of blood tests in the evaluation of a food-allergic child is that levels are not affected by antihistamine use. Although these tests also have the same inherent problems as do SPTs, a negative blood test virtually excludes an IgE-mediated sensitivity, but a positive result in isolation cannot be proof of clinical reactivity.

Studies have looked at the positive predictive value of only one type of serum immunoassay to date, the ImmunoCAP test in children undergoing oral food challenges (Sampson, 2001; Sampson & Ho, 1997). Clinical decision points indicating greater than 95% likelihood of reaction have been established for the most common food allergens, including milk (15 kU/L), egg (7 kU/L), peanut (14 kU/L), tree nuts (~15 kU/L), and fish (20 kU/L). These numbers

indicate that a child older than 2 years with a milk IgE antibody of at least 15 kU/L is highly likely (>95%) to react if milk was ingested accidentally or during a milk challenge. This would, in addition, imply that a milk challenge should be deferred unless there is convincing history that the child tolerates a significant quantity of milk without a reaction. Milk-specific IgE of 5 kU/L for a child aged 12 months or younger and egg-specific IgE of 2 kU/L for a child aged 24 months or younger have been shown to have a 95% positive predictive value (Sampson, 2001). Though research is limited, the ImmunoCAP has become instrumental in the diagnosis and evaluation of food allergies because of its ability to provide diagnostic values by which to assess food allergen reactivity. Additional research is needed to confirm the positive predictive cut-off points for other types of serum immunoassays such as the Immulite or Turbo RAST (Wang, Godbold, & Sampson, 2008; Wood, Segall, Ahlstedt, & Williams, 2007).

At this point in time, current diagnostic laboratory methods such as SPT and food-specific IgE cannot distinguish between individuals who will outgrow their food allergies and those who will not. Ongoing research using the microarray technology focuses on epitope recognition patterns that can be used to identify markers of persistent food allergy. This information can be significant when therapy for food allergy other than dietary avoidance becomes available (Beyer et al., 2003, 2005; Lin & Sampson, 2009).

Other forms of skin testing, such as intradermal or patch testing, can be used in the diagnostic process; however, there are inherent problems with these tests. Intradermal allergy skin tests are contraindicated because they have high false-positive rates and have been associated with systemic reactions (Sampson, 2004b; Sicherer & Teuber, 2004). The atopy patch test has been recently proposed as a useful screening tool in children with atopic dermatitis in diagnosing late-phase clinical reactions (Niggemann, Reibel, & Wahn, 2000; Roehr et al., 2001). More research is needed to study other subject groups, such as adults, and also to study the atopy patch test more closely in relation to optimal concentration of the food used in the patch test and in correlation with double-blind placebo-controlled food challenges before using this as another widespread screening tool for diagnosing food allergies.

A number of unproven tests should not be used, and they include provocation neutralization, cytotoxic test, applied kinesiology, hair analysis, and $IgG_4$ testing, among others (Beyer & Teuber, 2005; Ortolani et al., 1999; Sicherer & Teuber, 2004; Terr & Salvaggio, 1996).

A negative skin prick test or food-specific IgE in the blood essentially excludes IgE-mediated reactivity. A positive skin prick test or food-specific IgE in the blood indicates the presence of IgE antibody and does not represent symptomatic clinical reactivity. However, a positive SPT or food-specific IgE in the blood in an individual with a history of systemic anaphylactic reactions after ingestion of the isolated food may be considered diagnostic. Any food, whether it has a negative or positive skin and/or blood result, that has been identified through the history to have provoked a serious reaction should only be reintroduced under physician supervision with emergency medication immediately available.

### Elimination Diets and Oral Food Challenges

The next step in the diagnostic evaluation is confirming that the foods identified through testing are in fact problem foods. Elimination diets and oral food challenges both have a significant role in this process. Selection of single-ingredient foods without any contamination with other allergens is critical in this verification process.

A negative skin prick test (SPT) or a negative food-specific IgE blood test (sIgE) (<0.35 kU/L) essentially excludes IgE-mediated reactivity, and reintroduction of the particular food is not likely to elicit a reaction, at least acutely (Siles & Hsieh, 2011). However, if that same food with negative skin and/or blood test results has been identified through the history to have provoked a serious reaction, it should be reintroduced only under physician supervision with emergency medication immediately available (Mofidi & Bock, 2005).

If test results are positive, an elimination diet can be considered to determine whether the symptoms are caused by the foods that tested positive on either skin or blood test. If the elimination diet fails to show resolution of the underlying disorder, the foods can be reintroduced to the diet unless, again, a convincing history warrants a supervised food challenge. The possibility that the wrong foods were eliminated or the presence of some level of contamination has to be considered if there is no resolution of symptoms. Recent studies are testing specific components of allergens based on their potential for cross-reactivity (e.g., peanuts and grass or birch and hazelnuts) to potentially prevent the false-positive results seen in current SPT or sIgE methods and, thereby, avoid the arduous elimination diet process (Bublin et al., 2010; Hansen et al., 2009; Nicolaou et al., 2010).

If the elimination diet results in resolution of symptoms, an oral food challenge should be considered so foods can be added back to the diet one by one. Oral food challenges would not be appropriate for severe reactions of isolated food ingestion with a positive food-specific IgE antibody. These patients are at high risk for severe and potentially life-threatening reactions. Any food, whether it is one of the major allergens or not, that has been reported to provoke a serious reaction should only be reintroduced under physician supervision with emergency medications immediately available (Mofidi & Bock, 2005; Sicherer, 1999).

If any foods were eliminated from the child's diet based on positive skin and/or blood results even though there is a history of tolerance of those specific foods before the allergy testing, possible reintroduction of those foods to the child's diet should be discussed with the physician. If reintroduction of the foods is suggested, each food should be introduced one at a time over 5 to 7 days so any adverse reaction can be identified and further investigated. Some of the foods may also need to be introduced under physician supervision with emergency medication available.

There is no simple supporting laboratory test for non-IgE-mediated disease, and in many cases histopathologic confirmation via an endoscopy is required (Kelly, 2000; Liacouras, 2006; Noel, Putnam, Collins et al., 2004; Potter et al., 2004; Wang & Sampson, 2011). However, a positive biopsy for eosinophils does not specifically identify the responsible allergen in the diet. Elimination diets and oral food challenges are therefore needed to support this diagnosis as well. If an elimination diet results in resolution of symptoms, an oral food challenge is performed to identify the particular foods involved. Symptoms from ingestion of particular foods in enteropathy syndromes or eosinophilic esophagitis/gastroenteritis may require several days of ingestion to elicit symptoms. In these cases, foods are added one at a time with at least 1 to 2 weeks between each food introduction. The length of time between each food introduction depends on the specific food and the reported history in relation to that food (Mofidi & Bock, 2005). In food protein–induced enterocolitis syndrome, which is a symptom complex of profuse vomiting and diarrhea, symptoms completely resolve when the responsible food protein is removed from the diet. Most often cow's milk or soy are responsible for the symptoms observed in food protein-induced enterocolitis syndrome, although other food proteins such as egg, wheat, rice, oat, chicken, and fish have also been noted to induce enterocolitis symptoms (Nowak-Wegrzyn, Sampson, Wood, & Sicherer, 2003; Sicherer, 2000).

### Elimination Diets

**Elimination diets** are central to the diagnosis and treatment of food allergies and when used in the diagnostic process should be for a specified trial period. The symptoms attributed to the food should resolve and then reappear when the food is reintroduced. The duration of the diet can be from 1 week (for acute symptoms such as hives) to 8 weeks (for chronic symptoms such as vomiting and diarrhea), depending on the underlying suspected disorder. Success of the elimination diet depends on the correct identification of the allergen(s) and complete exclusion of the allergen(s) from the diet. In some gastrointestinal food allergies, an elemental diet using amino acid–based formulas may be required before significant resolution of gut pathology can be noted via endoscopy and biopsy.

**elimination diets** Diets in which suspected foods are eliminated in an attempt to identify and/or confirm offending allergenic foods.

When elimination diets are used in the management and essentially as the treatment of food allergies, education becomes the focal point for not only the healthcare providers but also for the child and the child's family. The physician and the dietitian teach the food-allergic individual and his or her family the principles of daily management of food allergies. The individual or his or her family will take on the role of the educator to teach other family members, teachers, and staff at day cares, schools, and camps and others involved in the day-to-day life of the food-allergic individual (Muñoz-Furlong, 2003). All involved in the care of the allergic individual need to be educated on the basic principles of food allergy because an accident or a simple mistake can lead to a reaction ranging from mild discomfort to life-threatening anaphylaxis.

The principles of management are quite simple in theory but can be quite complicated upon implementation. The identified allergenic food(s) must be removed from the diet while adequate nutrition is provided to promote appropriate growth and development despite the limitations of some of the major contributors to the diet. This is most critical in the management of food allergies in infants and children. The nutritional quality of the diet depends on the number of major foods restricted, the availability of appropriate food substitutes, and the palatability of the diet (Mofidi, 2003). It is certainly not necessary to limit an entire food family; the focus of the treatment plan is to minimize the list of restricted foods.

There are three types of elimination diets:

1. Elimination of one or several foods suspected of provoking symptoms
2. Elimination of all but a defined group of "allowed" foods, also called the "eat-only" diet
3. Use of an amino acid–based formula, or the elemental diet

The type of elimination diet used depends on the clinical situation being evaluated, the age of the child, the reported history, and the results of tests for food-specific IgE antibody (skin and blood). The first type of elimination diet is useful when an isolated food ingestion provokes a sudden acute reaction and there is a positive IgE to the food. By avoiding the specific food in all forms, symptoms should resolve (Mofidi, 2003).

When more than one particular food allergen is suspected, a limited eat-only diet is prescribed. The

foods allowed are those that cannot be related to an isolated reaction or that were negative on skin and/or blood testing. Individualization is critical in this type of elimination diet, not only for selection of the allowed foods but also for compliance. If the symptoms attributed to the foods still persist, a thorough review of the diet is necessary to ensure that no other food allergens have been inadvertently added to the diet. If a source of contamination or mistake cannot be found, reassessment of the allowed foods may be necessary to determine whether the causal food is still in the diet (Mofidi, 2003).

In the elemental diet, all nutrient needs are essentially obtained from the amino acid–based formula. Occasionally, one or two foods are allowed to provide textures to the individual on the elimination diet. Elemental diets are generally required for gastrointestinal food allergies such as **allergic eosinophilic esophagitis** and **allergic eosinophilic gastroenteritis**. Studies have looked at the use of systemic and inhaled corticosteroids in allergic eosinophilic esophagitis and have noted both clinical and histologic improvements (Liacouras, Wenner, Brown, & Ruchelli, 1998; Lucendo & Arias, 2012). However, the symptoms often recur upon the discontinuation of the medications (Liacouras et al., 2005; Noel, Putnam, & Rothenberg, 2004). The elemental diet has become a fundamental part of the treatment of these gastrointestinal food allergies (Kelly, 2000; Kelly et al., 1995; Liacouras, 2006; Markowitz et al., 2003; Spergel et al., 2005). It allows healing of the gut during the elimination process and further allows the identification and hence restriction of the offending food allergen during the slow introduction of foods. Upon accurate identification and restriction of the suspect foods, these individuals are symptom free and do not require medications. Even though all foods have been implicated in allergic eosinophilic esophagitis, the most common foods have been milk, eggs, nuts, beef, wheat, fish, shellfish, corn, and soy (Liacouras, 2006; Spergel et al., 2005).

**allergic eosinophilic esophagitis** A disease involving patchy infiltration of one or more layers of the esophagus with eosinophils.

**allergic eosinophilic gastroenteritis** A disease involving patchy infiltration of one or more layers of the stomach and/or small intestine with eosinophils.

*Label Reading*

Dietary elimination of any of the major allergens, such as egg, milk, soy, wheat, or peanut, is not a simple procedure. Particular allergens may be hidden in unsuspected foods, such as milk or egg proteins in bread products, milk or soy protein in canned tuna, peanut flour in cakes, sauces, or chili, and peanut butter as the glue that holds egg rolls together (Steinman, 1996). Education regarding, for example, elimination of egg protein from the diet includes not only avoidance of egg-based foods (mayonnaise, ice cream, and quiche) but also learning to identify words used on food labels that may indicate the presence of egg protein as a hidden ingredient in the food (Mofidi, 2003). When looking at a food label the parent should be able to recognize terms that can be easily identified to be an egg by-product (egg white), terms that are not so obvious (ovomucoid), or terms that may or may not be egg-based (lecithin, natural flavor).

"How to read a label" cards are available from the Food Allergy and Anaphylaxis Network (FAAN, a nonprofit organization located in Fairfax, Virginia) to assist patients, their families, dietitians, and physicians in this very difficult task of eliminating particular food allergens (see **Table 7.9**). FAAN provides accurate and current information on food allergies with resources ranging from handouts and cookbooks to videos and bimonthly newsletters. In addition, FAAN sponsors conferences to educate professionals and individuals about managing food allergies. The organization has numerous materials for dealing with travel, school, and eating out in addition to a dedicated website for children and teenagers dealing with specific topics related to those age groups and compliance issues.

**TABLE 7.9** FAAN's How to Read a Label Sheets

**How to Read a Label for a Milk-Free Diet**

Avoid foods that contain milk or any of these ingredients:

- artificial butter flavor
- butter, butter fat, butter oil
- buttermilk
- casein (casein hydrolysate)
- caseinates (in all forms)
- cheese
- cream
- cottage cheese
- curds
- custard
- ghee
- half & half
- lactalbumin, lactalbumin phosphate
- lactoferrin
- lactulose
- milk (in all forms including condensed, derivative, dry, evaporated, goat's milk and milk from other animals, low-fat, malted, milkfat, non-fat, powder, protein, skimmed, solids, whole)
- nisin
- nougat
- pudding
- recaldent
- rennet casein
- sour cream, sour cream solids
- sour milk solids
- whey (in all forms)
- yogurt

**May indicate the presence of milk protein:**

- carmel candies
- chocolate
- flavorings (including natural and artificial)
- high protein flour
- lactic acid started culture
- lactose
- luncheon meat, hot dogs, sausages
- margarine
- non-dairy products

**How to Read a Label for an Egg-Free Diet**

Avoid foods that contain eggs or any of these ingredients:

- albumin (also spelled as *albumen*)
- egg (dried, powdered, solids, white, yolk)
- eggnog
- lysozyme
- mayonnaise
- meringue (meringue powder)
- surimi

**May indicate the presence of egg protein:**

- flavoring (including natural and artificial)
- lecithin
- macaroni
- marzipan
- marshmallows
- nougat
- pasta

**How to Read a Label for a Peanut-Free Diet**

Avoid foods that contain peanuts or any of these ingredients:

- artificial nuts
- beer nuts
- cold pressed, expelled, or extruded peanut oil
- goobers
- ground nuts
- mixed nuts
- monkey nuts
- nutmeat
- nut pieces
- peanut
- peanut butter
- peanut flour

**May indicate the presence of peanut protein:**

- African, Asian (especially Chinese, Indian, Indonesian, Thai, and Vietnamese), and Mexican dishes
- baked goods (pastries, cookies, etc.)
- candy (including chocolate candy)
- chili
- egg rolls
- enchilada sauce
- flavoring (including natural and artificial)
- marzipan
- mole sauce
- nougat

Also note:

- Mandelonas are peanuts soaked in almond flavoring.
- Studies show most allergic individuals can surely eat peanut oil (*not* cold pressed, expelled, or extruded peanut oil).
- Arachis oil is peanut oil.
- Experts advise patients allergic to peanuts to avoid tree nuts as well.
- A study showed that unlike other legumes, there is a strong possibility of cross reaction between peanuts and lupine.
- Sunflower seeds are often produced on equipment shared with peanuts.

**TABLE 7.9** **FAAN's How to Read a Label Sheets (*Continued*)**

**How to Read a Label for a Wheat-Free Diet**
Avoid foods that contain wheat or any of these ingredients:

- bran
- bread crumbs
- bulgur
- club wheat
- couscous
- cracker meal
- durum
- einkorn
- emmer
- farina
- flour (all purpose, bread, cake, durum, enriched, graham, high gluten, high protein, instant, pastry, self-rising, soft wheat, steel ground, stone ground, whole wheat)
- gluten
- kamut
- matzoh, matzoh meal (also spelled as matzo)
- pasta
- seitan
- semolina
- spelt
- triticale
- vital gluten
- wheat (bran, germ, gluten, malt, sprouts)
- wheat grass
- whole wheat berries

**May indicate the presence of wheat protein:**

- flavoring (including natural and artificial)
- hydrolyzed protein
- soy sauce
- starch (gelatinized starch, modified starch, modified food starch, vegetable starch, wheat starch)
- surimi

**How to Read a Label for a Soy-Free Diet**
Avoid foods that contain soy or any of these ingredients:

- edamame
- hydrolyzed soy protein
- miso
- natto
- shoyu sauce
- soy (soy albumin, soy fiber, soy flour, soy grits, soy milk, soy nuts, soy sprouts)
- soya
- soybean (curd, granules)
- soy protein (concentrate, isolate)
- soy sauce
- tamari
- tempeh
- textured vegetable protein (TVP)
- tofu

**May indicate the presence of soy protein:**

- Asian cuisine
- flavoring (including natural and artificial)
- vegetable broth
- vegetable gum
- vegetable starch

Also note:

- Studies show most individuals allergic to soy may safely eat soybean oil.
- Most individuals allergic to soy can safely eat soy lecithin.
- Check with your doctor if you have questions about these ingredients.

**How to Read a Label for a Shellfish-Free Diet**
Avoid foods that contain shellfish or any of these ingredients:

- abalone
- clams (cherrystones, littleneck, pismo, quahog)
- cockle (periwinkle, sea urchin)
- crab
- crawfish (crayfish, ecrevisse)
- lobster (langouste, langoustine, scampo, coral, tomalley)
- mollusks
- mussels
- octopus
- oysters
- prawns
- scallops
- shrimp (crevette)
- snails (escargot)
- squid (calamari)

**May indicate the presence of shellfish protein:**

- bouillabaisse
- cuttlefish ink
- fish stock
- flavoring (including natural and artificial)
- seafood flavoring (such as crab or clam extract)
- surimi

Also note:

- Any food served in a seafood restaurant may be cross contaminated with fish or shellfish.

(*Continued*)

**TABLE 7.9** **FAAN's How to Read a Label Sheets (*Continued*)**

- For some individuals, a reaction may occur from cooking odors or from handling fish or shellfish.
- Always carry medications and use them as soon as symptoms develop.

**How to Read a Label for a Tree-Nut-Free Diet**

Avoid foods that contain nuts or any of these ingredients:

- almonds
- artificial nuts
- beech nut
- Brazil nuts
- butternut
- caponata
- cashews
- chestnuts
- chinquapin
- coconut
- filberts/hazelnuts
- gianduja (a nut mixture found in some chocolate)
- ginko nut
- hickory nuts
- lichee/lychee nut
- macadamia nuts
- marzipan/almond paste
- nan-gai nuts
- natural nut extract (i.e., almond, walnut)
- nougat
- nut butters (i.e., cashew butter)
- nut meal
- nutmeat
- nut oil
- nut paste (i.e., almond paste)
- nut pieces (Mashuga Nuts®)
- pecans
- pesto
- pili nut
- pine nuts (also referred to as Indian, piñon, pinyon, pignoli, pignolia, and pignon nuts)
- pistachios
- praline
- sheanut
- walnuts

Also note:

- Mandelonas are peanuts soaked in almond flavoring.
- Mortadella may contain pistachios.
- Natural and artificial flavoring may contain tree nuts.
- Experts advise patients allergic to tree nuts avoid peanuts as well.
- Talk to your doctor if you find other nuts not listed here.



The Food Allergen Labeling and Consumer Protection Act (FALCPA) took effect on January 1, 2006. This labeling law requires simple terms and plain language to be used to indicate the presence of the top eight food allergens in packaged foods. Labels must either identify an allergen, for example, casein, in a product with the words "contains milk" or declare it immediately after the term such as "albumin (egg)." These ingredients are to be listed even if they are present in colors, flavors, or even spice blends. Ideally, this new law should help with identification of safe foods for allergic individuals.

### *Nutritional Issues*

Teaching the parent how to replace, for example, egg in the diet by providing an alternate source of nutrients is essential. It is also important to provide appropriate substitutions so egg-free baked goods with appropriate texture and taste can be prepared. Palatability of these products determines the degree of compliance with the elimination diet. Cookbooks from FAAN and other resources are available with recipes and substitution guidelines that do not contain any milk, egg, soy, wheat, peanuts, or tree nuts.

When one food or a food group is eliminated from the diet, alternative sources of nutrients that are lost through the elimination diet need to be identified. For example, if the allergy is to milk, which is a major contributor to the diet, all dairy products need to be avoided. Hence, calcium, phosphorus, riboflavin, pantothenic acid, vitamin $B_{12}$, vitamin A, and vitamin D need to be supplied from other sources. It is rather difficult to obtain calcium from nondairy foods. There are, however, alternate milks (rice, soy, and potato) and juices (orange, apple, and grape) that are enriched with calcium. Also, some milk-free calcium supplements are available. Intake of other foods in the diet needs to examined to determine whether adequate amounts of, for example, vitamin A are provided and, if

**TABLE 7.10** **Problem Nutrients in Allergen-Restricted Diets**

| Allergen | Nutrients |
|---|---|
| Milk | Vitamin A, vitamin D, riboflavin, pantothenic acid, cyanocobalamin, calcium, and phosphorus |
| Egg | Cyanocobalamin, riboflavin, pantothenic acid, biotin, and selenium |
| Soy | Thiamin, riboflavin, pyridoxine, folate, calcium, phosphorus, magnesium, iron, and zinc |
| Wheat | Thiamin, riboflavin, niacin, iron, and folate if fortified |
| Peanut | Vitamin E, niacin, magnesium, manganese, and chromium |

necessary, to stress intake of foods high in vitamin A. **Table 7.10** provides a listing of the problem nutrients in milk-, egg-, soy-, wheat-, and peanut-free diets (Mofidi, 2003). Obviously, the distribution of carbohydrates, protein, and fats is altered and needs to be addressed in an elimination diet. The RDA for age and gender can be used as a guide to provide the child with appropriate nutrition.

Manufacturers change product ingredients and production procedures frequently, where products that were once safe may become unsafe and vice versa. Education of the family dealing with food allergies and specifically focusing on skills necessary to proceed with evaluating and identifying what is safe and what is not are critical for the management of food allergies. Thorough guidance, which may include providing information on label reading, brand identification, what to inquire when calling the manufacturers, restaurant dining, and recipe modification, is vital to ensure nutritional adequacy as well as management of symptoms.

*Cross-Contamination*

Another major source of hidden allergens is cross-contamination. Contact of one food with another even in trace amounts can pose a problem for a highly allergic child. Cross-contaminations can occur as a result of processing errors, where an ingredient is added to a batch of food by mistake, or by processing on shared lines, where the equipment is not adequately cleaned. It also can occur when purchasing food items from bulk bins or at a deli counter, where, for example, the same slicer is used for cutting both meats and cheese, or from accidents in food preparation at home where the same knife is used for peanut butter and then simply wiped clean and used for jelly to make a sandwich. In the food industry, the use of leftover products, or "rework," to the next batch of products is a common practice. For example, the addition of a small amount of cookie dough that has had the nuts filtered out to a new batch of chocolate chip cookies will add nut residues and possibly enough protein to elicit an allergic reaction in a nut-allergic child.

There are also nonedible sources of food allergens, such as milk or peanut products in pet foods, hacky sacks or bean bags filled with walnut shells, shampoos, cosmetics, or other body care products with a variety of food allergens (Bernhisel-Broadbent, 1999). Reviewing all ingredients and contacting the manufacturers regarding vague terms used on the labels are requirements in allergen avoidance.

### Oral Food Challenges

**oral food challenges** A procedure during which the patient eats or drinks the suspected food allergen gradually in small portions over a given period of time under a physician's supervision.

**Oral food challenges** are used to identify, confirm, or rule out a suspected allergy to food(s) in both IgE-mediated and non-IgE-mediated food allergies; oral food challenge remains the most accurate method for diagnosing food allergy (Bock et al., 1988; Mofidi & Bock, 2005; Sicherer, 1999; Wang & Sampson, 2011). It can also be used to determine whether clinical reactivity to a food is lost. When several foods are in question as possible causal foods and elimination of those foods from the diet resulted in resolution of symptoms, an oral food challenge to each food is warranted. An oral food challenge can also be used for complaints that are not typically associated with food allergy (headache, behavior, etc.). Food challenge in a child with a recent history of severe anaphylaxis to an isolated ingestion with positive specific IgE antibody to the particular food is contraindicated, although a physician-supervised follow-up challenge to determine resolution of the allergy may be indicated in some settings.

Several types of food challenges can be used depending on the history, results of IgE tests, and resources available to the physician. Food challenges can be open (parent and physician are aware of the challenge content), single blind (parent is unaware, but the physician is aware of the challenge content), or a double-blind placebo-controlled food challenge (neither parent nor the physician is aware of the challenge contents) (Mofidi & Bock, 2005; Sicherer, 1999). The double-blind placebo-controlled food challenge is considered the gold standard for the diagnosis of food allergy (Sampson, 2004a; Sicherer & Teuber, 2004; Wang & Sampson, 2011). In a clincal setting, use of double-blind placebo-controlled food challenges is recommended because bias in the diagnosis of allergy to a particular food is removed from all parties; however, it is very labor intensive. The advantage of an open challenge is that it is quick and easy and a good screening tool.

Food challenges are generally performed in a fasting state with gradually increasing amounts of the food given with the dose and the timing individualized to the child's history for a total of 60 to 90 minutes (Mofidi & Bock, 2005; Sicherer, 1999; Wang & Sampson, 2011). The child is evaluated for the development of symptoms throughout the challenge. Medical supervision and immediate access to emergency medications, including epinephrine, antihistamines, steroids, and inhaled beta agonists, in addition to equipment for cardiopulmonary resuscitation are required because reactions can be severe (Bock et al., 1988; Sicherer, 1999; Sicherer & Teuber, 2004). Challenges are terminated when a reaction becomes apparent, followed by administration of emergency medication. Patients are also observed and monitored for delayed reactions.

Negative challenges are always followed by an open feeding of a normal serving size of the challenge food to rule out a false-negative challenge (Mofidi & Bock, 2005). If the child can tolerate this portion of the challenge without any symptoms, the specific food can be added back to the diet.

The selection of the food for food challenges is determined by the history and results of IgE tests. Foods that are identified by positive skin or blood tests without a significant history of a reaction or that are unlikely to provoke a reaction can be screened by open challenges. In IgE-mediated food allergies, a negative food challenge is definite proof that the patient is no longer sensitive to the particular food and allows the addition of the food to the diet (Mofidi & Bock, 2005). Suspected food allergens should be eliminated for 7 to 14 days before the food challenge in IgE-mediated disorders and up to 12 weeks in some gastrointestinal disorders. Before the challenge the child should be off antihistamines long enough to promote a normal histamine response (Sampson, 1999, 2004b).

## Prevention of Food Allergies

Preliminary studies have looked at timing of exposure to allergenic foods and tried to generate reasonable recommendations for prevention of food allergies. Exclusive breastfeeding and introduction of solid foods after 4 to 6 months of age have been shown to be associated with decreased risk of atopic dermatitis and cow's milk allergy in infants with an atopic predisposition or positive family history of food allergies (American Academy of Pediatrics [AAP] Committee on Nutrition, 2008). If breastfeeding is not possible, extensively hydrolyzed formulas may potentially prevent atopic disease and food allergy, but, notably, studies to date are limited to delineating the benefits of these hydrolyzed formulas on infants at high risk for atopy (von Berg et al., 2003; Zeiger, 2003).

Avoidance of highly allergenic foods such as peanuts has not been shown to have a consistent protective effect when consumed during pregnancy and/or lactation. Additionally, more recent studies posit that the timing of food exposure may differ depending on the food (Wang & Sampson, 2011). Exposure to egg, for example, by 4 to 6 months may be protective, whereas delaying introduction of cow's milk in this same time frame may create the highest risk in infants for milk allergy (Katz et al., 2010; Koplin et al., 2010). Whereas previous recommendations suggested potential benefits of delaying the introduction of highly allergenic foods such as peanuts, tree nuts, and seafood to 3 to 4 years of age, a more current review of research shows no conclusive evidence for benefits beyond the recommendation to introduce solids at 4 to 6 months of age.

## Case Study 1

### An Overweight Vegetarian

Reed Mangels, PhD, RD, LD

Jacob is a 16-year-old lifelong lacto-ovo-vegetarian. His father has recently discovered he has elevated cholesterol. Jacob also has borderline high cholesterol and is about 20 pounds over his ideal weight.

A typical intake for a day for Jacob includes the following:

- Breakfast: Three scrambled eggs with melted cheese, three slices of toast with butter, and chocolate milk
- Lunch: Three slices of cheese pizza, french fries, and a milkshake
- Dinner: A large serving of macaroni and cheese, two slices of bread and butter, whole milk, and a bowl of ice cream
- Snacks include potato chips, soft drinks, and candy bars

#### Questions

1. What are your concerns with Jacob's diet?
2. What modifications can you suggest?

## Case Study 2

### An Underweight Vegetarian

Reed Mangels, PhD, RD, LD

Mollie is a 15-year-old dedicated cross-country runner. During the season (now), she runs 50 to 60 miles weekly. She has been a lacto-ovo-vegetarian for the past 2 years. She doesn't drink much milk, however, because she is lactose intolerant.

Mollie's parents are concerned because she has unintentionally lost 5 lb in the past month. Mollie is 5 foot 5 inches and weights 105 lb (BMI = 18). Mollie is concerned because she seems to get sick easily and doesn't have as much energy as she would like.

Mollie says she just doesn't have time to eat well. A typical day includes the following diet:

- Dry cereal with fruit for breakfast
- A veggie burger and fries for lunch
- A big salad with cheese cubes and beans and salad dressing along with a couple of rolls for dinner
- Snacks include air-popped popcorn and iced tea

Her estimated calorie requirement is 2,200 kcal whereas her estimated intake is 1,500 kcal.

#### Questions

1. What can Mollie do to improve her diet?
2. How can she get extra food when she has no time to cook or prepare food?

## Case Study 3

### Vegetarian Toddler

Julia Driggers, RD, LDN, CNSC

Isaac is a 4½-year-old vegan male who was referred to you [an outpatient dietitian] by a family physician with concern for dietary adequacy. Isaac is 105 cm (3 foot 5 inches) and weighs 15 kg (33 lb) with a BMI of 14.5, indicating normal growth with no weight loss. Isaac's family reports that during the last 3 months he has demonstrated a decreased energy level and has trouble focusing.

Isaac's parents changed the family's eating pattern from a vegetarian to a vegan diet a year ago. Since that time, Issac has been fed primarily unfortified grains, beans, lentils, fruits, vegetables, dark leafy greens, and occasionally tofu processed with calcium sulfate or meat analogues. He drinks juice, water, and 6 oz of fortified soy milk per day. He is offered three meals a day with two snacks and typically finishes 75–100% of his plate. He has never taken any vitamin supplements and no additional vitamin or protein powders are used.

Laboratory work taken prior to Isaac's visit included the following serum values: hemoglobin 9.8 g/dL (low), hematocrit 29 g/dL (low), mean corpuscular volume 70 fL (low), ferritin 5 ng/mL (low), indicating a possible iron deficiency.

#### Questions

1. What dietary advice would you give to increase iron intake? What is the recommended iron RDA (in milligrams) for a 4-year-old vegan male? Why?
2. What other key nutrients are of nutritional concern? What are vegan food sources of each you can recommend to the family?
3. Besides iron, what additional vitamin supplement(s) would you recommend, if any? Please explain why.

## Case Study 4

### Infant Obesity

Sari Edelstein, PhD, RD

Practitioners can use the CDC growth charts to plot the normal weight of an infant. Andrew was born weighing 10 lb 10 oz, which plots to higher than the 95th percentile region on the growth charts. At 2 years of age, he remains higher than the 95th percentile for his sex.

#### Question

1. What would be the strategy for Andrew's feeding plan from here?

## Case Study 5

### School-Aged Child with Nut Allergy

Shideh Mofidi, MS, RD, CSP

A 6-year-old boy has severe food allergies to peanut, dairy, egg, and soy. His mother is trying to find a margarine-like spread that does not contain any of these foods. She has been told by her son's allergist that he should avoid all tree nuts. The mother said that someone told her that almonds would be safe because they are more related to apricots (fruit) than to the tree nut family.

#### Questions

1. How should the mother go about finding an appropriate margarine-like spread for her son?
2. Would almonds be safe for this boy to eat? What professional advice would you give this mother?
3. What other precautions should this mother take to ensure the safety of her son at school and other places away from the home?

## Case Study 6

### Childhood Food Allergy

Georgianna Walker, MS, RD, LRD

Students will need access to a standard pediatric growth chart that shows height and weight percentiles for girls. Charts are available at www.cdc.gov/growthcharts/charts.htm#Set2.

Karen S. presents at the pediatric clinic with her daughter, Marcie, who has a persistent rash that seems to come and go with no observed consistency. Karen reports that the rash first showed up about 6 months ago following a lengthy bout of bronchitis that required hospitalization and "a lot of medicine" before Marcie recovered. She also stated, "It seems to be occurring with greater frequency in the past couple of months." The family history is positive for allergies in that Marcie's father reacts to various pollens, molds, and grasses but typically has respiratory difficulties, rather than skin problems. Her mother is suspicious that some sort of food allergy has developed because the rash seems to reoccur or worsen following meals but is inconsistent. She read about food allergies on the Internet and learned that children are often allergic to peanuts, eggs, and soy, so she has tried to eliminate these foods from Marcie's diet with no change in the status of the rash.

Following a physical exam that showed no other causes of the rash, the physician ordered a RAST and skin prick testing. His notes show that Marcie is 6 years old, weighs 44 lb, and is 45 inches in height. Her medical record indicates that she was at the 75th percentile for height and weight at 4 and 5 years of age. It will take a week for test results to come back. In the interim, the physician refers Marcie and her parents to you for help with managing food allergies. Because the specific source of a food allergy has yet to be identified and Marcie's symptoms seem sporadic you recommend that Marcie's parents keep a food and symptom diary over the next week.

#### Questions

1. What factors in Marcie's history put her at risk for developing a food allergy?
2. Explain the immunological process that results in a food allergy.
3. Assess Marcie's growth relative to her age using a standard pediatric growth chart. Is there evidence that the onset of a food allergy in the past months has affected her growth?
4. Explain why a child with severe food allergies is considered to be at nutritional risk.
5. Explain how the tests ordered by the physician are used to help diagnose a food allergy. What test is considered to be the gold standard for determining a food allergy and why?
6. Explain the purpose of a food and symptom diary in relation to identifying a food allergen. What useful information can be obtained from a food diary?

A week later, the physician calls Karen to report that Marcie's skin testing showed a 4+ reaction to casein (a milk protein) that was confirmed by the results of the RAST and that Marcie will have to stop drinking milk and eating foods that contain milk. Her mother, who has never heard of casein, is quite concerned about how to grocery shop, how to manage food preparation, what to do about lunches at school or with friends, and what to eat in restaurants now that her child has a food allergy.

#### Questions

1. What does a 4+ reaction indicate about the degree of reaction Marcie has experienced? What implications does this have for Marcie's mother as she selects and prepares foods?
2. What foods/food groups must be avoided? Are there any hidden sources of casein that Marcie's mother must know about?
3. What pointers and tips will help Marcie's mother deal with her daughter's food allergy?
4. Because Marcie cannot eat any foods containing casein, will she be at risk for deficiencies of any essential nutrients? Include your recommendations to address any identified nutrient needs.
5. Write a one-day sample menu for Marcie's mother to follow. Be sure to include any recommended supplements.
6. Develop a list of foods that Marcie *can* eat as a help for Karen when shopping for groceries.

#### Extra Credit

Research support groups for parents of children with food allergies to find successful coping tips that have helped these patients and their families.

## Issues to Debate

1. How would you advise a vegetarian family whose 12-year-old wants to begin eating meat?
2. Read the following two articles and discuss whether or not milk and dairy products are necessary for bone health in growing children and adolescents.
   Lanou, A. J., Berkow, S. E., & Barnard, N. D. (2005). Calcium, dairy products, and bone health in children and young adults: A reevaluation of the evidence. *Pediatrics*, *115*, 736–743.
   Greer, F. R. (2005). Bone health: It's more than calcium intake. *Pediatrics*, *115*, 792–794.
3. What suggestions could you make to the adolescent and the adolescent's nonvegetarian parents when the adolescent wants to be a vegetarian who will not eat dairy products, eggs, beans, tofu, or most vegetables?
4. Would it ever be appropriate to tell a vegetarian family that their children have to eat meat?
5. Physical activity and exercise are equally important as healthy eating in the pediatric population. How would you include physical activity as an intervention for children if you have limited training in this area?
6. What power (if any) does the government have to influence factors that affect childhood obesity?
7. Discuss how the media might promote childhood obesity and how it might be used to prevent this problem.
8. What should the healthcare practitioner consider when he or she plans or implements childhood obesity prevention interventions for families in clinic settings or special programs?
9. Discuss how a child with multiple food allergies can be nutritionally threatened.
10. How is a child with food allergies affected in school? What special provisions are necessary and are these really available?

## References


Alaimo, K., Olson, C. M., & Frongillo, Jr., E. A. (2001). Low family income and food insufficiency in relation to overweight in US children. Is there a paradox? *Archives of Pediatric and Adolescent Medicine*, *155*, 1161–1167.

Allen, L. H. (1998). Zinc and micronutrient supplements for children. *American Journal of Clinical Nutrition*, *68*(Suppl.), 495S–498S.

American Academy of Pediatrics, American Public Health Association, & National Resource Center for Health and Safety in Child Care and Early Education. (2011). Preventing childhood obesity in early care and education programs. Retrieved from http://nrckids.org/CFOC3/index.html

American Academy of Pediatrics Committee on Nutrition. (2000). Hypoallergenic infant formulas. *Pediatrics*, *106*, 346–349.

American Academy of Pediatrics Committee on Nutrition & American Academy of Pediatrics Section on Allergy and Immunology. (2008). Effects of early nutritional interventions on the development of atopic disease in infants and children: The role of maternal dietary restriction, breastfeeding, timing of introduction of complementary foods, and hydrolyzed formulas. *Pediatrics*, *121*, 183–191.

American Academy of Pediatrics Section on Breastfeeding. (2012). Breastfeeding and the use of human milk. *Pediatrics*,*129*(3), e827–841.

American Dietetic Association. (2002). Position paper. The role of dietetics professionals in health promotion and disease prevention. *Journal of the American Dietetic Association*, *102*, 1680–1687.

American Dietetic Association. (2010). Position paper: Food insecurity in the United States. *Journal of the American Dietetic Association*, *110*, 1368–1377.

American Dietetic Association, Society for Nutrition Education, & the American School Food Service Association. (2003). Position paper. Nutrition services: An essential component of comprehensive school health programs. *Journal of the American Dietetic Association*, *103*, 505–514.

Andersen, R. E., Crespo, C. J., Bartlett, S. J., Cheskin, L. J., & Pratt, M. (1998). Relationship of physical activity and television watching with body weight and level of fatness among children: Results from the Third National Health and Nutrition Examination Survey. *Journal of the American Medical Association*, *279*, 938–942.

Armstrong, J., & Reilly, J. J. (2002). Breastfeeding and lowering the risk of childhood obesity. Child Health Information Team. *Lancet*, *359*, 2003–2004.

Barr, S. I. (1999). Vegetarianism and menstrual cycle disturbances: Is there an association? *American Journal of Clinical Nutrition*, *70*(Suppl.), 549S–554S.

Barr, S. I., & Chapman, G. E. (2002). Perceptions and practices of self-defined current vegetarian, former vegetarian, and non-vegetarian women. *Journal of the American Dietetic Association*, *102*, 354–360.

Baumann, U., & Brown R. (2006). Review: Non alcoholic fatty liver disease in childhood. *British Journal of Diabetes and Vascular Disease*, *6*, 264–268.

Bernhisel-Broadbent, J. (1999). Diagnosis and management of food hypersensitivity. *Immunology and Allergy Clinics of North America*, *19*, 463–477.

Beyer, K., Ellman-Grunther, L., Jarvinen, K. M., Wood, R. A., Hourihane, J., & Sampson, H. A. (2003). Measurement of peptide-specific IgE as an additional tool in identifying patients with clinical reactivity to peanuts. *Journal of Allergy and Clinical Immunology*, *112*, 202–207.

Beyer, K., Jarvinen, K. M., Bardina, L., Mishoe, M., Turjanmaa, K., Niggemann, B., … Sampson, H. A. (2005). IgE-binding peptides coupled to a commercial matrix as a diagnostic

instrument for persistent cow's milk allergy. *Journal of Allergy and Clinical Immunology*, *116*, 704–705.
Beyer, K., & Teuber, S. S. (2005). Food allergy diagnostics: Scientific and unproven procedures. *Current Opinion in Allergy and Clinical Immunology*, *5*, 261–266.
Birch, L. L., & Fischer, J. O. (1998). Development of eating behaviors among children and adolescents. *Pediatrics*, *101*, 539–547.
Birch, E. E., Garfield, S., Hoffman, D. R., Uauy, R., & Birch, C. G. (2000). A randomized controlled trial of early dietary supply of long-chain polyunsaturated fatty acids and mental development in term infants. *Developmental Medicine and Child Neurology*, *42*, 174–181.
Biro, F.M., Wien, M. (2010). Childhood obesity and adult morbidities. *American Journal of Clinical Nutrition*, *91*(*5*),1499S–1505S.
Black, R. E., Williams, S. M., Jones, I. E., & Goulding, A. (2002). Children who avoid drinking cow milk have low dietary calcium intakes and poor bone health. *American Journal of Clinical Nutrition*, *76*, 675–680.
Bleich, S. N., Herring, B. J., Flagg, D. D., & Gary-Webb, T. L. (2012). Reduction in purchases of sugar-sweetened beverages among low-income, black adolescents after exposure to caloric information. *American Journal of Public Health*, *102*, 329–335,
Bock, S. A. (2000). Evaluation of IgE-mediated food hypersensitivities. *Journal of Pediatric Gastroenterology and Nutrition*, *30*, S20–S27.
Bock, S. A., Sampson, H. A., Atkins, F. M., Zeiger, R. S., Lehrer, S., Sachs, M., … Metcalfe, D. D. (1988). Double-blind, placebo-controlled food challenge (DBPCFC) as an office procedure: A manual. *Journal of Allergy and Clinical Immunology*, *82*, 986–997.
Bogle, V., & Sykes, C. (2011). Psychological interventions in the treatment of childhood obesity: What we know and need to find out. *Journal of Health Psychology*, *16*, 997–1014.
Bogen, D. L., Hanusa, B. H., & Whitaker, R. C. (2004).The effect of breastfeeding with and without formula use on the risk of obesity at 4 years of age. *Obesity Research*, *12*, 1527–1535.
Booth, S. L., Sallis, J. F., Ritenbaugh, C., Hill, J. O., Birch, L. L., Frank, L. D., … Hays, N. P (Working Group II). (2001). Environmental and societal factors affect food choice and physical activity: Rationale, influences, and leverage points. *Nutrition Reviews*, *59*(3, Pt. 2), S21–S39.
Borzekowski, D. L. G., & Robinson, T. N. (2001). The 30-second effect: An experiment revealing the impact of television commercials on food preferences of preschoolers. *Journal of the American Dietetic Association*, *101*, 42–46.
Bowman, S. A., Gortmaker, S. L., Ebbeling, C. B., Pereira, M. A., & Ludwig, D. S. (2004). Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics*, *113*, 112–118.
Britten, P., Marcoe, K., Yamini, S., & Davis, C. (2006). Development of food intake patterns for the MyPyramid Food Guidance System. *Journal of Nutrition Education and Behavior*, *38*, S78–92.
Bruemmer, B., Krieger, J., Saelens, B. E., & Chan, N. (2012). Energy, saturated fat, and sodium were lower in entrées at chain restaurants at 18 months compared with 6 months following the implementation of mandatory menu labeling regulation in King County, Washington. *Journal of the Academy of Nutrition and Dietetics*, *112*, 1169–1176.
Bublin, M., Pfister, M., Radauer, C., Oberhuber, C., Bulley, S., Dewitt, A.M., … Ballmer-Weber, B. K. (2010). Component-resolved diagnosis of kiwifruit allergy with purified natural and recombinant kiwifruit allergens. *Journal of Allergy and Clinical Immunology*, *125*, 687–694.
Burgenson, C. R., Wechsler, H., Brener, N. D., Young, J. C., & Spain, C. G. (2001). Physical education and activity: Results from the School Health Policies and Programs Study 2000. *Journal of School Health*, *71*, 279–293.
Campbell, K. J., Crawford, D. A., & Ball, K. (2006). Family food environment and dietary behaviors likely to promote fatness in 5–6-year-old children. *International Journal of Obesity* (London), *30*,1272–1280.
Carvalho, N. F., Kenney, R. D., Carrington, P. H., & Hall, D. E. (2001). Severe nutritional deficiencies in toddlers resulting from health food milk alternatives. *Pediatrics*, *107*, E46.
Casey, P., Goolsby, S., Berkowitz, C., Frank, D., Cook, J., Cutts, D., … & Meyers, A. (2004). Maternal depression, changing public assistance, food security, and child health status. *Pediatrics*, *113*(2), 298–304.
Centers for Disease Control and Prevention. (2012). About the National Health and Nutrition Examination Survey. Retrieved from www.cdc.gov/nchs/nhanes/about_nhanes.htm
Centers for Disease Control and Prevention. (2003). Neurologic impairment in children associated with maternal dietary deficiency of cobalamin—Georgia, 2001. *Morbidity and Mortality Weekly Report, 52*, 61–64.
Centers for Disease Control and Prevention. (2011). Overweight and obesity: Division of Nutrition, Physical Activity, and Obesity state program highlights. Retrieved from http://www.cdc.gov/nccdphp/dnpao
Chafen, J. J., Newberry, S. J., Riedl, M. A., Bravata, D. M., Maglione, M., Suttorp, M. J., … Shekelle, P. G. (2010). Diagnosing and managing common food allergies: A systematic review. *Journal of the American Medical Association*, *303*, 1848.
Centers for Disease Control and Prevention. (2003). Neurologic impairment in children associated with maternal dietary deficiency of cobalamin—Georgia, 2001. *Morbidity and Mortality Weekly Report*, *52*, 61–64.
Conner, W. E. (2000). Importance of n-3 fatty acids in health and disease. *American Journal of Clinical Nutrition*, *71*, 171S–175S.
Conquer, J. A., & Holub, B. J. (1996). Supplementation with an algae source of docosahexaenoic acid increases (n-3) fatty acid status and alters selected risk factors for heart disease in vegetarian subjects. *Journal of Nutrition*, *126*, 3032–3039.
Craig, W. J., & Mangels, A. R. (2009). Position of the American Dietetic Association: Vegetarian diets. *Journal of the American Dietetic Association*, *109*, 1266–1282.
Crawford, P., Gosliner, W., Anderson, C., Strode, P., Becerra-Jones, Y., Samuels, S., … Ritchie, L. D. (2004). Counseling Latina mothers of preschool children about weight issues: Suggestions for a new framework. *Journal of American Dietetic Association*, *104*, 387–394.
Dagnelie, P. C., van Staveren, W. A., & van den Berg, H. (1991). Vitamin B-12 from algae appears not to be bioavailable. *American Journal of Clinical Nutrition*, *53*, 695–697.

Dagnelie, P. C., van Staveren, W. A., van Klaveren, J. D., & Burema, J. (1988). Do children on macrobiotic diets show catch-up growth? *European Journal of Clinical Nutrition*, *42*, 1007–1016.

Dagnelie, P. C., van Staveren, W. A., Vergote, F. J. V. R. A., Burema, J., Van't Hof, M. A., van Klaveren, J. D., & Hautvast, J. G. (1989). Nutritional status of infants aged 4 to 18 months on macrobiotic diets and matched omnivorous control infants: A population-based mixed-longitudinal study. II. Growth and psychomotor development. *European Journal of Clinical Nutrition*, *43*, 325–338.

Dagnelie, P. C., van Staveren, W. A., Vergote, F. J. R. V. A., Dingjan, P.G., van den Berg, H., & Hautvast, J. G. A. J. (1989). Increased risk of vitamin B-12 and iron deficiency in infants on macrobiotic diets. *American Journal of Clinical Nutrition*, *50*, 818–824.

Dagnelie, P. C., van Staveren, W. A., Verschuren, S. A. J. M., & Hautvast, J. G. A. J. (1989). Nutritional status of infants aged 4 to 18 months on macrobiotic diets and matched omnivorous control infants: A population-based mixed longitudinal study. I. Weaning pattern, energy and nutrient intake. *European Journal of Clinical Nutrition*, *43*, 311–323.

Dagnelie, P. C., Vergote, F. J. R. V. A., van Staveren, W. A., van den Berg, H., Dingjan, P. G., & Hautvast, J. G. A. J. (1990). High prevalence of rickets in infants on macrobiotic diets. *American Journal of Clinical Nutrition*, *51*, 202–208.

Davis, B., & Kris-Etherton, P. (2003). Achieving optimal essential fatty acid status in vegetarians: Current knowledge and practical implications. *American Journal of Clinical Nutrition*, *78*(Suppl.), 640S–646S.

de Boo, H. A., & Harding, J. E. (2006). The developmental origins of adult disease (Barker) hypothesis. *Australian and New Zealand Journal of Obstetrics and Gynaecology*, *46*, 4–14.

Dhonukshe-Rutten, R. A., van Dusseldorp, M., Schneede, J., de Groot, L. C., & van Staveren, W. A. (2005). Low bone mineral density and bone mineral content are associated with low cobalamin status in adolescents. *European Journal of Nutrition*, *44*, 341–347.

Dibba, B., Prentice, A., & Ceesay, M. (2000). Effect of calcium supplementation on bone mineral accretion in Gambian children accustomed to a low-calcium diet. *American Journal of Clinical Nutrition*, *71*, 544–549.

Dietz, W. H. (2001). Breastfeeding may help prevent overweight children. *Journal of the American Medical Association*, *285*, 2506–2507.

Dietz, W. H. (2004). Overweight in childhood and adolescence. *New England Journal of Medicine*, *350*, 855–857.

Diliberti, N., Bordi, P. L., Conklin, M. T., Roe, L. S., & Rolls, B. J. (2004). Increased portion size leads to increased energy intake in a restaurant meal. *Obesity Research*, *12*(3), 562–568.

Donovan, U. M., & Gibson, R. S. (1995). Iron and zinc status of young women aged 14 to 19 years consuming vegetarian and omnivorous diets. *Journal of the American College of Nutrition*, *14*, 463–472.

Donovan, U. M., & Gibson, R. S. (1996). Dietary intakes of adolescent females consuming vegetarian, semi-vegetarian, and omnivorous diets. *Journal of Adolescent Health*, *18*, 292–300.

Dowda, M., Ainsworth, B. E., Addy, C. L., Saunders, R., & Riner, W. (2001). Environmental influences, physical activity, and weight status in 8- to 16-year-olds. *Archives of Pediatric Adolescent Medicine*, *155*, 711–717.

Drewnowski, A., & Specter, S. E. (2004). Poverty and obesity: The role of energy density and energy costs. *American Journal of Clinical Nutrition*, *79*, 6–16.

DuRant, R. H., Baranowski, T., Johnson, M., & Thompson, W. O. (1994). The relationship among television watching, physical activity, and body composition of young children. *Pediatrics*, *4*(1), 449–455.

Eigenmann, P. A., Sicherer, S. H., Borowski, T. A., Cohen, B. A., & Sampson, H. A. (1998). Prevalence of IgE-mediated food allergy among children with atopic dermatitis. *Pediatrics*, *101*, E8.

Ellis, K. J., Abrams, S. A., & Wong, W. W. (1999). Monitoring childhood obesity: Assessment of weight/height$^2$ index. *American Journal of Epidemiology*, *150*, 939–946.

Ellis, R., Kelsay, J. L., Reynolds, R. D., Morris, E. R., Moser, P. B., & Frazier, C. (1987). Phytate:zinc and phytate x calcium:zinc millimolar ratios in self-selected diets of Americans, Asian Indians, and Nepalese. *Journal of the American Dietetic Association*, *87*, 1043–1047.

Erickson, S. J., Robinson, T. N., Haydel, K. F., & Killen, J. D. (2000). Are overweight children unhappy? *Archives of Pediatric and Adolescent Medicine*, *154*, 931–935.

Fischer, J. O., & Birch, L. L. (1999). Restricting access to palatable foods affects children's behavioral response, food selection, and intake. *American Journal of Clinical Nutrition*, *69*, 1264–1272.

Francois, C. A., Connor, S. L., Bolewicz, L. C., & Connor, W. E. (2003). Supplementing lactating women with flaxseed oil does not increase docosahexaenoic acid in their milk. *American Journal of Clinical Nutrition*, *77*, 226–233.

Freedman, D. S., Khan, L. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2001). Relationship of childhood obesity to coronary heart disease risk factors in adulthood: The Bogalusa Heart Study. *Pediatrics*, *108*, 712–718.

Freeman, H. J., Chopra, A., Clandinin, M. T., & Thomson, A. B. (2011). Recent advances in celiac disease. *World Journal of Gastroenterology*, *17*, 2259–2272.

Frumkin, H. (2002). Urban sprawl and public health. *Public Health Reports*, *117*, 201–217.

Furuta, G. T., Liacouras, C. A., Collins, M. H., Gupta, S. K., Justinich, C., Putnam, P. E., … First International Gastrointestinal Eosinophil Research Symposium (FIGERS) Subcommittees. (2007). Eosinophilic esophagitis in children and adults: A systematic review and consensus recommendations for diagnosis and treatment. *Gastroenterology*, *133*, 1342–1363.

Galvez, M. P., Pearl, M., & Yen, I. H. (2010). Childhood obesity and the built environment. *Current Opinion in Pediatrics*, *22*, 202–207.

Geppert, J., Kraft, V., Demmelmair, H., & Koletzko, B. (2005). Docosahexaenoic acid supplementation in vegetarians effectively increases omega-3 index: A randomized trial. *Lipids*, *40*, 807–814.

Gibson, D. (2004). Long-term Food Stamp Program participation is differently related to overweight in young girls and boys. *Journal of Nutrition*, *134*, 372–379.

Gibson, L. Y., Byrne, S. M., Davis, E. A., Blair, E., Jacoby, P., & Zubrick, S. R. (2007). The role of family and maternal factors in childhood obesity. *Medical Journal of Australia*,*186*, 591–595.

Gibson, R. S., Yeudall, F., Drost, N., Mtitmunit, B., & Cullinan, T. (1998). Dietary interventions to prevent zinc deficiency. *American Journal of Clinical Nutrition*, *68*(Suppl.), 484S–487S.

Gortmaker, S. L., Must, A., Sobol, A. M., Peterson, K., Colditz, G. A., & Dietz, W. H. (1996). Television viewing as a cause of increasing obesity among children in the United States, 1986–1990. *Archives of Pediatric and Adolescent Medicine*, *150*, 356–362.

Grummer-Strawn, L. M., Mei, Z., & Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System. (2004). Does breastfeeding protect against pediatric overweight? Analysis of longitudinal data from the Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System. *Pediatrics*, *113*, e81–86.

Haire-Joshu, D., & Nanney, M. S. (2002). Prevention of overweight and obesity in children: Influences on the food environment. *Diabetes Educator*, *28*, 415–423.

Hallberg, L., & Hulthen, L. (2000). Prediction of dietary iron absorption: An algorithm for calculating absorption and bioavailability of dietary iron. *American Journal of Clinical Nutrition*, *71*, 1147–1160.

Hansen, K. S., Ballmer-Weber, B. K., Sastre, J., Lidholm, J., Andersson, K., Oberhofer, H., … Poulsen, L. K. (2009). Component-resolved in vitro diagnosis of hazelnut allergy in Europe. *Journal of Allergy and Clinical Immunology*, *123*, 1134–1141.

Hardy, L. L., Baur, L. A., Garnett, S. P., Crawford, D., Campbell, K. J., Shrewsbury, V. A., … Salmon, J. (2006). Family and home correlates of television viewing in 12–13 year old adolescents: The Nepean Study. *International Journal of Behavioral Nutrition and Physical Activity*, *10*, 24–32.

Harnack, L., Stang, J., & Story, M. (1999). Soft drink consumption among US children and adolescents: Nutritional consequences. *Journal of the American Dietetic Association*, *99*, 436–441.

Harrison, K., & Marske, A. L. (2005). Nutritional content of foods advertised during the television programs children watch most. *American Journal of Public Health*, *95*, 1568–1574.

Hassink, S. (2003). Problems in childhood obesity. *Primary Care Office Practice*, *30*, 357–374.

Heaney, R. P., Dowell, M. S., Rafferty, K., & Bierman, J. (2000). Bioavailability of the calcium in fortified soy imitation milk, with some observations on method. *American Journal of Clinical Nutrition*, *71*, 1166–1169.

Heaney, R. P., Rafferty, K., & Bierman, J. (2005). Not all calcium-fortified beverages are equal. *Nutrition Today*, *40*, 39–45.

Hebbelinck, M., & Clarys, P. (2001). Physical growth and development of vegetarian children and adolescents. In J. Sabate (Ed.), *Vegetarian nutrition* (pp.173–193). Boca Raton, FL: CRC Press.

Hermann, W., & Geisel, J. (2002). Vegetarian lifestyle and monitoring vitamin $B_{12}$ status. *Clinica Chimica Acta; International Journal of Clinical Chemistry*, *26*, 47–59.

Hill, J. O., & Trowbridge, F. L. (1998). Childhood obesity: Future directions and research priorities. *Pediatrics, 101*(3 Pt 2):570–574.

Hill, J. O., Wyatt, H. R., Reed, G. W., & Peters, J. C. (2003). Obesity and the environment: Where do we go from here? *Science*, *299*, 853–855.

Holick, M. F. (2004). Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. *American Journal of Clinical Nutrition*, *80*(6 Suppl.), 1678S–1688S.

Hood, M. Y., Moore, L. L., Sundarajan-Ramamurti, A., Singer, M., Cupples, L. A., & Ellison, R. C. (2000). Parental eating attitudes and the development of obesity in children. The Framingham Children's Study. *International Journal of Obesity Related Metabolic Disorders*, *24*, 1319–1325.

Houghton, L. A., Green, T. J., Donovan, U. M., Gibson, R. S., Stephen, A. M., & O'Connor, D. L. (1997). Association between dietary fiber intake and the folate status of a group of female adolescents. *American Journal of Clinical Nutrition*, *66*, 1414–1421.

Hunt, J. R. (2002). Moving toward a plant-based diet: Are iron and zinc at risk? *Nutrition Review*, *60*, 127–134.

Hunt, J. R., Matthys, L. A., & Johnson, L. K. (1998). Zinc absorption, mineral balance, and blood lipids in women consuming controlled lacto-ovo vegetarian and omnivorous diets for 8 wk. *American Journal of Clinical Nutrition*, *67*, 421–430.

Imataka, G., Mikami, T., Yamanouchi, H., Kano, K., & Eguchi, M. (2004). Vitamin D deficiency rickets due to soybean milk. *Journal of Paediatrics and Child Health*, *40*, 154–155.

Institute of Medicine, Food and Nutrition Board. (1998). *Dietary Reference Intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2002). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2011). *Dietary Reference Intakes for calcium and vitamin D*. Washington, DC: National Academies Press.

Janelle, K. C., & Barr, S. I. (1995). Nutrient intakes and eating behavior scores of vegetarian and nonvegetarian women. *Journal of the American Dietetic Association*, *95*, 180–186, 189.

Johnson, S. L. (2000). Improving preschoolers' self-regulation of energy intake. *Pediatrics*, *106*, 1429–1435.

Johnson, S. L., & Birch, L. L. (1994). Parents' adiposity and children's eating style. *Pediatrics*, *94*, 653–661.

Jones, R. B., Robins, G. G., & Howdle, P. D. (2006). Advances in celiac disease. *Current Opinion in Gastroenterology*, *22*, 117–123.

Jones, S. J., Jahns, L., Laraia, B. A., & Haughton, B. (2003). Lower risk of overweight in school-aged food insures girls who participate in food assistance. *Archives of Pediatric and Adolescent Medicine*, *157*, 780–784.

Jordan, A. B., & Robinson, T. N. (2008). Children, television viewing, and weight status: Summary and recommendations from and expert panel meeting. *Annals of the*

*American Academy of Political and Social Science*, *615*, 119–132.

Justinich, C. J. (2000). Update in gastrointestinal allergic disease. *Current Opinion in Pediatrics*, *12*, 456–459.

Justinich, C. J., Katz, A., Gurbindo, C., Lepage, G., Chad, Z., Bouthillier, L., & Seidman, E. (1996). Elemental diet improves steroid-dependent eosinophilic gastroenteritis and reverses growth failure. *Journal of Pediatric Gastroenterology and Nutrition*, *23*, 81–85.

Kaiser Family Foundation. (2010). *Daily media use among children and teens up dramatically from five years ago*. A Kaiser Family Foundation report. Retrieved from http://kff.org/disparities-policy/press-release/daily-media-use-among-children-and-teens-up-dramatically-from-five-years-ago

Kalkwarf, H. J., Khoury, J. C., & Lanphear, B. P. (2003). Milk intake during childhood and adolescence, adult bone density, and osteoporotic fractures in US women. *American Journal of Clinical Nutrition*, *77*, 257–265.

Katz, Y., Rajuan, N., Goldberg, M.R., Eisenberg, E., Heyman, E., Cohen, A., & Leshno, M. (2010). Early exposure to cow's milk protein is protective against IgE-mediated cow's milk protein allergy. *Journal of Allergy and Clinical Immunology*, *1*,77–82.

Kelly, K. J. (2000). Eosinophilic gastroenteritis. Jour*nal of Pediatric Gastroenterology and Nutrition*, *30*, S28–S35.

Kelly, K. J., Lazenby, A. J., Rowe, P. C., Yarley, J. H., Perman, J. A., & Sampson, H. A. (1995). Eosinophilic esophagitis attributed to gastroesophageal reflux: Improvement with an amino acid-based formula. *Gastroenterology*, *109*, 1503–1512.

Kissinger, D. G., & Sanchez, A. (1987). The association of dietary factors with the age of menarche. *Nutrition Research*, *7*, 471–479.

Koplin, J. J., Osborne, N. J., Wake, M., Martin, P. E., Gurrin, L. C., Robinson, M. N., … Allen, K. J. (2010). Can early introduction of egg prevent egg allergy in infants? A population-based study. *Journal of Allergy and Clinical Immunology*, *4*, 807–813.

Krajcovicova-Kudlackova, M., Simoncic, R., Babinska, K., & Bederova, A. (1995). Levels of lipid peroxidation and antioxidants in vegetarians. *European Journal Epidemiology*, *111*, 207–211.

Krajcovicova-Kudlackova, M., Simoncic, R., Bederova, A., Grancicova, E., & Megalova, T. (1997). Influence of vegetarian and mixed nutrition on selected haematological and biochemical parameters in children. *Die Nahrung*, *41*, 311–314.

Krajcovicova-Kudlackova, M., Simoncic, R., Bederova, A., & Klvanova, J. (1997). Plasma fatty acid profile and alternative nutrition. *Annals of Nutrition Metabolism*, *41*, 365–370.

Kurtzweil, P. (1996). Taking the fat out of food. *FDA Consumer Magazine*, *30*(6), 15–16.

Lake, A. M. (2012). Pediatric obesity: Preventative measures in early childhood. *Nutrition in Clinical Practice*, *36*, 76S–80S.

Larsson, C. L., & Johansson, G. K. (2002). Dietary intake and nutritional status of young vegans and omnivores in Sweden. *American Journal of Clinical Nutrition*, *76*, 100–106.

Larsson, C. L., & Johansson, G. K. (2005). Young Swedish vegans have different sources of nutrients than young omnivores. *Journal of the American Dietetic Association*, *105*, 1438–1441.

Larsson, C. L., Ronnlund, U., Johansson, G., & Dahlgren, L. (2003). Veganism as status passage: The process of becoming a vegan among youths in Sweden. *Appetite*, *41*, 61–67.

Leung, S. S., Lee, R., Sung, S., Luo, H. Y., Kam, C. W., Yuen, M. P., … Lee, S. H. (2001). Growth and nutrition of Chinese vegetarian children in Hong Kong. *Journal of Paediatrics and Child Health*, *37*, 247–253.

Liacouras, C. A. (2003). Eosinophilic esophagitis in children and adults. *Journal of Pediatric Gastroenterology and Nutrition*, *37*, S23–S28.

Liacouras, C. A. (2006). Eosinophilic esophagitis: Treatment in 2005. *Current Opinion in Gastroenterology*, *22*, 147–152.

Liacouras, C. A., Spergel, J. M., Ruchelli, E., Verma, R., Mascarenhas, M., Semeao, E., … Markowitz, J. E. (2005). Eosinophilic esophagitis: A 10-year experience in 381 children. *Clinical Gastroenterology and Hepatology*, *3*, 1198–1206.

Liacouras, C. A., Wenner, W. J., Brown, K., & Ruchelli, E. (1998). Primary eosinophilic esophagitis in children: Successful treatment with oral corticosteroids. *Journal of Pediatric Gastroenterology and Nutrition*, *26*, 380–385.

Licht, D. J., Berry, G. T., Brooks, D. G., & Younkin, D. P. (2001). Reversible subacute combined degeneration of the spinal cord in a 14-year-old due to a strict vegan diet. *Clinical Pediatrics*, *40*, 413–415.

Lin, J., & Sampson, H. A. (2009). The role of immunoglobulin E-binding epitopes in the characterization of food allergy. *Current Opinion in Allergy and Clinical Immunology*, *9*, 357–361.

Lindholm, R. (2011). Combating childhood obesity: A survey of laws affecting the built environments of low-income and minority children. *Reviews on Environmental Health*, *26*, 155–167.

Linn, S., & Novosat, C. L. (2008). Calories for sale: Food marketing to children in the twenty-first century. *Annals of the American Academy of Political and Social Science*, *615*, 133–155.

Louwman, M. W. J., van Dusseldorp, M., van de Vijver, F. J. R., Thomas, C. M., Schneede, J., Ueland, P. M., … van Staveren, W. A. (2000). Signs of impaired cognitive function in adolescents with marginal cobalamin status. *American Journal of Clinical Nutrition*, *72*, 762–769.

Lucendo, A. J., & Arias, A. (2012). Eosinophilic gastroenteritis: An update. *Expert Review of Gastroenterology and Hepatology*, *6*, 591–601.

Ludwig, D. S., Peterson, K. E., & Gortmaker, S. L. (2001). Relation between consumption of sugar-sweetened drinks and childhood obesity: A prospective, observational analysis. *Lancet*, *357*, 505–508.

Malik, V. S., Willett, W. C., & Hu, F. B. (2009). Sugar-sweetened beverages and BMI in children and adolescents: Reanalyses of a meta-analysis. *American Journal of Clinical Nutrition*, *89*, 438–439.

Mangels, A. R., & Messina, V. (2001). Considerations in planning vegan diets. I. Infants. *Journal of the American Dietetic Association*, *101*, 670–677.

Mangels, R., Messina, V., & Messina, M. (2011). *The dietitian's guide to vegetarian diets* (3rd ed.). Burlington, MA: Jones & Bartlett Learning.

Markowitz, J. E., Spergel, J. M., Ruchelli, E., & Liacorous, C. A. (2003). Elemental diet is an effective treatment for eosinophilic esophagitis in children and adolescents. *American Journal of Gastroenterology*, *98*, 777–782.

Martins, Y., Pliner, P., & O'Connor, R. (1999). Restrained eating among vegetarians: Does a vegetarian eating style mask concerns about weight? *Appetite*, *32*, 145–154.

McBean, L. D., & Miller, G. D. (1999). Enhancing the nutrition of America's youth. *Journal of the American College of Nutrition*, *18*, 563–571.

McCann, J. C., & Ames, B. N. (2005). Is docosahexaenoic acid, an n-3 long-chain polyunsaturated fatty acid, required for development of normal brain function? An overview of evidence from cognitive and behavioral tests in humans and animals. *American Journal of Clinical Nutrition*, *82*, 281–295.

McCrory, M. A., Fuss, P. J., Hays, N. P., Vinken, A. G., Greenberg, A. S., & Roberts, S. B. (1999). Overeating in America: Association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. *Obesity Research*, *7*(6), 546–571.

Messina, M., & Messina, V. (2010). The role of soy in vegetarian diets. *Nutrients*, *2*(8), 855–888.

Messina, V., & Mangels, A. R. (2001). Considerations in planning vegan diets: Children. *Journal of the American Dietetic Association*, *101*, 661–669.

Metallinos-Katsaras, E., Sherry, B., & Kallio, J. (2009). Food insecurity is associated with overweight in children younger than 5 years of age. *Journal of the American Dietetic Association*, *109*,1790–1794.

Mofidi, S. (2003). Nutritional management of pediatric food hypersensitivity. *Pediatrics*, *111*, 1645–1653.

Mofidi, S., & Bock, S. A. (Eds.). (2005). *A health professional's guide to food challenges*. Fairfax, VA: Food Allergy and Anaphylaxis Network.

Munoz, K. A., Krebs-Smith, S. M., Ballard-Barbash, R., & Cleveland, L. E. (1997). Food intakes of US children and adolescents compared with recommendations. *Pediatrics*, *100*, 323–329.

Munoz-Furlong, A. (2003). Daily coping strategies for patients and their families. *Pediatrics*, *111*, 1654–1661.

Nathan, I., Hackett, A. F., & Kirby, S. (1996). The dietary intake of a group of vegetarian children aged 7–11 years compared with matched omnivores. *British Journal of Nutrition*, *75*, 533–544.

Nathan, I., Hackett, A. F., & Kirby, S. (1997). A longitudinal study of the growth of matched pairs of vegetarian and omnivorous children, aged 7–11 years, in the north-west of England. *European Journal of Clinical Nutrition*, *51*, 20–25.

National Center for Health Statistics. (1999). Prevalence of overweight and obesity among adults. Retrieved from www.cdc.gov/nchs/products/pubs/pubd/hestats/obese/obse99.htm

National Resource Center for Health and Safety in Child Care and Early Education. (2011). *Caring for our children* (3rd ed.). Washington, DC: American Public Health Association.

Nelson, J., Chiasson, M., & Ford, V. (2004). Childhood Overwieght in a New York City WIC Population. American Journal of Public Health, 94(3), 458-462.

Neumark-Sztainer, D., Hannan, P. J., Story, M., Croll, J., & Perry, C. (2003). Family meal patterns: Associations with sociodemographic characteristics and improved dietary intake among adolescents. *Journal of the American Dietetic Association*, *103*(3), 317–322.

Neumark-Sztainer, D., Story, M., Hannan, P. J., Tharp, T., & Rex, J. (2003). Factors associated with changes in physical activity: A cohort study of inactive adolescent girls. *Archives of Pediatrics and Adolescent Medicine*, *157*, 803–810.

Neumark-Sztainer, D., Story, M., Resnick, M. D., & Blum, R. W. (1997). Adolescent vegetarians: A behavioral profile of a school-based population in Minnesota. *Archives of Pediatric and Adolescent Medicine*, *151*, 833–838.

Nicklas, T. A., Baranowski, T., Cullen, K. W., & Berenson, G. (2001). Eating patterns, dietary quality, and obesity. *Journal of the American College of Nutrition*, *20*, 599–608.

Nicolaou, N., Poorafshar, M., Murray, C., Simpson, A., Winell, H., Kerry, G., … Custovic, A. (2010). Allergy or tolerance in children sensitized to peanut: Prevalence and differentiation using component-resolved diagnostics. *Journal of Allergy and Clinical Immunology*, *125*, 191–197.

Nielsen, S. J., & Popkin, B. M. (2003). Patterns and trends in food portion sizes, 1977–1998. *Journal of the American Medical Association*, *289*(4), 450–453.

Niggemann, B., Reibel, S., & Wahn, U. (2000). The atopy patch test (APT)—a useful tool for the diagnosis of food allergy in children with atopic dermatitis. *Allergy*, *55*, 281–285.

Noel, R. J., Putnam, P. E., Collins, M. H., Assa'ad, A. H., Guajardo, J. R., Jameson, S. C., & Rothenberg, M. E. (2004a). Clinical and immunopathologic effects of swallowed fluticasone for eosinophilic esophagitis. *Clinical Gastroenterology and Hepatology*, *2*, 568–575.

Noel, R. J., Putnam, P. E., & Rothenberg, M. E. (2004b). Eosinophilic esophagitis. *New England Journal of Medicine*, *351*, 940–941.

Nowak-Wegrzyn, A., Sampson, H. A., Wood, R. A., & Sicherer, S. H. (2003). Food protein-induced enterocolitis syndrome caused by solid food proteins. *Pediatrics*, *111*, 829–835.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in US children and adolescents, 2007–2008. *Journal of the American Medical Association*, *303*, 242–249.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). *Prevalence of obesity in the United States, 2009–2010*. NCHS data brief, no 82. Hyattsville, MD: National Center for Health Statistics.

Ogden, C. L., Flegal, K. M., Carroll, M. D., & Johnson, C. L. (2002). Prevalence and trends in overweight among US children and adolescents, 1999–2000. *Journal of the American Medical Association*, *288*, 1728–1732.

O'Keefe, J. H., & Cordain, L. (2004). Cardiovascular disease resulting from a diet and lifestyle at odds with our Paleolithic genome: How to become a 21st century hunter-gatherer. *Mayo Clinical Proceedings*, *79*, 101–108.

Orenstein, S. R., Shalaby, T. M., Di Lorenzo, C., Putnam, P. E., Sigurdsson, L., Mousa, H., & Kocoshis, S. A. (2000). The spectrum of pediatric eosinophilic esophagitis beyond

infancy: A clinical series of 30 children. *American Journal of Gastroenterology*, *95*, 1422–1430.
Ortolani, C., Bruijnzeel-Koomen, C., Bengtsson, U., Bindslev-Jensen, C., Bjorksten, B., Host, A., … Wüthrich, B. (1999). Position paper: Controversial aspects of adverse reactions to food. *Allergy*, *54*, 27–45.
Ortolani, C., Ispano, M., Pastorello, E. A., Ansaloni, R., & Magri, G. C. (1989). Comparison of results of skin prick tests with fresh foods and commercial food extracts and RAST in 100 patients with oral allergy syndrome. *Journal of Allergy and Clinical Immunology*, *83*, 683–690.
Ortolani, C., Ispano, M., Pastorello, E. A., Bigi, A., & Ansaloni, R. (1988). The oral allergy syndrome. *Annals of Allergy*, *61*, 47–52.
Ortolani, C., Pastorello, E., Farioli, L., Ispano, M., Pravettoni, V., Berti, C., … Zanussi, C. (1993). IgE-mediated allergy from vegetable allergens. *Annals of Allergy*, *71*, 470–476.
Paeratakul, S., Ferdinand, D. P., Champagne, C. M., Ryan, D. H., & Bray, G. A. (2003). Fast-food consumption among US adults and children: Dietary and nutrient intake profile. *Journal of the American Dietetic Association*, *103*, 1332–1338.
Perry, C. L., McGuire, M. T., Newmark-Sztainer, D., & Story, M. (2001). Characteristics of vegetarian adolescents in a multiethnic urban population. *Journal of Adolescent Health*, *29*, 406–416.
Perry, C. L., McGuire, M. T., Neumark-Sztainer, D., & Story, M. (2002). Adolescent vegetarians. How well do their dietary patterns meet the Healthy People 2010 objectives? *Archives of Pediatric and Adolescent Medicine*, *156*, 431–437.
Potter, J. W., Saeian, K., Staff, D., Massey, B. T., Komorowski, R. A., Shaker, R., & Hogan, W. J. (2004). Eosinophilic esophagitis in adults: An emerging problem with unique esophageal features. *Gastrointestinal Endoscopy*, *59*, 355–361.
Raman, R. P. (2002). Obesity and health risks. *Journal of the American College of Nutrition*, *21*, 134S–139S
Reilly, J. J., Methven, E., McDowell, Z. C., Hacking, B., Alexander, D., Stewart, L., Kelnar, C. J. (2003). Health consequences of obesity. *Archives of Disease in Childhood*, *88*(9), 748–752.
Roberts, I. F., West, R. J., Ogilvie, D., & Dillon, M. J. (1979). Malnutrition in infants receiving cult diets: A form of child abuse. *British Medical Journal*, *1*, 296–298.
Robinson, T. N., Hammer, L. D., Killen, J. D., Kraemer, H. C., Wilson, D. M., Hayward, C., & Taylor, C. B. (1993). Does television viewing increase obesity and reduce physical activity? Cross-sectional and longitudinal analyses among adolescent girls. *Pediatrics*, *91*, 273–280.
Roehr, C. C., Reibel, S., Zeigert, M., Sommerfeld, C., Wahn, U., & Niggeman, B. (2001). Atopy patch tests, together with determination of specific IgE levels, reduce the need for oral food challenges in children with atopic dermatitis. *Journal of Allergy and Clinical Immunology*, *107*, 548–553.
Rolls, B. J., Roe, L. S., & Meengs, J. S. (2007). The effect of large portion sizes on energy intake is sustained for 11 days. *Obesity* (Silver Spring), *15*(6), 1535–1543.
Roseboom, T., de Rooij, S., & Painter, R. (2006). The Dutch famine and its long-term consequences for adult health. *Early Human Development*, *82*(8), 485–491.
Rosell, M. S., Lloyd-Wright, Z., Appleby, P. N., Sanders, T. A., Allen, N. E., & Key, T. J. (2005). Long-chain n-3 polyunsaturated fatty acids in plasma in British meat-eating, vegetarian, and vegan men. *American Journal of Clinical Nutrition*, *82*, 327–334.
Rothenberg, M. E. (2004). Eosinophilic gastrointestinal disorders. *Journal of Allergy and Clinical Immunology*, *113*, 11–28.
Rothenberg, M. E., Mishra, A., Collins, M. H., & Putnam, P. E. (2001). Pathogenesis and clinical features of eosinophilic esophagitis. *Journal of Allergy and Clinical Immunology*, *108*, 891–894.
Sabaté, J., Linsted, K. D., Harris, R. D., & Johnston, P. K. (1990). Anthropometric parameters of schoolchildren with different life-styles. *American Journal of Diseases of Children*, *144*, 1159–1163.
Sabaté, J., Linsted, K. D., Harris, R. D., & Sanchez, A. (1991). Attained height of lacto-ovo vegetarian children and adolescents. *European Journal of Clinical Nutrition*, *45*, 51–58.
Sabaté, J., Llorca, C., & Sanchez, A. (1992). Lower height of lacto-ovovegetarian girls at preadolescence: An indicator of physical maturation today? *Journal of the American Dietetic Association*, *92*, 1263–1264.
Sampson, H. A. (1999). Food allergy. Part 2: Diagnosis and management. *Journal of Allergy and Clinical Immunology*, *103*, 981–989.
Sampson, H. A. (2001). Utility of food-specific IgE concentrations in predicting symptomatic food allergy. *Journal of Allergy and Clinical Immunology*, *107*, 891–896.
Sampson, H. A. (2003). Food allergy. *Journal of Allergy and Clinical Immunology*, *111*, S540–S547.
Sampson, H. A. (2004a). Food-induced anaphylaxis. *Novartis Foundation Symposium*, *257*, 161–171.
Sampson, H. A. (2004b). Update on food allergy. *Journal of Allergy and Clinical Immunology*, *113*, 805–819.
Sampson, H. A., & Anderson, J. A. (2000). Summary and recommendations: Classification of gastrointestinal manifestations due to immunologic reactions to foods in infants and young children. *Journal of Pediatric Gastroenterology and Nutrition*, *30*, S87–S94.
Sampson, H. A., & Ho, D. G. (1997). Clinical aspects of allergic disease: Relationship between food-specific IgE concentrations and the risk of positive food challenges in children and adolescents. *Journal of Allergy and Clinical Immunology*, *100*, 444–451.
Sampson, H. A., Munoz-Furlong, A., Campbell, R. L., Adkinson, N. F., Jr., Bock, S. A., Branum, A., … Decker, W. W. (2006). Second symposium on the definition and management of anaphylaxis: Summary report—Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. *Annals of Emergency Medicine*, *47*, 373–380.
Sampson, H. A., Sicherer, S. H., & Birnbaum, A. H. (2001). American Gastroenterological Association practice guidelines—AGA technical review of the evaluation of food allergy in gastrointestinal disorders. *Gastroenterology*, *120*, 1026–1040.
Sanders, T. A. B. (1988). Growth and development of British vegan children. *American Journal of Clinical Nutrition*, *48*, 822–825.
Sanders, T. A. B. (1995). Vegetarian diets and children. *Pediatric Clinics of North America*, *42*, 955–965.

Sanders, T. A. B., & Manning, J. (1992). The growth and development of vegan children. *Journal of Human Nutrition and Dietetics*, *5*, 11–21.

Sanders, T. A. B., & Reddy, S. (1992). The influence of a vegetarian diet on the fatty acid composition of human milk and the essential fatty acid status of the infant. *Journal of Pediatrics*, *120*, S71–S77.

Sandstrom, B. (2001). Micronutrient interactions: Effects on absorption and bioavailability. *British Journal of Nutrition*, *85*(Suppl. 2), S181–S185.

SanGiovanni, J. P., Parra-Cabrera, S., Colditz, G. A., Berkey, C. S., & Dwyer, J. T. (2000). Meta-analysis of dietary essential fatty acids and long-chain polyunsaturated fatty acids as they relate to visual resolution acuity in healthy preterm infants. *Pediatrics*, *105*, 1292–1298.

Satter, E. (1987). *How to get kids to eat … but not too much*. Palo Alto, CA: Bull Publishing.

Satter, E. (1999). *Secrets of feeding a healthy family*. Madison, WI: Kelcy Press.

Satter, E. (2000). *Child of mine: Feeding with love and good sense*. Palo Alto, CA: Bull Publishing.

Schwimmer, J. B, Burwinkle, T. M., & Varni, J. W. (2003). Health-related quality of life of severely obese children and adolescents. *Journal of the American Medical Association*, *289*(14), 1813–1819.

Scott, M. (2003). Nutrition for the full-term infant. In N. Nevin-Folino (Ed.), *Pediatric manual of clinical dietetics* (2nd ed., pp. 55–71). Chicago, IL: American Dietetic Association.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F., & Byers, T. (1993). Do obese children become obese adults? A review of literature. *Preventive Medicine*, *65*, 167–177.

Sicherer, S. H. (1999). Food allergy: When and how to perform oral food challenges. *Pediatric Allergy and Immunology*, *10*, 226–234.

Sicherer, S. H. (2000). Food protein-induced enterocolitis syndrome: Clinical perspectives. *Journal of Pediatric Gastroenterology and Nutrition*, *30*, S45–S49.

Sicherer, S. H. (2002). Food allergy. *Lancet*, *360*, 701–710.

Sicherer, S. H., Muñoz-Furlong, A., Godbold, J. H., & Sampson, H. A. (2010). US prevalence of self-reported peanut, tree nut, and sesame allergy: 11-year follow-up. *Journal of Allergy and Clinical Immunology*, *125*, 1322–1326

Sicherer, S. H., Muñoz-Furlong, A., & Sampson, H. A. (2004). Prevalence of seafood allergy in the United States determined by a random telephone survey. *Journal of Allergy and Clinical Immunology*, *114*, 159–165.

Sicherer, S. H., & Sampson, H. A. (2006). Food allergy. *Journal of Allergy and Clinical Immunology*, *117*, S470–S475.

Sicherer, S. H., & Sampson, H. A. (2010). Food allergy. *Journal of Allergy and Clinical Immunology*, *125*, S116–S125.

Sicherer, S. H., & Teuber, S. (2004). Current approach to the diagnosis and management of adverse reactions to foods. *Journal of Allergy and Clinical Immunology*, *114*, 1146–1150.

Siles, R. I., & Hsieh, F. H. (2011). Allergy blood testing: A practical guide for clinicians. *Cleveland Clinic Journal of Medicine*, *78*, 585–592.

Simopoulos, A. P. (1999). Evolutionary aspects of omega-3 fatty acids in the food supply. *Prostaglandins, Leukotrienes, and Essential Fatty Acids*, *60*, 421–429.

Simopoulos, A. P. (2002). The importance of the ratio of omega-6/omega-3 fatty acids. *Biomedical Pharmacotherapy*, *56*, 365–379.

Skelton, J. A., Irby, M. B., Guzman, M. A., & Beech, B. M. (2012). Children's perspective of health and obesity: A focus group study with Hispanic boys. *Infant, Child and Adolescent Nutrition*, *4*, 289–296.

Spergel, J. M., Andrews, T., Brown-Whitehorn, T. F., Beausoleil, J. L., & Liacouras, C. A. (2005). Treatment of eosinophilic esophagitis with specific food elimination diet directed by a combination of skin prick and patch tests. *Annals of Allergy, Asthma and Immunology*, *95*, 336–343.

Sporik, R., Hill, D. J., & Hosking, C. S. (2000). Specificity of allergen skin testing in predicting positive open food challenges to milk, egg, and peanut in children. *Clinical and Experimental Allergy*, *30*, 1540–1546.

Stepaniak, J., & Melina, V. (2003). *Raising vegetarian children*. Chicago, IL: Contemporary Books.

Stahler, C. (2005). How many youth are vegetarian. *Vegetarian Journal*, 4.

Stahler, C. (2010). How many youth in the U.S. are vegetarian? *Vegetarian Journal*, 3.

Strauss, R. S., & Pollack, H. A. (2003). Social marginalization of overweight children. *Archives of Pediatrics and Adolescent Medicine, 157*(8), 746–752.

Steinbeck, K. S. (2001). The importance of physical activity in the prevention of overweight and obesity in childhood: A review and an option. *Obesity Reviews*, *2*, 117–130.

Steinman, H. A. (1996). "Hidden" allergens in food. *Journal of Allergy and Clinical Immunology*, *98*, 241–250.

Sweetman, C., McGowan, L., Croker, H., & Cooke, L. (2011). Characteristics of family mealtimes affecting children's vegetable consumption and liking. *Journal of the American Dietetic Association*, *111*(2), 269–273.

Taylor, R. W., Jones, I. E., Williams, S. M., & Goulding, A. (2002). Body fat percentages measured by dual-energy x-ray absorptiometry corresponding to recently recommended body mass index cutoffs for overweight and obesity in children and adolescents aged 3–18 y. *American Journal of Clinical Nutrition*, *76*, 1416–1421.

Terr, A. I., & Salvaggio, J. E. (1996). Controversial concepts in allergy and clinical immunology. In C. W. Bierman, D. S. Pearlman, G. G. Shapiro, & W. W. Busse (Eds.), *Allergy, asthma, and immunology from infancy to adulthood* (pp. 749–760). Philadelphia, PA: Saunders.

Thane, C. W., & Bates, C. J. (2000). Dietary intakes and nutrient status of vegetarian preschool children from a British national survey. *Journal of Human Nutrition and Dietetics*, *13*, 149–162.

Thane, C. W., Bates, C. J., & Prentice, A. (2003). Risk factors for low iron intake and poor iron status in a national sample of British young people aged 4–18 years. *Public Health Nutrition*, *6*, 485–496.

Truesdell, D. D., & Acosta, P. B. (1985). Feeding the vegan infant and child. *Journal of the American Dietetic Association*, *85*, 837–840.

Uauy, R., Peirano, P., Hoffman, D., Mena, P., Birch, D., & Birch, E. (1996). Role of essential fatty acids in the function of the developing nervous system. *Lipids*, *31*, S167–S176.

U.S. Department of Agriculture & U.S. Department of Health and Human Services. (2010). *Dietary Guidelines for Americans, 2010* (7th ed.). Washington, DC: U.S. Government Printing Office.

U.S. Department of Agriculture & U.S. Department of Health and Human Services. Food Away from Home Continues to Increase as a Share of Household Expenditures. Retrieved from http://usda.mannlib.cornell.edu/usda/nass/general/aer/aer829.pdfhttp

U.S. Department of Health and Human Services. (2010). Healthy People 2020. Retrieved from http://healthypeople.gov/2020/topicsobjectives2020

U.S. Department of Health and Human Services. (2008). *Physical Activity Guidelines for Americans*. Retrieved from www.cdc.gov/physicalactivity/everyone/health/index.html

van den Berg, H., Dagnelie, P. C., & van Staveren, W. A. (1988). Vitamin B12 and seaweed. *Lancet*, *1*, 242–243.

van Dusseldorp, M., Arts, I. C. W., Bergsma, J. S., de Jong, N., Dagnelie, P. C., & van Staveren, W.A. (1996). Catch-up growth in children fed a macrobiotic diet in early childhood. *Journal of Nutrition*, *126*, 2977–2983.

von Berg, A., Koletzko, S., Grubl, A., Filipiak-Pittroff, B., Wichmann, H. E., Bauer, C. P., … German Infant Nutritional Intervention Study Group. (2003). The effect of hydrolyzed cow's milk formula for allergy prevention in the first year of life: The German Infant Nutritional Intervention Study, a randomized double-blind trial. *Journal of Allergy and Clinical Immunology*, *111*, 533–540.

Wang, J., Godbold, J. H., & Sampson, H. A. (2008). Correlation of serum allergy (IgE) tests performed by different assay systems. *Journal of Allergy and Clinical Immunology*, *121*, 1219–1924.

Wang, J., & Sampson, H. A. (2011). Food allergy. *Journal of Clinical Investigation*, *121*, 827–835.

Wang, Y. C., Bleich, S. N., & Gortmaker, S. L. (2008). Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988–2004. *Pediatrics*, *121*(6), e1604–1614.

Wansink, B., & Kim, J. (2005). Bad popcorn in big buckets: Portion size can influence intake as much as taste. *Journal of Nutrition Education and Behavior*, *37*(5), 242–245.

Watanabe, F., Katsura, H., Takenaka, S., Fujita, T., Abe, K., Tamura, Y., … Nakano, Y. (1999). Pseudovitamin B (12) is the predominant cobalamide of an algal health food, Spirulina tablets. *Journal of Agricultural and Food Chemistry*, *47*, 4736–4741.

Weaver, C. M., & Plawecki, K. L. (1994). Dietary calcium: Adequacy of a vegetarian diet. *American Journal of Clinical Nutrition*, *59*(Suppl.), 1238S–1241S.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *New England Journal of Medicine*, *337*(13), 869–873.

Wilson, L. F. (2007). Adolescents' attitudes about obesity and what they want in obesity prevention programs. *Journal of School Nursing*, *23*, 229–238.

Wood, R. A., Segall, N., Ahlstedt, S., & Williams, P. B. (2007). Accuracy of IgE antibody laboratory results. *Annals of Allergy, Asthma, and Immunology*, *99*, 34–41.

World Health Organization & UNICEF. (2009). Baby Friendly Hospital Initiative, revised, updated and expanded for integrated care. Retrieved from www.who.int/nutrition/publications/infantfeeding/bfhi_trainingcourse/en/index.html

Worsley, A., & Skrzypiec, G. (1998). Teenage vegetarianism: Prevalence, social and cognitive contexts. *Appetite*, *30*, 151–170.

Young, V. R., & Pellett, P. L. (1994). Plant proteins in relation to human protein and amino acid nutrition. *American Journal of Clinical Nutrition*, *59*, 1203S–1212S.

Zeiger, R. S. (2003). Food allergen avoidance in the prevention of food allergy in infants and children. *Pediatrics*, *111*, 1662–1671.

Zocca, J. M., Shomaker, L. B., Tanofsky-Kraff, M., Columbo, K. M., Raciti, G. R., Brady, S. M., … Yanovski, J. A. (2011). Links between mothers' and children's disinhibited eating and children's adiposity. *Appetite*, *56*(2), 324–331.

# Special Section on Celiac Disease

*Anne R. Lee, MSEd, RD, and Stefanie A. Giampa, MS, RD, LDN*



## CHAPTER OUTLINE



### Reader Objectives

**After studying this special section and reflecting on the contents, you should be able to**

1. Identify safe foods and grains within the gluten-free dietary pattern.
2. Understand and complete an in-depth nutritional assessment of an individual with celiac disease.
3. Understand and identify the different modes of presentation of celiac disease.
4. Know where to obtain accurate information on celiac disease and the gluten-free dietary pattern.

## Celiac Disease/Gluten Enteropathy

To begin a discussion of celiac disease we must first understand what gluten is. *Gluten* is often used as the generic term for the storage protein in wheat, rye, and barley. It is the specific amino acid sequence in wheat (gliadin), rye (secalin), and barley (hordein) that renders these grains toxic for individuals with celiac disease. The specific amino acid sequence

in these three grains triggers the immune response in individuals with celiac disease. Different grains have unique protein sequences, thus rendering them as either toxic or safe. Oats do not share the same protein sequence and therefore are chemically safe. They have been considered a questionable grain, however, because of frequent cross-contamination from wheat during the harvesting, milling, and processing (Kasarda, 1997, 2000, 2001; Thompson, 2005). Several companies are beginning to grow, process, and mill uncontaminated oats. In some studies, the inclusion of oats was linked to acceptance of the gluten-free diet with no negative effect on the intestinal biopsy. The toxic grains are wheat (and all its derivatives), rye, and barley. See **Table 1** for the fundamentals of a gluten-free diet and **Table 2** for gluten-free starch alternatives.

**TABLE 1** **Fundamentals of the Gluten-Free Diet**

| Safe Grains | Toxic Grains |
|---|---|
| Amaranth | Wheat (includes spelt, kamut, semolina, triticale) |
| Millet | Rye |
| Quinoa | Barley (including malt) |
| Corn sorghum | |
| Teff | |
| Oats | |

**TABLE 2** **Gluten-Free Starch Alternatives (Flours Used as a Substitute for Wheat Flour)**

**Cereal Grains (seeds of cultivated grasses)**
Amaranth, buckwheat, corn (polenta), millet
Quinoa, sorghum, teff
Rice (white, brown, wild, basmati, jasmine)
Montina (Indian rice grass)

**Tubers (swollen underground plant stem)**
Arrowroot, jicama, taro
Potato: white, sweet
Tapioca (cassava, manioc, yucca)

**Legumes (edible seeds from a pod)**
Beans: Including chickpea, lentil, kidney, navy, peas, soybean

**Nuts (edible kernel of a hard shell)**
Almonds, walnuts, chestnuts, hazelnuts, peanuts, cashews

**Seeds**
Sunflower, flax, pumpkin

## Physiologic Description of Celiac Disease

### Presentation

Individuals with celiac disease or gluten enteropathy react to the specific protein sequences found in wheat, rye, and barley. More specifically, when gluten is ingested the digestive process fails and a toxic fragment of gliadin remains. It is this 33-amino-acid molecule that appears to be the cause for the inflammatory response (Shan et al., 2002). This protein sequence enters the intestinal mucosa and cannot be broken down by either digestive or pancreatic enzymes. It then enters the lamina propria, which causes the release of T cells. The presence of T cells in the lamina propria triggers the activation of cytokines, the production of the antibodies, and the inflammation response (Alaedini & Green, 2005). The resulting villous atrophy and inflammation of the mucosa lead to malabsorption.

Celiac disease has been categorized into four main classes, according to the National Institutes of Health Consensus Conference: classical, atypical, silent, and latent (National Institutes of Health Consensus Development Program, 2004). Classical presentation is the malnourished individual with diarrhea, bloated belly, weight loss, abdominal pain, wasting, malabsorption, and failure to thrive (grow). However, not all individuals have symptoms. In clinical practice, Green and colleagues (2001) showed that celiac disease is being diagnosed in many individuals who are seeking treatment for other conditions that are actually secondary to their celiac disease. Often, such individuals, classified as having atypical symptoms, present with extraintestinal manifestations such as dermatitis herpatiformis, anemia, osteoporosis, peripheral neuropathy, or fatigue (Gujral, Freeman & Thomson, 2012; Murray, 1999). Silent celiac disease presents with no symptoms but is often found in individuals who are considered high risk by screening of celiac disease in first-degree relatives. Last, individuals with latent celiac disease are asymptomatic initially but may develop symptoms or the presence of villous atrophy later. See the section titled "Celiac Disease in Pediatrics" later in this special section for further explanation of these categories.

### Prevalence

Historically, it was thought that celiac disease was a rare disease affecting only children. It was

also assumed that children grew out of celiac disease. In Europe, the prevalence was thought to be 1 in 100–250, whereas in the United States it was very rare, at 1 in 1,000. Research indicates that celiac disease can be diagnosed at any age and is as common in the United States as it is in Europe, with a prevalence of 1%, a rate growing higher among women as compared to men (Green & Cellier, 2007; Thomas et al., 2009). It is becoming clear that celiac disease is found not just in those of northern European ancestry but also in places where it was believed to have been previously undiagnosed such as Africa, the Middle East, Asia, South America, New Zealand, and Australia (Catassi & Yachha, 2008; Shan et al., 2002).

### Diagnosis

Typically in the United States and other parts of the world including Sweden and Canada, there is a long lag time between the initial onset of symptoms and diagnosis of celiac disease, averaging 9.7–11 years despite increased awareness of celiac disease worldwide (Green & Jabri, 2006; Norström, Lindholm, Sandström, Nordyke, & Ivarsson, 2011). The delay in diagnosis is not believed to be to the result of patients not seeking health care (Shan et al., 2002), but rather because celiac disease is still thought by the medical community to be a rare disorder and symptoms can often be vague. The diagnostic process generally consists of serologic testing for antibody markers (IgA antihuman tissue transglutaminase or TTG and IgA endomysial antibody immunofluorescence, or EMA) and the presence of a certain genotype called human leukocyte antigen (HLA) DQ2/8; if those are positive, endoscopic biopsy is performed. Endoscopic biopsy is considered the gold standard because of the characteristic histologic features of celiac disease. These biopsies are graded according to a scale developed by Marsh (1992) and later modified by Oberhuber, Granditsch, and Vogelsang (1999), ranging from Marsh I, which indicates only blunting of the villi, to Marsh IV, which indicates total villous atrophy with hyperplasia of the crypts and increased intraepithelial lymphocyte count (Shan et al., 2002). Proposed in 2007 in response to the many limitations inherent to the Marsh-Oberhuber classification system is a new system for grading villous morphologies using only three categories: A, indicating no atrophy; B, further divided into B1 and B2, indicating detectable or nondetectable villi, respectively (Corazza et al., 2007). The Marsh system, however, appears to prevail as the industry standard for comparing intestinal biopsies in the diagnostic process for celiac disease. Notably, because such villous atrophy may not be present in silent or latent presentations of celiac disease, diagnosis for these individuals must often rely on the presence of HLA-DQ2 and/or HLA-DQ8 genes and/or positive serology (Gujral et al., 2012; National Institutes of Health Consensus Development Program, 2004).

## Treatment

Regardless of the severity of the intestinal damage or lack of intestinal symptoms, the only treatment for celiac disease is strict, life-long adherence to a gluten-free diet. Although this management may sound simple, it requires diligence to maintain a gluten-free diet, which has prompted recent studies to focus on modifying gluten from toxic to safe to provide a less restrictive dietary alternate (Donnelly, Ellis, & Ciclitira, 2011). Until such studies can offer sustained effective alternatives to the gluten-free diet, those with celiac disease must avoid the obvious sources of gluten found in wheat, rye, and barley and their derivatives. There are also hidden sources of gluten in many common foods about which those with celiac disease must be mindful. It takes only one-eighth of a teaspoon of flour to cause visible damage to the intestinal mucosa (Semrad, 2004), but with a strict gluten-free diet the intestine will recover. However, the long-term effect of repeated small doses of gluten remains unknown. One case reported the effects of repeated ingestion of a piece of a communion wafer, which caused sustained elevated antibody levels as well as intestinal damage on an otherwise strict gluten-free diet (Biagi et al., 2004).

Noncompliance has serious consequences. Untreated celiac disease is associated with increased mortality rates of 1.9 to 3.8 compared with the normal population (Green, Rostami, & Marsh, 2005). Increased incidence of infertility, osteoporosis, peripheral neuropathy, lymphomas, and cancers of the small bowel and esophagus are associated with untreated celiac disease. Of note, however, is the protective effect of the gluten-free diet. One group of patients followed for 5 years had no increased mortality compared with control subjects (Green et al., 2005).

### Hidden Sources of Gluten

It is important to remember that a gluten-free diet affects the starch and grain portion of a meal plan. The other food groups offer many naturally gluten-free options, which should be emphasized for a nutritionally balanced intake. However, many foods are considered to be questionable. Wheat flour and malt from barley are common ingredients in medications and in foods such as soups, gravies, soy sauce, seasonings, processed or packaged foods, and even cake icings. The list of ingredients must be checked thoroughly; if the ingredient list is not clear, the manufacturer must be contacted before the food can be considered safe to eat.

The Food Allergen Labeling and Consumer Protection Act passed by Congress in 2004 requires food manufacturers to clearly state whether a product contains any of the eight major food allergens, of which wheat is one. The law went into effect in 2006, and new labels now must state whether wheat is an ingredient. However, the law does not cover barley and rye. The U.S. Food and Drug Administration proposed new rules for gluten-free labeling in January 2007. The use of the term *gluten-free* provides both manufacturers and patients a clear definition of a food's safety from all sources of gluten.

### Safe Foods

The traditional cornerstone foods of the gluten-free diet are rice, corn, and potato. Any foods derived from wheat, rye, and barley were historically believed to be toxic. Foods such as vinegars, distilled alcohol, spices, and many ancient or alternative grains are safe. Grains such as quinoa and millet are not related to wheat, rye, or barley, and they do not share the same protein sequence and therefore are safe. These grains provide texture, fiber, iron, B complex vitamins, and many minerals to a gluten-free diet. The usual staples of the gluten-free diet—rice, corn, and potato—do not provide adequate amounts of nutrients (Thompson, 2000). Special dietary products are not required to be fortified or enriched as are their wheat-based counterparts.

## Nutritional Assessment

As understanding of gluten intolerance unfolds, medical nutrition therapy must also continue to evolve. The standard assessments, including weight and laboratory data, are the cornerstone of the nutritional evaluation of an individual with celiac disease. However, for a complete assessment of an individual with celiac disease we need to look beyond the basics. One must be aware of the need for nutritional supplementation, the various modes of presentation, and the social and emotional components of the gluten-free lifestyle to fully assess and educate these individuals.

Nutrition counseling of an individual with celiac disease must be all-inclusive. The nutrition care plan should include an in-depth nutritional assessment that includes a diet history and/or a 3-day intake record, height, weight, BMI, review of medical and family history, review of symptoms, review of laboratory data, and other tests (see **Table 3**). Of equal importance is a review of eating and shopping behaviors, a social and emotional assessment, and an in-depth education component (Lee, 2003).

For the individual with celiac disease, special attention should be placed on laboratory data. With damage to the small intestine, malabsorption is common. Therefore, iron, calcium, electrolytes, and albumin should be closely monitored. Other tests to perform are thyroid function, bone density, and calcium absorption.

The last component of the nutrition care plan is education. This component is probably the most intense in terms of time and depth of information. Although instructing a patient on a gluten-free diet is enmeshed in the social aspects of a gluten-free lifestyle, health practitioners must be mindful of information overload and individual readiness to learn. Instruction on the gluten-free lifestyle should be broken up into at least two sessions. The first session should focus on survival skills, any nutritional deficiencies, shopping for gluten-free products, and social issues. The second session should focus on alternate grains, exercise, and general health maintenance (Lee, 2003). School and social issues such as travel and dining out should be reviewed at each session because these have been reported to be the areas most negatively affected by the gluten-free lifestyle (Green et al., 2001; Lee & Green, 2004; Lee, Ng, Diamond, Ciaccio, & Green, 2012).

## Noncompliance and Complications

Green found that some individuals would "intentionally cheat" on their diet during social situations, dining out, parties, and other functions outside the home. Only 68% of individuals reported following the diet "all the time," and 30% reported

**TABLE 3** Nutrition Care Plan for Celiac Disease or Gluten-Sensitive Enteropathy

| Tools | Assessment | Interventions |
|---|---|---|
| Diet history/food record | Look for adequate | General diet recommendations |
| Physical | • Calories | ✓ 1–2 g protein/kg body weight |
| Test/labs | • Protein | ✓ 35–40 kcal/kg body weight |
| Medical | • Calcium | ✓ Possibly low fiber at first, gradually increasing fiber content |
| Social/emotional assessment | • Iron | ✓ Possibly lactose free |
| Symptoms review | • B complex | ✓ Possibly fat restricted |
| | • Fruits/vegetables | ✓ Use food record and suggest ways to correct nutrient deficits. Include recipe modification and snacks as ways to increase nutrient intake. |
| | • Iron | |
| | Eating and shopping behaviors | ✓ Add fruits and vegetables if intake is inadequate |
| | • Cultural/religious preferences | General recommendation |
| | • Cooking experience | ✓ Calcium supplement |
| | • Willingness/time to cook | ➤ Children 500 mg/day |
| | • Use of prepared vs. whole foods | ➤ Adults 1,000/1,500 mg/day |
| | • Eating at restaurants vs. home | ✓ Multivitamin if needed |
| | • Favorite foods/preferences | ➤ Children standard 1/day |
| | • Height, weight, BMI, growth chart for children | ➤ Adults 18–55 years |
| | • Skin, hair, and nails | ➤ Male standard 1/day |
| | • Bone density/endoscopy results | ➤ Female prenatal with moderate vitamin A content |
| | • Any other tests | ➤ If older than 56 years, senior formula |
| | • Albumin, cholesterol, high-density and low-density lipoproteins | ✓ Iron supplementation if needed |
| | • Hemoglobin, hematocrit, iron, transferrin, TIBC, $B_{12}$ | ✓ Modify diet for any other medical conditions, i.e., diabetes, hypertension |
| | • Na, $K^+$, $Ca^+$ | ✓ Check medications for possible gluten or food–medication interactions |
| | • Skin, hair, and nails | ✓ Referral to social services if needed |
| | • Family history | ✓ Positive reinforcement of health benefits of gluten-free diet |
| | • Associated symptoms or related illnesses. | ✓ Include family in nutrition education |
| | • Medications/supplements | ✓ Trial of lactose-free diet |
| | • Query response to diagnosis and diet | ✓ If lactose free, highlight nondairy sources of calcium and stress adequate protein |
| | • Family support | ✓ Use lactose enzyme tablets or drops when using dairy products |
| | • Literacy level | ✓ Use soluble fiber from fruits, vegetables, and grains to resolve diarrhea/constipation |
| | • Bloating/gas | ✓ Encourage adequate fluid intake |
| | • Diarrhea | ✓ Use 3 small meals and 3 snacks pattern; include protein at each |
| | • Constipation | ✓ If fat tolerance is diminished, digestive or pancreatic enzymes may be beneficial |

following the diet "most of the time" (Green et al., 2001). Although this adherence rate may be viewed as positive among other diet regimes, the consequences of nonadherence for the individual with celiac disease are grave: increased risks of infertility, peripheral neuropathies, bone loss, lymphomas, and cancers of the small bowel and esophagus (Green & Jabri, 2006).

### Label Reading

**Additives to avoid:**

- Wheat starch
- Modified food starch (from wheat)
- Malt, malt flavoring
- Malt vinegar
- Hydrolyzed vegetable or plant protein
- Soy sauce

**Safe additives:**

- Citric acid
- Maltodextrin
- Dextrin
- Vinegar
- Mono- and diglycerides
- Artificial colors and flavors
- Natural colors and flavors
- MSG (monosodium glutamate)
- Caramel color

In a subsequent study it was found that both males and females reported a high rate of compliance (98% of males and females). However, when asked about where they intentionally went off the diet, a surprising number admitted to dietary indiscretion. Males reported intentionally going off the diet at social activities 81% of the time, 82% at restaurants, and 58% with friends. Females reported 88% intentional noncompliance to the diet at social activities, 88% in restaurants, and 67% with friends. The number one reason these individuals were noncompliant in those settings was because the diet was reportedly too restrictive (73%), but other reasons included the diet is uncomfortable in social settings (69%), too difficult (68%), tasteless (45%), and too expensive (33%) (Lee, Ng, Zivin, & Green, 2007).

## Celiac Disease in Pediatrics

The pediatric presentation of celiac disease covers a wide spectrum of symptoms and related illnesses. To define the range of potential presentations in this population, the National Institutes of Health criteria are used as benchmarks (National Institutes of Health Consensus Development Program, 2004). Classic symptoms are usually seen starting between 6 and 24 months of age. These symptoms usually coincide with the introduction of gluten into the diet. Infants and young children typically present with impaired growth, chronic diarrhea, abdominal distention, muscle wasting and hypotonia, poor appetite, and unhappy behavior (Fasano & Catassi, 2005). Within weeks to months of starting to ingest gluten, weight gain velocity decreases, and finally weight loss can be observed. A celiac crisis, characterized by explosive watery diarrhea, marked abdominal distention, dehydration, electrolyte imbalance, hypotension, and lethargy, was more commonly described at the beginning of the century, but now it is rarely observed. This classic form of celiac disease is a common presentation internationally.

- Currently, there is a general trend of delayed onset of symptomatic celiac disease involving older children, usually ages 5 to 7 years. These children tend to experience unusual intestinal complaints such as recurrent abdominal pain, nausea, vomiting, bloating, and constipation. These children could also present with no gastrointestinal complaints but rather with short stature, pubertal delay, iron-deficiency anemia, dental enamel defects, and abnormalities in liver function tests. Dermatitis herpetiformis, a blistering skin manifestation of celiac disease, is rarely seen in the pediatric population (Fasano & Catassi, 2005).
- *Silent presentation* refers to those patients who otherwise appear healthy and asymptomatic except for the typical gluten-sensitive intestinal enteropathy. Significant numbers of silent cases have been reported from population screenings in both the at-risk group (those with family members with celiac disease and those with insulin-dependent diabetes) and the general population (Fasano & Catassi, 2005).
- Individuals with latent celiac disease have positive antibody serologies and/or the genetic markers (HLA DQ2 or DQ8) but a normal or minimally abnormal mucosal architecture at the intestinal biopsy. These individuals are at risk of developing typical celiac disease later in life (Fasano & Catassi, 2005).

A study on feeding practices in the United States found differences in presentation and symptoms related to infant feeding practices. Those children who were exclusively breastfed reported fewer symptoms and presented with symptoms later (D'Amico et al., 2005). Introduction of wheat after 6 months also delayed onset of symptoms. This finding is in sharp contrast to what was discovered in Sweden (Ivarsson et al., 2000). The unusual celiac epidemic in Sweden does highlight the influence of environmental factors. In the 1980s, there was a dramatic threefold increase in the number of diagnosed cases. In reviewing

the many levels of factors that play a role in the development of celiac disease, it appeared that breastfeeding as well as the timing of gluten introduction did have an impact on both the severity and presentation time of symptoms. The epidemic was linked to a decrease in breastfeeding at the same time as a later but larger introduction of gluten-containing foods in the infant's diet.

In the celiac adolescent population an increased association was also found with depression and behavioral disorders. A 31% prevalence of lifetime major depressive disorder was found. It was also suggested that treatment with a gluten-free diet and early diagnosis may decrease the vulnerability to depression (Pynnonen et al., 2004).

### Quality of Life in the Pediatric Population with Celiac Disease

Travel, social occasions, school and college, or sleep-away camp are all areas of special concerns to children with celiac disease. Elementary and middle school years are all about learning to be social and interact in a group. Compliance to a gluten-free diet makes this a very difficult task for children with celiac disease. Not only must their food be gluten free, it also cannot be mixed with the other meals and snacks because of cross-contamination issues. In addition to the hazards of meal and snack time, the classroom itself poses several threats to these children. Many art supplies contain gluten, and many projects requiring glue and other materials are usually performed at the child's desk. This seemingly simple task renders the child's own desk contaminated, and therefore it must be cleaned before the child has his or her snack or lunch at the same table or desk. (See the sidebar for a list of classroom products that may contain wheat.) Also, careful hand washing avoids any potential trace ingestion: Remember, it only takes one-eighth of a teaspoon of flour to cause intestinal damage (Semrad, 2004).

#### Potential Gluten-Containing Classroom Supplies

- Crayons
- Glue
- Stickers
- Paint
- Play dough
- Paper mâché

Another area of concern for teens is leaving the safety of their home and going away to college. The prospect of entering that semiadult world of college is daunting enough on its own. Adolescents with celiac disease must also maneuver the dining services to make sure they can obtain safe, uncontaminated, gluten-free meals.

The pediatric population presents a special concern in the area of quality of life for two reasons. First, childhood and adolescence are often difficult years filled with learning and subsequent vulnerability, even without the added burden of a chronic disease. Second, the lack of comprehensive studies in this age group provides incomplete data on the quality of life and social implications of a rigid lifestyle over an extended period of time. However, several resources specifically target this population group. In addition, several fact sheets and handouts have been developed to help navigate social situations while maintaining a strict gluten-free lifestyle.

## Summary

Celiac disease is a common autoimmune disorder. Individual presentation varies from asymptomatic to the extraintestinal skin manifestation of dermatitis herpetiformis to severe wasting and malnutrition. Regardless of presentation, the only treatment is following a strict gluten-free diet for life. Because the dietary regime requires strict adherence in a wheat-laden world, quality of life is affected. Though the overall quality of life of individuals improves with the gluten-free diet, the social impact of the diet and diagnosis on the quality of life remains an issue. The future of treatment of celiac disease may lay with gluten modification, but continued clinical trials are necessary.

For now, important questions remain in the treatment and care of individuals with celiac disease. Further research is needed in the areas of long-term health risks in the pediatric population, the benefits associated with vitamin and mineral supplementation, and the appropriate levels of nutrient supplementation. The questions of what constitutes an acceptable amount of gluten ingestion as well as the long-term effects of a naturally gluten-free versus a wheat starch–based gluten-free diet need to be evaluated. Probably the most pressing question remains: Do we treat the child with potential or latent celiac disease? Only further research will help answer these queries.

## Case Study

### Celiac Disease

Stefanie Giampa, MS, RD, LD

Patient J (age 12 years) presents to the Pediatric Nutrition, Gastroenterology, and Hepatology clinic for his annual check on his reflux disease. He presents with reflux, weight gain, and constipation. These symptoms have returned after a short hiatus during the summer months. His usual routine at home, school, and during the summer was reviewed. It detailed the usual diet mix of fast foods, school lunch program, and some home-cooked meals. The exception was during the summer months when he visited his grandmother in Jamaica where he consumed a traditional diet.

- Lab Data
  Ht: 5 foot 2 inches
  Wt: 134 lb
  Albumin: 4.4
  WBC: 5.0
  RBC: 4.22
  HGB: 12.3
  HCT: 37.4

#### Questions

1. Should this patient be screened for celiac disease?
2. If so, what tests or clinical pathways would you follow?
3. What were the factors that influenced the change in symptoms during the summer months?

## References


Alaedini, A., & Green, P. (2005). Narrative review: Celiac disease. Understanding a complex autoimmune disorder. *Annals of Internal Medicine*, *142*, 289–298.

Biagi, F., Campanella, J., Martucci, S., Pezzimenti, D., Ciclitira, P., Ellis, H., & Corazza, G. R. (2004). A milligram of gluten a day keeps the mucosal recovery away: A case report. *Nutrition Reviews*, *62*, 360–363.

Catassi, C., & Yachha, S. K. (2008). The global village of celiac disease. In A. Fasano, R. Troncone, & D. Branski (Eds.), *Frontiers in celiac disease* (pp. 23–31). Basel, Switzerland: Karger.

Corazza, G. R., Villanacci, V., Zambelli, C., Milione, M., Luinetti, O., Vindigni, C., . . . Donato, F. (2007). Comparison of the interobserver reproducibility with different histologic criteria used in celiac disease. *Clinical Gastroenterology and Hepatology*, *5*, 838–843.

D'Amico, M., Holmes, J., Stavropoulos, S., Frederick, M., Levy, J., DeFelice, A., . . . Green, P. H. (2005). Presentation of pediatric celiac disease in the United States: Prominent effect of breastfeeding. *Clinical Pediatrics*, *44*, 249–258.

Donnelly, S. C., Ellis, H. J., & Ciclitira, P. J. (2011). Pharmacotherapy and management strategies for coeliac disease. *Expert Opinion on Pharmacotherapy*, *12*, 1731–1744.

Fasano, A., & Catassi, C. (2005). Coeliac disease in children. Best Practice and Research Clinical. *Gastroenterology*, *19*, 467–478.

Green, P. H., & Cellier, C. (2007). Celiac disease. *New England Journal of Medicine*, *357*, 1731–1743.

Green, P. H., & Jabri, B. (2006). Celiac disease. *Annual Review of Medicine*, *57*, 207–221.

Green, P. H., Rostami, K., & Marsh, M. N. (2005). Diagnosis of coeliac disease. Best Practice and Research. *Clinical Gastroenterology*, *19*, 389–400.

Green, P. H. R., Stavropoulos, S., Pangagi, S., Goldstein, S., McMahon, D. J., Absan, H., & Neugut, A. I. (2001). Characteristics of adult celiac disease in the USA: Results of a national survey. *American Journal of Gastroenterology*, *96*, 126–131.

Gujral, N., Freeman, H. J., & Thomson, A. B. (2012). Celiac disease: Prevalence, diagnosis, pathogenesis and treatment. *World Journal of Gastroenterology*, *18*, 6036–6059.

Ivarsson, A., Persson, L. A., Nyström, L., Ascher, H., Cavell, B., Danielsson, L., . . . Hernell, O. (2000). Epidemic of coeliac disease in Swedish children. *Acta Paediatrica*, *89*, 165–171.

Kasarda, D. (1997). Gluten and gliadin: Precipitating factors in celiac disease. In M. Maki, P. Collin, & J. K. Visakorpi (Eds.), *Coeliac disease* (Proceedings of the 7th International Symposium on Coeliac Disease, 1996, Tampere, Finland, pp. 195–212). Tampere, Finland: Coeliac Disease Study Group.

Kasarda, D. (2000). Celiac disease. In *The Cambridge World History of Food* (Vol. 1, pp. 1008–1022). Cambridge, England: Cambridge University Press.

Kasarda, D. (2001). Grains in relation to celiac disease. *Cereal World*, *46*, 209–210.

Lee, A. R. (2003). Nutritional assessment and care of celiac disease. *American Dietetic Association Clinical Connections*, *24*, 1–8.

Lee, A. R., & Green, H. R. (2004). Impact on quality of life in patients with celiac disease in the United States. *Proceedings of the 11th International Symposium*, Belfast, Ireland.

Lee, A. R., Ng, D. L., Diamond, B., Ciaccio, E. J., & Green, P. H. (2012). Living with coeliac disease: Survey results from the U.S.A. *Journal of Human Nutrition and Dietetics*, *25*, 233–238.

Lee, A. R., Ng, D., Zivin, J., & Green, P. (2007). Economic burden of a gluten-free diet. *Journal of Human Nutrition and Dietetics*, *20*, 423–430.

Marsh, M. N. (1992). Gluten, major histocompatibility complex, and the small intestine. A molecular and immunobiologic approach to the spectrum of gluten sensitivity ("celiac sprue"). *Gastroenterology*, *102*, 330–354.

Murray, J. (1999). The widening spectrum of celiac disease. *American Journal of Clinical Nutrition*, *69*, 354–365.

National Institutes of Health Consensus Development Program. (2004). NIH Consensus Development Conference on Celiac Disease: National Institutes of Health consensus development conference statement. Retrieved from http://consensus.nih.gov/2004/2004celiacdisease118html.htm

Norström, F., Lindholm, L., Sandström, O., Nordyke, K., & Ivarsson, A. (2011). Delay to celiac disease diagnosis and its implications for health-related quality of life. *BMC Gastroenterology*, *11*, 118.

Oberhuber, G., Granditsch, G., & Vogelsang, H. (1999). The histopathology of coeliac disease: Time for a standardized report scheme for pathologists. *European Journal of Gastroenterology and Hepatology*, *11*, 1185–1194.

Pynnonen, P., Isometsa, E., Aronen, E., Verkasalo, M., Savilahti, E., & Aalberg, V. (2004). Mental disorders in adolescents with celiac disease. *Psychosomatics*, *45*, 325–335.

Semrad, C. (2004, October 23). *How much gluten is too much?* Paper presented at the Annual Patient Education Day 2004. Celiac Disease Center, Columbia University, New York City.

Shan, L., Molberg, O., Parrot, I., Hausch, F., Filiz, F., Gray, G. M., . . . Khosla, C. (2002). Structural basis for gluten intolerance in celiac sprue. *Science*, *297*, 2275–2279.

Thomas, H. J., Ahmad, T., Rajaguru, C., Barnardo, M., Warren, B. F., & Jewell, D. P. (2009). Contribution of histological, serological, and genetic factors to the clinical heterogeneity of adult-onset coeliac disease. *Scandinavian Journal of Gastroenterology*, *44*, 1076–1083.

Thompson, T. (2000). Folate, iron, and dietary fiber content of the gluten free diet. *Journal of the American Dietetic Association*, *100*, 1389–1393.

Thompson, T. (2005). Contaminated oats and other gluten-free foods in the United States. *Journal of the American Dietetic Association*, *105*, 348–349.

CHAPTER 8

# Special Topics in Preadolescent and Adolescent Nutrition: Dietary Guidelines for Athletes, Pediatric Diabetes, and Disordered Eating

*Pamela S. Hinton, PhD, and*
*Karen Chapman-Novakofski, RD, LDN, PhD*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Determine fuel utilization during exercise.
2. Describe optimal nutrition before, during, and after an endurance competition.
3. Describe the consequences of chronic undernutrition and how they differ between males and females.
4. Recognize the prevalence and incidence for pediatric diabetes in the United States.
5. Understand the pathogenesis of types 1 and 2 diabetes in children.
6. Identify risk factors for types 1 and 2 diabetes in children.
7. Recognize conditions associated with pediatric diabetes.
8. Understand management principles to achieve optimal glycemic goals.
9. Describe the similarities and differences among the three eating disorders defined by the American Psychiatric Association.
10. Know the medical consequences of eating disorders.
11. Describe effective treatment for eating disorders, including recommendations for nutrition interventions.
12. Know the prevalence, etiology, and recommended treatment practices for eating disorders in special populations.
13. Identify childhood eating disorders and explain how they differ from anorexia nervosa and bulimia nervosa.

# *Dietary Guidelines for Athletes*

*Pamela S. Hinton, PhD*

## Energy

Total energy expenditure is the sum of resting energy expenditure, dietary-induced thermogenesis, and physical activity and varies with gender, age, and body size. Typically, physical activity accounts for 10% to 15% of total energy expenditure, but athletes in strenuous training programs or who participate in endurance sports may increase total energy expenditure by two- to threefold as a result of physical activity. For example, cyclists competing in stage races such as the Tour de France may expend up to 8,000 kcal/day (Saris, van Erp-Baart, Brouns, Westerterp, & ten Hoor, 1989). The Institute of Medicine recently published formulas for estimated energy requirement based on gender, age, weight, height, and physical activity (see **Table 8.1**).

Accurately determining energy needs from prediction equations is difficult. Interindividual variation

**TABLE 8.1 Institute of Medicine Equations for Estimating Energy**

**Requirements in Healthy Adults**

**Males**

EER = 662 – 9.53 × age (yr) + PA × [5.91 × weight (kg) + 539.6 × height (m)]

**Females**

EER = 354 – 6.91 × age (yr) + PA × [9.36 × weight (kg) + 726 × height (m)]

PA = 1.25 if you are "active" and 1.48 if you are "very active"

EER, estimate energy requirement; PA, physical activity.

Data from Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, protein and amino acids*. Washington, DC: National Academies Press.

in resting energy expenditure is large, and energy expended during physical activity varies with exercise duration and intensity. Tables of **metabolic equivalent** values for various activities can be used to estimate energy expenditure in physical activity and activities of daily living (Ainsworth et al., 2011).

**metabolic equivalent** The ratio of the work metabolic rate to the resting metabolic rate. One metabolic equivalent is defined as 1 kcal/kg/h or 3.5 mL $O_2$ consumed/kg/min.

Even if we have an estimate of how many calories are needed to be in energy balance, the error associated with evaluating dietary intake is largely caused by over- or underreporting, various methods of food preparation and accuracy of food composition tables. Changes in body weight and/or composition may be the best way for athletes to self-monitor their energy balance over time.

## Carbohydrate

**metabolizable energy** Gross energy in a food minus the energy lost in feces, urine, and combustible gases. The metabolizable energies for protein and carbohydrate are 4 kcal/g, and the metabolizable energy for fat is 9 kcal/g.

Dietary carbohydrates (sugar and starch) have a **metabolizable energy** density of 4 kcal/g and are readily digested and absorbed. During exercise, glucose is the preferred substrate for ATP production. For this reason, and to replenish hepatic and muscle glycogen, athletes should consume 60% to 75% of their energy from carbohydrate. It is recommended that endurance athletes consume from 6 to 10 g carbohydrate per kilogram of body weight (BW) (Rodriguez, DiMarco, & Langley, 2009). This recommendation assumes that an athlete is not over- or underweight. For example, an athlete weighing 70 kg needs between 420 and 720 g carbohydrate/day (which is 1,680 to 2,880 kcal of carbohydrate). To equate grams of carbohydrate to foods, a serving from the grains, breads, and cereals group contains approximately 15 g of carbohydrate. A serving is one slice of bread, 1 oz of cereal, or ½ cup of cooked rice or pasta (U.S. Department of Health and Human Services [U.S. DHHS], 2005). This recommendation applies only to athletes who are regularly depleting their glycogen stores (i.e., 2 to 3 hours of aerobic exercise a day 5 to 7 days a week).

There are several reasons for the emphasis on dietary carbohydrates, especially for athletes. First, the central nervous system and red blood cells have a high glucose requirement. Red blood cells do not have mitochondria, so they cannot oxidize fat; ATP must be produced from glycolysis and anaerobic metabolism of pyruvate to lactate. Second, dietary carbohydrates are needed to replenish liver and muscle glycogen stores. Inadequate carbohydrate intake necessitates gluconeogenesis from dietary or endogenous amino acids because fat cannot be used to make glucose.

Glucose is by far the most abundant dietary monosaccharide; the most prevalent dietary disaccharides are sucrose (glucose + fructose), more commonly referred to as table sugar; lactose (glucose + galactose), which is milk sugar; and maltose (glucose + glucose). The mono- and disaccharides fall into the category of "sugars" and are sometimes referred to as "simple carbohydrates." "Complex carbohydrates" refers to oligosaccharides and polysaccharides. Polysaccharides contain 10,000 to 1,000,000 glucose molecules and comprise the starch in grains, legumes, and some vegetables.

Carbohydrates differ not only in their complexity but also in their effect on blood sugar (Foster-Powell, Holt, & Brand-Miller, 2002). The effect of carbohydrates on blood sugar is important for overall health, fueling muscle during exercise, and replenishing glycogen stores after exercise. The effect of a food on blood glucose concentrations depends on the type and amount of carbohydrate in the food and on how quickly that carbohydrate can be digested and absorbed. Some foods cause a rapid increase in blood glucose concentrations,

whereas others produce a slower and more prolonged rise. The glycemic index (GI) is used to quantify the effect of a food on blood glucose and to make comparisons among foods. The GI is defined as the increase in blood glucose concentration above baseline during the 2 hours after eating a test food relative to the response to glucose for an equivalent amount of carbohydrate. The glycemic response to glucose is set at 100, and the GI for all other foods is less than 100.

**Learning Point** Foods that have rapidly digestible carbohydrate and contain glucose have the highest glycemic indices. For example, the sports drink Gatorade has a GI of 80.

The actual GIs of foods may vary from the published values because they are affected by multiple factors. The physical form of the food alters the GI. Breaking down the food matrix increases the GI. For example, the GI of potatoes is increased by 25% if the potatoes are mashed. And, thin linguini has a GI of 87, but thick linguini is lower, at 68. The ripeness of fruit also has an effect on the GI because as a fruit ripens the starch is converted into sugar. Underripe bananas have a GI of 30 versus 50 for a ripe banana. Food processing and cooking methods also alter the GI. Heating, moisturizing, and pressing foods make the starch easier to digest, increasing the GI. For example, raw carrots have a GI of 20, and peeling and boiling them increases the GI to 40.

The GI of a food assumes that only that food has been eaten. However, most humans do not eat single foods but eat meals and snacks composed of several foods. Protein and fat do not cause an increase in blood glucose; only foods containing carbohydrate have this effect. However, the GI of a food eaten alone is different from the same food eaten as part of a mixed meal of carbohydrate, protein, and fat. For example, a plain French baguette has a GI of 95; bread with butter has a GI of 60. Meals that are high in soluble fiber or that have a high acid content also lower the GI because the rate of gastric emptying is decreased, slowing the increase in blood glucose.

Glycemic load is the net effect of a food on blood glucose concentrations. Glycemic load depends not just on the GI of that food, but on how much of it is eaten. For example, jelly beans have a GI of 80, but eating only five jelly beans causes a much smaller increase in blood glucose than eating a handful of candy-coated chocolate-covered peanuts, which have a GI of 30.

Despite these limitations, the concept of GI still has some practical value for athletes. Even if the GI of a food cannot be accurately determined when it is consumed as part of a meal, foods can be categorized as generally high or low GI. For example, a dinner of whole wheat fettuccine with chunky tomato sauce and sautéed vegetables has a lower GI than does canned spaghetti because of the whole-grain flour, size of the pasta, greater acidity of the tomato sauce, and the fiber in the vegetables.

For overall health, most dietary carbohydrate should come from unrefined whole grains, legumes, and fresh fruits and vegetables (Murphy & Johnson, 2003). Unrefined grains are superior to refined varieties because they provide more fiber and vitamins. Unrefined grains also have lower GIs, which may reduce the risk of non-insulin-dependent (type 2) diabetes. High-GI foods may contribute to insulin resistance because the rapid increase in blood glucose levels after eating these foods stimulates a greater insulin response than do lower-GI foods. Although somewhat controversial, there is evidence that high-GI foods contribute to elevated blood triglycerides (fats), which is a risk factor for heart disease.

## Protein

Dietary protein has a metabolizable energy density of 4 kcal/g. However, oxidation of amino acids for ATP production supplies only a very small fraction of ATP during exercise (< 10% of total energy expenditure). Instead, most dietary protein in meat, dairy products, eggs, and legumes provides the amino acids that are used to repair or build new tissues. Protein needs vary with athlete sex, age, exercise duration, intensity, as well as energy and carbohydrate availability (Rodriguez et al., 2009). Recommended protein intake is slightly increased for athletes (1.2 to 1.7 g/kg BW) compared with nonathletes (0.8 g/kg BW) (Rodriguez et al., 2009; Wolfe, 2000). Endurance athletes should consume 1.2 to 1.4 g protein/kg BW to maintain lean body mass, and strength-trained athletes need 1.2 to 1.7 g/kg to maximize muscle hypertrophy. If energy or carbohydrate intake is insufficient, amino acids from dietary protein or catabolism of endogenous protein is used for gluconeogenesis in the liver. Athletes who follow vegetarian diets have greater

protein requirements (1.3 to 1.8 g/kg BW) because of the lower quality of plant-derived proteins. A 70-kg athlete who participates in a team sport such as basketball or soccer needs 84 to 98 g protein/day.

**Learning Point** To put grams of protein into food terms, a serving from the protein food group is equivalent to approximately 20 g. A serving from this group is 2 to 3 oz cooked meat, 1 cup of cooked beans, 3 tablespoons of peanut butter, or ¼ cup of nuts. Excess dietary protein is metabolized to glucose and fatty acids and stored as glycogen and triglycerides in adipose tissue.

## Fat

Dietary fat has a metabolizable energy density of 9 kcal/g, making it a more concentrated source of energy than carbohydrate or protein is. During moderate-intensity (~70% VO2 max) exercise, fatty acids provide approximately 50% of energy, making dietary fat, as well as triglycerides stored in adipose and skeletal muscle, an important fuel source. In addition, dietary fat plays an essential role in maintenance of optimal health. It is needed for absorption of the fat-soluble vitamins (vitamins A, D, E, and K), and vegetable oils are excellent sources of the antioxidant vitamin E. Fatty acids are incorporated into cell membranes and are required for normal immune function. It is recommended that athletes consume moderate amounts of fat (20% to 35% of energy) and no less than 20% of their total energy from dietary fat, preferably polyunsaturated fats (Rodriguez et al., 2009). As an example, if an athlete needs 3,000 kcal/day, at least 600 of those kilocalories should be from fat. This would be equivalent to approximately 67 g of fat (i.e., 600 calories divided by 9 kcal/g of fat). The easiest way to change the amount of dietary fat is to increase or decrease the amount of fat added to foods, such as butter or cream cheese on bagels, dressing on salads, mayonnaise on sandwiches, and oil in cooking. One tablespoon of butter, cream cheese, dressing, or oil contains about 10 g of fat and 100 calories.

Athletes should make an effort to consume fats from different sources to ensure they consume adequate amounts of the essential fatty acids—alpha-linolenic acid (ALA, an omega-3 fatty acid) and linoleic acid (an omega-6 fatty acid). Omega-6 fatty acids are plentiful in the food supply because they are abundant in vegetable oils. In contrast, omega-3 fatty acids are more likely to be missing from the typical U.S. diet. In addition to ALA, other biologically important omega-3 fatty acids are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Although the essential ALA can be converted into EPA and DHA, this conversion is very limited. Therefore, it is important to consume not only the essential omega-3 ALA but also DHA and EPA. These different omega-3 fatty acids are found in different foods: walnuts, flax seed, soy products, and canola oil are good sources of ALA but are devoid of DHA and EPA that are found in fatty fish (e.g., salmon, mackerel, tuna). Purified ALA, DHA, and EPA also are sold as dietary supplements. Potential health benefits of regular consumption of omega-3 fatty acids include reductions in serum cholesterol and triglycerides and in inflammation of the lining of the blood vessels (Connor, 2000; James, Gibson, & Cleland, 2000).

**Learning Point** There is no direct evidence that omega-3 fatty acids improve athletic performance in humans. In one study of highly trained soccer players, 10 weeks of omega-3 supplementation in the form of fish oil had no effect on maximal aerobic power, anaerobic power, or running performance (Guzmán et al., 2011).

Omega-3 fatty acids alter cell metabolism by activating genes that are needed for cellular transport and use of fat. Omega-3 fatty acids promote the cellular uptake of fat by increasing the activity of lipoprotein lipase (Park & Harris, 2003) and by increasing the amount of fatty acid binding protein, which carries fatty acids across the cell membrane into the cells. Omega-3 fatty acids also facilitate production of energy from fat as a result of increased hepatic carnitine palmitoyltransferase-2 activity and accelerated transport of fatty acids into the mitochondria (Ukropec et al., 2003).

Regardless of whether or not omega-3 fatty acids improve performance, they are needed to optimize health and may reduce the risk of osteoporosis (Watkins, Li, Lippman, & Seifert, 2001), rheumatoid arthritis, asthma, cancer, and heart disease (Connor, 2000). According to the Food and Nutrition Board of the Institute of Medicine (2003), adult males should consume 1.6 g/day and adult females 1.1 g/day. Four ounces of cold-water fish, such as salmon, swordfish, or bluefish, contain about 1.5 g omega-3 fatty acids. One ounce of walnuts or flaxseeds (or 1 tablespoon of the oil) has about 2 g of omega-3 fatty acids. As mentioned previously, it is important to include food sources of both ALA and DHA/EPA.

## Vitamins and Minerals

Athletes often believe their vitamin and mineral needs are significantly higher than those of the typical nonathlete, so they consume micronutrients in excess of the Recommended Dietary Allowances (RDAs). What athletes do not realize is that RDAs are set above the mean requirement for the general population, so there is a safety factor built into them. For example, the RDA for iron for women ages 19 to 50 years is 18 mg/day, whereas the mean requirement is 8 mg/day (Institute of Medicine, Food and Nutrition Board, 2001). Athletes may require more of some nutrients than nonathletes do because of increased nutrient excretion, metabolic waste production, and tissue synthesis and repair. However, the increment is small relative to the safety factor.

**Learning Point** Most athletes meet their nutrient needs if they consume the RDAs, which usually can be achieved by eating a varied and balanced diet. The exceptions are athletes who restrict their food intake and vegetarian and vegan athletes. Iron, zinc, and calcium are the most common inadequate nutrient intakes in these populations.

## Iron

In the general population of adults in the United States, 3% to 5% of women are anemic and 11% to 13% are iron deficient; less than 1% of men are iron deficient (Looker, Dallman, Carroll, Gunter, & Johnson, 1997). The reasons for the relatively high prevalence of iron deficiency in women are increased iron losses in menstrual blood flow and lower dietary iron intake. Endurance athletes, regardless of gender, are more likely to become iron deficient than are nonathletes. A recent study of recreationally competitive runners, cyclists, and triathletes found 36% of women and 6% of men to be iron deficient but not anemic (Sinclair & Hinton, 2005). The increased prevalence of iron deficiency in endurance athletes is because of greater iron losses, primarily via sweat and occult gastrointestinal bleeding.

Recently, exercise-induced inflammation has been implicated as a possible cause of iron deficiency in athletes (Peeling, Dawson, Goodman, Landers, & Trinder, 2008). High-intensity/long-duration exercise increases both the inflammatory marker interleukin-6 (IL-6) and the hepatic protein hepcidin, which blocks export of iron from storage sites in enterocytes, liver, and spleen (Peeling et al., 2009a, 2009b; Roecker, Meier-Buttermilch, Brechtel, Nemeth, & Ganz, 2005). Although these factors are likely to play a role, more data are needed to establish causality of chronic inflammation and hepcidin in the iron deficiency associated with sports participation, particularly endurance sports.

Iron plays a critical role in oxidative metabolism. Hemoglobin and myoglobin bind dioxygen via the porphyrin ring of heme. Hemoglobin carries oxygen from the environment to the tissues; myoglobin transfers oxygen from erythrocytes to muscle cells. The electron transport chain depends on heme-containing cytochromes ($a$, $a_3$, $b$, $b_5$, $c$, $c_1$) and on nonheme iron–sulfur enzymes (NADH dehydrogenase, succinate dehydrogenase, and ubiquinone-cytochrome $c$ reductase).

Iron deficiency is a degenerative condition that progresses through three stages (Institute of Medicine, Food and Nutrition Board, 2001): depleted iron stores but functional iron is unchanged, early functional iron deficiency without anemia, and iron-deficiency anemia. The clinical indicators used to assess each stage of iron deficiency are shown in **Table 8.2**. The clinical signs and symptoms of iron depletion are determined by the severity of the deficiency. Early functional iron deficiency reduces endurance capacity and energetic efficiency during submaximal work in young women (Brownlie, Utermohlen, Hinton, Giordano, & Haas, 2002; Brownlie, Utermohlen, Hinton, & Haas, 2004; Brutsaert et al., 2003; Hinton, Giordano, Brownlie, & Haas, 2000). Based on animal studies, this deficit is caused by decreased activity of iron-containing oxidative enzymes and cytochromes (Davies et al., 1984; Finch et al., 1976; Willis, Brooks, Henderson, & Dallman, 1987). The hallmark symptoms of anemia are fatigue, lack of energy, and apathy. Anemia impairs maximal work performance (maximal oxygen consumption) by reducing oxygen delivery to the body (Celsing, Ekblom, Sylven, Everett, & Astrand, 1988).

Daily basal iron losses are small compared with total body iron (0.9 to 1.2 mg iron/day): 0.6 mg/day are lost from the gastrointestinal tract, mostly in sloughed mucosal cells; 0.08 mg/day are lost in urine; and 0.2 to 0.3 mg/day are lost through the skin. Menstruating women require an additional 0.6 to 0.7 mg/day to account for menstrual blood loss. The estimated average requirement for iron was set to maintain the functional iron

**TABLE 8.2** **Laboratory Measurements Commonly Used in the Evaluation of Iron Status**

| Stage of Iron Deficiency | Indicator | Diagnostic Range |
|---|---|---|
| Depleted stores | Stainable bone marrow iron | Absent |
| | Total iron binding capacity | > 400 mg/dL |
| | Serum ferritin concentration | < 12 mg/L |
| Early functional iron deficiency | Transferrin saturation | < 16% |
| | Free erythrocyte protoporphyrin | > 70 mg/dL |
| | Serum transferrin receptor | > 8.5 mg/L |
| Iron-deficiency anemia | Hemoglobin concentration | < 130 g/L male |
| | | < 120 g/L female |
| | Mean cell volume | < 80 fL |

Data from Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, protein and amino acids.* Washington, DC: National Academies Press.

pool with minimal iron stores (i.e., serum ferritin > 15 µg/L) using factorial modeling of basal and menstrual iron losses and iron accretion (Institute of Medicine, Food and Nutrition Board, 2001).

Increased iron losses and low dietary intake are associated with increased risk of iron deficiency. Blood donation results in a loss of 200 to 250 mg iron/0.5 L of blood. Intense endurance exercise increases whole-body iron turnover and iron losses. The increased loss via the gastrointestinal tract, hematuria, and hemoglobinuria may elevate the estimated average requirement for athletes by 30% to 70% (Institute of Medicine, Food and Nutrition Board, 2001). Because of lower bioavailability of nonheme iron compared with heme iron, vegetarians have an iron requirement that is 1.8 times that of individuals who consume a mixed diet.

To avoid becoming iron deficient, endurance athletes, especially women, should pay attention to how much iron they consume and to the source of iron in their diet. The current daily recommended intake for women ages 18 to 50 years is 18 mg iron/day. This is difficult to achieve through diet alone because the amount of iron in the U.S. food supply is about 6 mg per 1,000 calories. Iron from plant sources is nonheme iron, which is poorly absorbed. For example, a 1-cup serving of raw spinach contains 6 mg of iron, but only 2% to 15%, or 0.1 to 0.6 mg of that, is absorbed in the small intestine. In contrast, animal sources of iron (i.e., meat) contain heme iron that has a higher bioavailability. A 3-oz serving of steak contains 4 mg of iron, up to 50% of which is absorbed in the intestine for an actual intake of 2 mg iron. Thus, women who follow a vegetarian diet must be especially careful to consume enough iron.

Supplemental ferrous iron is available in complexes with sulfate, succinate, citrate, lactate, fumarate, and gluconate. Iron supplements should be taken with ascorbic acid to enhance absorption. Hematocrit and hemoglobin respond to supplemental iron after about 2 weeks; it may take up to 12 months to replete iron stores. Unlike most other minerals, iron cannot be actively excreted from the body, so the potential for toxicity is high. Iron supplements should be used only under medical supervision.

## Calcium

Calcium also is a mineral that is likely to be lacking in the diets of athletes, particularly vegans and women. Studies of the diets of athletes have repeatedly shown that many women do not meet the recommended intake for adolescents of 1,300 mg calcium/day (Institute of Medicine, Food and Nutrition Board, 2010), primarily because they do not consume three to four servings of dairy products. Women who are concerned about their body weight or about cardiovascular disease might avoid dairy products because they believe they are high in fat (Leachman Slawson et al., 2001; Turner & Bass, 2001).

Inadequate calcium intake contributes to loss of bone mineral and/or failure to maximize bone

mineral density during skeletal growth, which increases the risk of osteoporosis. Low dietary calcium and/or vitamin D consumption causes mobilization of calcium from bone to maintain the concentration of calcium in blood, allowing muscle contraction and nerve impulse transmission to continue uninterrupted.

Individuals who are lactose intolerant because they do not have adequate amounts of lactase cannot consume dairy products. Recently, food manufacturers have begun fortifying other foods with calcium, and women can now choose calcium-fortified orange juice, breakfast cereal, and soy milk as alternatives to dairy products.

Calcium also is available in dietary supplements. Calcium derived from oyster shells, coral, bone meal, or dolomite is poorly absorbed because of poor solubility in the intestine. The best types of supplemental calcium are calcium carbonate or calcium citrate; both are available in chewable tablets or soft chews. Some chewable antacid tablets contain calcium carbonate and are cheaper than most other calcium supplements.

**Learning Point** There is no advantage to taking more than 1,200 mg calcium/day (U.S. DHHS, n.d.).

## Vitamin D

Vitamin D deficiency (25(OH)D < 20 ng/mL) and insufficiency (25(OH)D < 32 ng/mL) are very common worldwide. Vitamin D is unique among vitamins in that it can be obtained from the diet or synthesized in the skin by exposure to ultraviolet B radiation in sunlight. Skin production of vitamin D is the predominant source of vitamin D (Halliday et al., 2011).

As early as the 1930s, Russian and German scientists reported that ultraviolet irradiation improved physical performance (Cannell, Hollis, Sorenson, Taft, & Anderson, 2009). Subsequently, vitamin D deficiency was associated with muscle atrophy, particularly of type II (fast-twitch) fibers, and with reduced muscular strength and power. With the identification of the vitamin D receptor (VDR) in skeletal muscle, we now have a much better understanding of the muscular effects of vitamin D, which include both genomic and nongenomic mechanisms. Vitamin D can alter expression of proteins involved in calcium metabolism, which is a key regulator of muscle function. In addition, vitamin D enhances development of muscle cells and increases synthesis of muscle contractile proteins (Girgis, Clifton-Bligh, Hamrick, Holick, & Gunton, 2012). In addition, vitamin D alters expression of insulin-like growth factor-I (IGF-I) and its binding proteins, thereby regulating muscle hypertrophy and regeneration (Hamilton, 2010). The activity of membrane calcium channels in skeletal muscle cells responds very rapidly to vitamin D binding to the VDR, affecting muscle contraction and glucose uptake.

In addition to its effects on skeletal muscle, vitamin D is an important regulator of immune function and inflammation, and vitamin D deficiency has been associated with stress fractures, musculoskeletal pain, and respiratory infections (Cannell et al., 2009). A recent study of collegiate athletes reported a negative association between vitamin D status and frequency of illness (Halliday et al., 2011). Because of the potential for vitamin D status to affect an athlete, athletes should have their vitamin D status monitored. Adequate vitamin D status can be achieved via diet and sun exposure during summer and fall, and supplemental vitamin D may be required during the winter to maintain optimal blood concentrations.

## Fluid and Electrolytes

Dehydration caused by an imbalance between fluid loss and intake is the most common cause of heat-related illness in athletes (Convertino et al., 1996). Athletes may lose water at a rate of 0.5 to 1.5 L/hour and up to 6% to 10% of their body weight. Water is lost from all fluid compartments, resulting in decreased sweating and impaired heat dissipation. The decline in blood volume decreases blood pressure and cardiac output. Heart rate increases 3 to 5 beats/minute for every 1% of body weight lost to compensate for decreased stroke volume. Eventually, skin blood flow also is decreased, further reducing the ability to decrease body temperature. Symptoms of heat-related illness are headache, nausea, dizziness, apathy, confusion, exhaustion, and chills. Performance declines markedly because of decreased muscle perfusion. Paradoxically, gastric emptying is slowed, impairing fluid absorption and restoration of fluid balance. The risk of heat-related illness is increased by exercise in hot and humid environments, diuretics use, and older age.

A disproportionate amount of fluid lost in sweat is from the extracellular fluid compartment, the fluid outside of the cells, including the blood plasma. The average concentration of sodium in sweat is 1,150 mg/L but can vary greatly (230 to 2,300 mg/L) (Convertino et al., 1996; Sawka et al., 2007). Assuming a sweat rate of 1.5 L/hour, an athlete with sweat of average saltiness would lose about 1,700 mg sodium/hour. Excessive sweating, combined with consumption of plain water in copious amounts (e.g., 10 L in 4 hours), results in a sodium deficit, referred to as **dilutional hyponatremia**. The symptoms of hyponatremia are disorientation, confusion, seizure, and coma. This condition is quite rare and most often occurs in marathon and ultramarathon type events lasting longer than 3 hours and in individuals who ingest large volumes of fluid without electrolytes.

**dilutional hyponatremia**
Low blood sodium concentrations; in athletes, typically resulting from sodium losses in sweat and excessive consumption of plain water during exercise.

Because potassium is located in the intracellular fluid, much smaller amounts are lost in sweat compared to sodium losses. The average potassium concentration in sweat is about 350 mg/L, so the quantity lost in sweat is negligible compared with the total amount of potassium in the body (180,000 mg for a typical adult male). The recommended potassium intake during exercise is approximately 80–200 mg/L (Sawka et al., 2007). The Institute of Medicine recommends a daily potassium intake of 4,700 mg, which can easily be achieved by consuming fresh fruits and vegetables (Institute of Medicine, Food and Nutrition Board, 2005).

Like potassium, the amount of magnesium lost in sweat is minimal (about 40 mg/L). This is because most of the magnesium in the body is part of the bone mineral matrix or is in the intracellular fluid of muscle cells. It is not necessary to replace magnesium while exercising, but it is important to consume adequate amounts of magnesium in the diet. The recommended intake is about 300 mg daily for adult females and about 400 mg daily for adult males (Institute of Medicine, Food and Nutrition Board, 1997). Nuts, legumes, whole grains, green leafy vegetables, and chocolate are good food sources of magnesium. Similar recommendations apply to calcium, for which sweat losses (about 80 mg/L) also are small relative to the body pool. Although it is not necessary to replace calcium while exercising, it is important for adolescents to consume at least 1,300 mg calcium per day. There is some evidence that shows that calcium losses in sweat of athletes with high sweat volumes can contribute to loss of bone mineral over time (Barry & Kohrt, 2008).

**Learning Point**

Magnesium supplements should be used with caution. Because magnesium and calcium are chemically similar, magnesium can interfere with intestinal absorption of calcium and with calcium's function in the body. For example, magnesium can block calcium binding to muscle cells and inhibit normal muscle contraction.

### Preexercise Hydration

The first step in preventing dehydration is adequate hydration before exercise or competition. Athletes can ensure euhydration by drinking fluid volumes that produce colorless urine in the 24 hours before competition. Glycerol ingestion induces hyperhydration (Anderson, Cotter, Garnham, Casley, & Febbraio, 2001); however, the performance benefit of overhydration while competing in hot and humid conditions can be attributed to the volume of fluid consumed and not to glycerol, per se (Marino, Kay, & Cannon, 2003). The day of the event, consumption of 16 oz of fluid 2 to 3 hours before the start allows time for excretion of excess water in urine before the competition begins.

### Hydration During Exercise

The goal of hydration strategy is to prevent excessive dehydration, that is, loss of more than 2% of body weight. The longer the exercise duration, the greater the likelihood that a small imbalance between fluid loss and fluid intake will result in dehydration (Sawka et al., 2007). Ideally, athletes should drink 8 to 12 oz of fluid every 15 to 20 minutes during exercise. If a training session or competition exceeds 1 hour, a commercial fluid replacement beverage that contains carbohydrates and sodium is superior to plain water. Exogenous carbohydrate maintains blood glucose concentrations, so glycogenolysis is delayed. Sodium increases the palatability of the beverage and enhances fluid consumption; replacing some of the sodium lost in sweat reduces the risk of hyponatremia. The recommended concentration of sodium in a fluid replacement beverage is 500 to 700 mg/L (Convertino et al., 1996; Sawka et al., 2007). Most sports drinks contain sodium, although the amount varies from 300 to 650 mg/L. An alternative to commercial fluid replacement beverages is easily prepared by adding ¼ to ½ teaspoon of salt to 1 L (32 oz) of water, which is equivalent to about 600 and 1,200 mg sodium/L. Salt (sodium chloride) tablets are available, but 8 oz of fluid

(250 mL) must be consumed with every 200 mg of sodium so that the concentration of sodium in blood does not rise too rapidly. Salt tablets are more effective and better tolerated (they may cause gastrointestinal problems in some people) if they are crushed and mixed with water.

The fluid that is consumed must be emptied from the stomach and absorbed from the intestine to be of any benefit. The rate of gastric emptying can reach 1 L/hour and is maximized when gastric volume is high (> 600 mL), solutions are hypotonic, and the carbohydrate concentration is 4% to 10% (Sawka et al., 2007). The rate of fluid absorption is negatively affected by high-intensity exercise (> 80% maximal oxygen consumption), carbohydrate concentrations that exceed 10%, and dehydration (> 4% BW) (Convertino et al., 1996; Sawka et al., 2007).

### Postexercise Hydration

Rehydration after exercise is important because most athletes do not consume enough fluids during exercise to replenish the fluid lost in sweat and respiration. In general, an athlete should consume 24 oz of fluid for every pound of weight lost during an exercise session (Convertino et al., 1996). Excess fluid consumption offsets "obligatory urine losses" that occur when a large volume of water is consumed within a short period of time and the plasma becomes relatively dilute, stimulating renal water excretion. Obligatory urine losses can be minimized by drinking a beverage that contains sodium (Shirreffs & Maughan, 1998) and by eating foods that are high in sodium after exercise (Sawka et al., 2007), such as pretzels, pickles, pizza, cheese, tomato sauce, soy sauce, and ketchup.

## Nutrition During Exercise

During prolonged exercise, depletion of glycogen stores is associated with the onset of fatigue (Costill et al., 1988; Sherman, Doyle, Lamb, & Strauss, 1993). By consuming carbohydrate (glucose) during exercise, the onset of fatigue may be delayed by providing the body with an alternate glucose supply, which spares muscle glycogen (Couture, Massicotte, Lavoie, Hillaire-Marcel, & Peronnet, 2002). The recommended carbohydrate intake during exercise is 30 to 60 g carbohydrate/hour. Consuming carbohydrate at a rate greater than 70 g/hour may exceed the rate of absorption, resulting in gastrointestinal distress. Drinking 16 to 32 oz of a 4% to 8% carbohydrate commercial fluid replacement beverage every hour meets the recommended intake of 30–60 g per hour. The potential for erosion of dental enamel is partially negated by increasing the pH and calcium concentration of the sports drink (Venables et al., 2005). Solutions that have a carbohydrate concentration greater than 8% slow gastric emptying and those that exceed 10% cause a net efflux of water from the intestine, contributing to dehydration. Energy gels contain about 25 g carbohydrate per packet and can be used if consumed with water to avoid gastrointestinal distress. Note that the recommendation for carbohydrate consumption during exercise maintains blood glucose concentrations but does not replenish energy used during exercise.

### Sports Beverages

Sports beverages or gels contain either simple sugars or short-chain complex carbohydrates that are rapidly digested into monosaccharides. The significant difference among products is what simple sugars they contain or result from enzymatic hydrolysis in the small intestine. Glucose and galactose are absorbed quickly against a concentration gradient, using $Na^+$/glucose cotransporters on the mucosal side of the enterocytes. This is an energy-consuming process because the intracellular concentration of $Na^+$ must be maintained via a serosal $Na^+/K^+$ ATPase. Glucose and galactose are transported out of the enterocyte into the blood via glucose-lactate uptake transporters (GLUT) at the serosal surface. Galactose, however, must be converted into glucose in the liver before it can be used to generate ATP, so a sports beverage with galactose does not increase the energy available to the muscle as quickly as a glucose-only beverage does.

Fructose is absorbed by GLUT-5 transporters, and its rate of absorption is slower than that of glucose and galactose. Fructose is phosphorylated to fructose-1 phosphate in cells. Most fructose is metabolized in the liver to glyceraldehyde and dihydroxyacetone phosphate. These intermediates can proceed through glycolysis to pyruvate and can be used in fatty acid synthesis or in gluconeogenesis. Aldolase B is the rate-limiting enzyme in fructose metabolism and has a much lower affinity for fructose-1 phosphate than for fructose-1,6 bisphosphate. As a result, the aldolase B reaction proceeds very slowly and fructose-1 phosphate tends to accumulate in the liver after ingestion of a large amount of fructose. Other tissues also can metabolize fructose but do so at a rate that is even slower than the rate of hepatic metabolism. In extrahepatic tissues, fructose must be phosphorylated by hexokinase to fructose-6 phosphate. The affinity of hexokinase is much greater for glucose than it is for fructose, so the phosphorylation of fructose proceeds very slowly. Because of the way it is metabolized, fructose is not an optimal carbohydrate source during exercise.

Use of dietary carbohydrate for energy during exercise is limited by the rate of absorption (i.e., how fast the sugar molecule gets across the intestine into the bloodstream). One way to increase the rate of glucose absorption is to add a small amount of fructose (or sucrose) to the sports beverage. This strategy works because glucose and fructose are absorbed by different pathways (Jentjens, Achten, & Jeukendrup, 2004; Jentjens, Moseley, Waring, Harding, & Jeukendrup, 2004; Jentjens, Venables, & Jeukendrup, 2004). However, the fructose content should not exceed 2% to 3% because ingestion of large

amounts of fructose could overwhelm the absorptive capability of the intestine. Fructose that is not absorbed and remains in the gut can cause diarrhea, bloating, and intestinal cramps.

Some athletes drink soda pop as a carbohydrate replacement beverage during endurance events. A 12-oz can of cola provides about 150 calories as sugar. Soda pop is not the optimal choice during exercise. The concentration of carbohydrate in soda pop is about 11%. Once the sugar concentration of the beverage exceeds 8%, gastric emptying is slowed, meaning it takes longer for the sugar to reach the bloodstream. This also has negative implications for rehydration during exercise. Most sodas are sweetened with high-fructose corn syrup that is 55% fructose and 45% glucose by weight. When combined with glucose, small amounts of fructose (2% to 3%, i.e., 2 to 3 g fructose/100 mL) enhance fluid absorption. However, the concentration of fructose in soda pop sweetened with high-fructose corn syrup is about 6.5%. This amount of fructose decreases the rate of fluid and carbohydrate absorption. To make matters worse, the fructose stays in the intestine and may cause gastrointestinal distress. The optimal rehydration solution not only contains glucose to maintain blood sugar but contains sodium as well. A beverage that has 500 to 700 mg of sodium per liter enhances fluid retention, increases voluntary fluid intake as a result of enhanced taste, and prevents low blood sodium levels. The concentration of sodium in cola is much less than this recommendation—about 100 mg of sodium per liter.

Performance benefits during endurance exercise have been demonstrated for consumption of sports beverages containing 4% to 8% carbohydrate. Two recent studies demonstrated an additional benefit when protein was added to the carbohydrate-containing fluid replacement drink (Ivy, Res, Sprague, & Widzer, 2003; Saunders, Kane, & Todd, 2004). Subjects had increased endurance (time to fatigue) and reduced muscle damage with consumption of carbohydrate and protein compared with carbohydrate alone. The drinks were not isoenergetic, so it cannot be determined whether the benefit was the result of additional energy consumption or from protein per se.

## Nutrition After Exercise

After exercise, elevating blood glucose levels quickly is beneficial to replenish glycogen stores, so high-GI foods are recommended. Athletes should aim to consume 1.5 g carbohydrate/kg BW in the first 30 minutes after exercise and again every 2 hours for 4 to 6 hours after exercise (Rodriguez et al., 2009). Insulin secretion is needed for glycogen synthesis. Insulin increases glucose uptake into the muscle via GLUT-4 and stimulates glycogen synthase. Therefore, after exercise the greater insulin secretion associated with high-GI foods is advantageous. For maximal glycogen repletion, it is the GI of a carbohydrate, rather than whether it is simple or complex, that is important. Some complex carbohydrates are also high GI—synthetic sweeteners such as maltodextrin, for example. In contrast, most complex carbohydrates in fruits, vegetables, grains, and legumes are lower on the GI scale.

High-GI foods that are consumed in the proximal postexercise period are not converted into fatty acids and stored in adipose tissue. After exercise, skeletal muscle uses fatty acids, not glucose, for energy so that the glucose is available for glycogen storage (Wolfe, Klein, Carraro, & Weber, 1990). This diversion of glucose toward glycogen synthesis and away from lipogenesis occurs because acetyl-CoA carboxylase and glycerol-3-phosphate acyltransferase are inhibited by AMPK phosphorylase (Hildebrandt, Pilegaard, & Neufer, 2003; Rasmussen, Hancock, & Winder, 1998; Ruderman et al., 2003). At the same time, the rate of fatty acid release from adipose tissue into the blood is increased to supply the muscles. There also is an increase in fatty acid transport into the mitochondria of skeletal muscle for beta-oxidation to produce ATP. The rate of glucose transport into muscle also increases after exercise. The net result is that there is more glucose getting into the muscle where it is used for glycogen synthesis.

The effect of protein on glycogen repletion is equivocal because of differences in study design. Studies that compare isoenergetic postexercise carbohydrate with carbohydrate plus protein usually conclude that there is no advantage to adding protein (Jentjens, van Loon, Mann, Wagenmakers, & Jeukendrup, 2001). Studies that compare treatments that are equivalent in carbohydrate but differ in protein and, therefore, energy content generally find that carbohydrate plus protein is superior to carbohydrate alone (Ivy et al., 2002; Williams, Raven, Fogt, & Ivy, 2003). Other factors affecting the conclusion are the dose and timing of the supplements after exercise. Studies providing large amounts of carbohydrate every 30 minutes during recovery from exercise maximized glycogen repletion compared with studies with less frequent feedings. If glycogen repletion is maximized with carbohydrate alone, it is not possible for protein to enhance the response. Studies that found added protein increased glycogen synthesis after exercise could not attribute the difference to increased insulin levels, so the mechanism of the effect remains unknown (Ivy et al., 2002).

The best dietary strategy to optimize replacement of intramuscular lipid storage (IMTG stores) without compromising glycogen repletion is somewhat controversial. High-carbohydrate diets, regardless of fat intake, seem to interfere with the restoration of intramuscular fat stores to preexercise levels (Johnson et al., 2003; van Loon et al., 2003). One study of highly trained cyclists found that intramuscular fat stores were replenished after

48 hours of a diet providing 39% of energy as fat and 49% as carbohydrate, but an isoenergetic low-fat diet providing 24% of energy as fat and 62% as carbohydrate was ineffective. However, there is a tremendous downside to consuming inadequate carbohydrates after exercise. Glycogen stores are then not repleted, and it is well established that preexercise glycogen levels are strongly associated with time to fatigue during endurance exercise.

**Learning Point** The best practical recommendation is to consume carbohydrates immediately after exercise and then a mixed diet containing carbohydrate, protein, and fat thereafter.

Exercise increases the rates of protein breakdown and synthesis in skeletal muscle, and with adequate nutrition it has an anabolic effect on skeletal muscle (i.e., it results in a net increase in protein synthesis). Carbohydrate consumed after exercise is beneficial because it reduces the rate of protein degradation (Borsheim, Aarsland, & Wolfe, 2004; Borsheim, Cree, et al., 2004). However, to increase protein synthesis and achieve a net increase in muscle mass, it is important to consume protein after exercise. Studies have shown that consuming about 0.2 g of protein/kg BW per hour during the first 2 to 3 hours after exercise results in net protein synthesis (Borsheim, Tipton, Wolf, & Wolfe, 2002; Tipton, Borsheim, Wolf, Sanford, & Wolfe, 2003; Tipton, Ferrando, Phillips, Doyle, & Wolfe, 1999).

## Alcohol, Caffeine, and Soda

### ALCOHOL

Ethanol is metabolized in the liver to acetate and acetaldehyde. ATP can be generated from beta-oxidation of acetate. Acetate (like fatty acids) cannot be used to make glucose, so alcohol cannot be used to replenish glycogen stores. Dietary carbohydrate consumed with ethanol can be used in glycogen synthesis. This was shown in a study examining the effects of alcohol ingestion on glycogen synthesis in muscle of well-trained cyclists, who exercised for 2 hours to deplete their glycogen stores and then consumed one of three test meals (Burke et al., 2003). The three test meals were carbohydrate, providing 7 g carbohydrate and 24 kcal per kilogram of BW; alcohol, providing 1 g carbohydrate, 1.5 g alcohol, and 24 kcal per kilogram of BW; and carbohydrate + alcohol, providing 7 g carbohydrate, 1.5 g alcohol, and 34 kcal per kilogram of BW. The alcohol, equivalent to about 10 drinks, was consumed during the first 3 hours after exercise. Biopsies of the quadriceps muscle were taken 8 and 24 hours after exercise. Although both groups that consumed alcohol had lower blood glucose levels at 8 and 24 hours after exercise than did the carbohydrate group, muscle glycogen was lower only in the alcohol group.

The acute effects of alcohol ingestion on performance are mostly negative and are dose dependent. Psychomotor function is impaired, and small to moderate doses of alcohol (2 to 4 oz of alcohol) slow reaction time; interfere with eye–hand coordination, accuracy, and balance; and make performance of gross motor skills more difficult. Alcohol causes vasodilation of blood vessels and loss of body heat, increasing the risk of hypothermia when exercising in the cold. Alcohol lowers blood glucose and decreases glucose uptake by skeletal muscle, so an athlete experiences earlier onset of fatigue.

### CAFFEINE

Caffeine is a mild stimulant; it interferes with the binding of adenosine, a neurotransmitter with calming effects, to its receptor. Hence, caffeine has stimulatory effects on many systems of the body, and it increases heart rate and blood pressure, water excretion by the kidneys, and secretion of the "stress hormones" adrenaline and cortisol by the adrenal gland. Considerable evidence from scientific studies shows that caffeine improves athletic performance in sprints and in endurance events. Enhanced alertness and reaction time contribute to improvements in sprinting. During endurance events, caffeine improves performance by stimulating the release of fatty acids into the blood as a result of increased activity of hormone-sensitive lipase. This allows increased utilization of fat rather than glucose, so muscle glycogen is not depleted as rapidly and the onset of fatigue is delayed. The effects of caffeine on lipolysis are mediated, at least in part, via the sympathetic nervous system, as evidenced by partial abrogation of the caffeine effect with pharmacologic beta-adrenergic blockade (Van Baak & Saris, 2000). Caffeine also may improve performance during submaximal exercise through other mechanisms. Time to fatigue during performance of submaximal isometric contractions was increased with caffeine (6 mg/kg BW) as a result of maintenance of force output and not because of altered firing rates (Acheson et al., 2004).

Caffeine is metabolized rapidly by the body; peak blood levels occur 30 minutes after oral ingestion, and the half-life of caffeine is 4 hours. For this reason caffeine should be consumed within 1 hour of an athletic event to have an effect on performance. An effective dose is 2 to 9 mg caffeine/kg BW, and no additional benefit is derived from higher doses. The caffeine content of coffee varies significantly; an 8-oz cup may contain 100 to 300 mg. Drinking 1 to 2 cups of coffee should not produce the adverse effects of excess caffeine consumption, such as anxiety, jitteriness, heart arrhythmias, dehydration, and dry mouth.

Athletes may develop a tolerance to caffeine, but the variation in individual response to the drug is large—some people develop tolerance and others remain responsive (Lovallo et al., 2004). The rate at which tolerance to caffeine develops and the magnitude of the effect vary with the physiologic response (Watson, Deary, & Kerr, 2002). For example, 400 to 500 mg of caffeine (2 to 3 cups of regular coffee) per day for 7 days results in complete tolerance to its sleep-disrupting effects. Regular consumption of caffeine, however, does not eliminate the hypertensive response. Caffeine is not considered a "Prohibited Substance" by the World Anti-Doping Agency (World Anti-Doping Agency, 2013).

### SODA

Endurance athletes abstain from carbonated beverages because they believe the carbon dioxide interferes with oxygen use. In reality, the potential negative effect of drinking carbonated beverages has nothing to do with oxygen transport or blood pH but with hydration. Most of the $CO_2$ in blood arises as a waste product of cellular metabolism. This carbon dioxide is dissolved in blood and carried as carbonic acid and bicarbonate to the lungs, where it is expired. When blood concentrations of $CO_2$ are high, the body responds by breathing more deeply and frequently to expire the excess $CO_2$.

The $CO_2$ in carbonated beverages has no effect on the amount of $CO_2$ in blood or the acid–base balance of the blood. This is because the $CO_2$ that is in soda never gets into the circulation. Most of the carbonation is lost before it is even swallowed. The increased temperature of the mouth versus the environment causes a large

amount of the dissolved gas to come out of solution. The physical process of swallowing also reduces the amount of $CO_2$ in solution. Any $CO_2$ that does make it to the stomach is released from the body via the mouth (sometimes audibly) as a "belch." This is because the low pH (acid) in the stomach also makes the $CO_2$ less soluble. Because a carbonated beverage helps create a sensation of fullness in the stomach that is out of proportion to the amount consumed, voluntary fluid intake may be reduced (Lambert, Bleiler, Chang, Johnson, & Gisolfi, 1993).

The main source of dietary phosphoric acid is soda pop. Because of the association between soda consumption and increased risk of bone fractures, the constituents of soda (caffeine, phosphoric acid, citric acid, and fructose) have been scrutinized for their potential to cause calcium loss from bone. Anyone who has ever soaked a bone in vinegar would agree that phosphoric acid probably causes a loss of calcium and a weakening of the bones. However, what is observed in the test tube is not always what happens in the body, which has the ability to maintain constant internal conditions, including acidity of the blood.

Heaney and Rafferty (2001) compared the effects of caffeine, phosphoric acid, and citric acid (consumed in soda) on calcium excretion in the urine. Subjects consumed 20 oz of either caffeinated cola (Coke; containing caffeine and phosphoric acid), noncaffeinated cola (Coke-Free; phosphoric acid only), caffeinated noncola (Mountain Dew; caffeine and citric acid), or noncaffeinated noncola (Sprite; citric acid only). Neither phosphoric acid nor citric acid increased calcium loss. Consumption of the caffeinated sodas caused an increase in calcium excretion, but the effect was small (6 to 14 mg) compared with the recommended daily calcium intake (1,000 mg).

When the kidney's ability to excrete acid is compromised, as in chronic renal failure, and the body is exposed to very high acid loads, appreciable amounts of calcium may be lost from bone. However, in a healthy individual the acid load that results from the phosphoric acid in 20 oz of soda is very small (4.5 to 5.0 mEq), which is much less than the acid load produced during normal metabolism of food (50 to 100 mEq). Consuming seven 12-oz colas per day only produces an acid load of about 20 mEq, which is well within the excretory capacity of the kidney.

## Body Composition

Body weight and composition influence an athlete's performance because body size and relative muscle mass affect strength, speed, and appearance. Body fat increases body weight without increasing strength, thereby reducing the power to body weight ratio. In sports that require moving the body over long distances or against the gravitational force, such as running and cycling, a high power to weight ratio is advantageous. In "appearance" sports such as gymnastics, figure skating, diving, and dance, a lean physique also is desirable. However, being too lean can negatively affect performance and overall physical and psychological health. The physical risks associated with excessive leanness are overtraining syndrome, frequent illness and injury, and loss of bone mass. Unrealistic body weight or composition goals can lead to increased body dissatisfaction, disordered eating, and negative effect.

In setting body weight goals, athletes must consider gender, body composition, genetics, and overall physical and mental health. Five percent to 10% body fat may be appropriate for an elite male athlete, but the healthy range for a female athlete is 12% to 28%. Athletes may be classified as overweight based on standard height and weight tables, when in fact they are lean, simply because they have more muscle mass than the "reference" man or woman. Each person has a set-point weight, which is largely determined by genetics, that the body maintains over time. Muscle mass, like body weight, is highly regulated (Keesey & Corbett, 1984). Attempts to achieve a body weight or muscle mass above or below the set-point range result in metabolic adaptations to maintain the set point (Loucks, 2004).

## Guidelines to Increase Muscle Mass

With adequate energy and protein intake, resistance training causes an increase in muscle mass. Protein turnover in skeletal muscle is increased for up to 48 hours after resistance exercise. Exercise reduces nitrogen losses during fasting by suppressing protein breakdown, although protein balance remains negative. Consuming a mixture of carbohydrates and amino acids before or immediately after resistance training results in net protein synthesis. Mammalian target of rapamycin (mTOR) is a kinase that stimulates protein synthesis via transcription and translation of messenger RNAs coding translation proteins, ribosome biosynthesis, and cell proliferation; mTOR also inhibits autophagy. Growth factors, insulin, and amino acids activate mTOR (Deldicque, Theisen, & Francaux, 2005). Resistance exercise activates mTOR, whereas endurance exercise inhibits mTOR activity as a result of activation of AMPK by AMP (Atherton et al., 2005). This finding has practical applications for athletes who engage in endurance and strength training. Protein synthesis in response to resistance training can be maximized by allowing adequate recovery after endurance exercise in conjunction with dietary carbohydrate and protein.

Similarly, muscle protein synthesis following either resistance training or endurance exercise is enhanced by ingestion of 20 to 25 g of high-quality protein (e.g., egg, milk, meat, soy) immediately after exercise. Milk-based protein, in particular, whey

protein, appears to result in the greatest increase in postexercise muscle protein synthesis. This is likely because of two factors: (1) the high leucine content of whey; and (2) the rapid digestion of whey and appearance of leucine in the blood. Leucine activates the mTOR pathway, thereby increasing initiation of protein translation (Phillips & Van Loon, 2011). From a practical perspective, cow's milk and flavored milk, which has added sugar, are cost-effective alternatives to commercially available "recovery" drinks.

A reasonable rate of weight gain is 0.5 kg/week. This requires an additional 500 kcal/day, in addition to the energy required to maintain current body weight. To increase skeletal muscle mass, additional amino acids are required. Athletes who are involved in strenuous resistance training should consume 1.2 to 1.7 g protein/kg BW. Protein in food can be scored based on how closely the proportion of amino acids it contains matches the amino acid composition of muscle protein, correcting for digestibility of the protein. Proteins that are high quality have the right mix of amino acids and receive a score of 1.00, whereas proteins that are missing an essential amino acid or are poorly digested receive a lower score. Typically, protein from animal sources such as meat (0.9) and egg whites (1.0) is high quality, and protein from plant sources such as beans (0.6) and wheat (0.4) is lower quality. For this reason vegetarians need to combine plant sources of protein so they get all the essential amino acids. Examples of complementary foods are beans and rice, peanut butter and wheat bread, and tofu and rice. Because of the lower protein quality of plant-based foods, vegetarian athletes should consume 1.3 to 1.8 g protein/kg BW; this is higher than the recommendation for nonvegetarians of 1.2 to 1.7 g/kg BW.

### Weight and Fat Loss: Misconceptions and Bad Ideas

#### LOW-INTENSITY EXERCISE IS BETTER FOR BURNING FAT

Energy balance is where the rubber hits the road when it comes to weight loss. Although it is true that a greater proportion of the energy comes from fat oxidation during low-intensity exercise, the total energy used is less (if the duration is constant) during low- versus high-intensity exercise. It follows, then, that the absolute amount of fat oxidized is less during low- versus high-intensity activities. For example, cycling at 10 mph uses 6 kcal/kg/hour and 80% of those kilocalories come from fat. One hour of cycling at 10 mph uses a total of 600 kcal, and 480 of those kilocalories are from fat. Contrast that with cycling at 18 mph, which uses 12 kcal/kg/hour but only 50% of the energy from fat. At the faster speed, the total energy cost is 1,200 kcal, and 600 kcal come from fat.

#### LOW-CARBOHYDRATE DIETS INCREASE WEIGHT LOSS

Low-carbohydrate, high-protein weight loss diets have increased in popularity recently, even among athletes. These diets restrict carbohydrate intake to 30 to 120 g/day. The RDA for carbohydrate was determined based on the carbohydrate requirement of the brain. For healthy adults, the RDA is 130 g of carbohydrate per day (Institute of Medicine, Food and Nutrition Board, 2003).

Skeletal muscle can use fat for energy at rest or during low-intensity exercise. As exercise intensity increases, skeletal muscle relies increasingly on carbohydrate for energy. The primary source of carbohydrate during exercise is what is stored in the muscle as glycogen. Athletes need more carbohydrate than the brain's minimum requirement to replete their muscle and liver glycogen stores. Training and performance are negatively affected with consumption of a low-carbohydrate diet.

These diets are popular because, by design, they promote rapid weight loss. During the first 24 to 48 hours of a low-carbohydrate diet, glycogen stores that remain are used to fuel the brain. When glycogen is broken down, the water that is part of the glycogen is released and excreted in the urine. This water loss can be 3 to 4 pounds. With continued inadequate carbohydrate intake, catabolism of skeletal muscle ensues and the amino acids that make up muscle proteins are used in gluconeogenesis. Muscle is about 73% water. So, catabolism of skeletal muscle results in a significant change in body weight resulting from the loss of body water. The number on the scale may drop rapidly on this diet, but losing water and not body fat will do nothing to enhance performance.

## Weight Cycling

The information on the effects of weight cycling in athletes is limited. There is a positive association between "yo-yo dieting" and heart disease in populations with other risk factors for cardiovascular disease, such as smoking, excessive weight and **obesity**, and lack of physical activity (Petersmarck et al., 1999). Weight cycling in conjunction with *ad libitum* consumption of a high-fat diet increases transcription of lipogenic genes in adipose tissue: fatty acid synthase, acetyl CoA carboxylase, malic enzyme, pyruvate kinase, and lipoprotein lipase (Sea, Fong, Huang, & Chen, 2000).

**obesity** Category of body weight above overweight; for children, a body mass index for age and gender percentile is used.

The body weight changes that occur in athletes who are relatively lean, nonsmoking, and active are very different from the weight fluctuations in people who start out overweight or obese. Athletes may experience in- and out-of-season fluctuations in body weight, but even then most athletes stay within the normal range of weight for height. In the general population, weight cycling is prevalent among people who are overweight and have repeatedly tried and failed to maintain a lower body weight. As a result, it is difficult to separate the effects of weight cycling from the effects of being overweight with regard to a person's overall risk of cardiovascular disease.

Studies that found a relationship between weight cycling and risk factors for heart disease or type 2 diabetes saw that relationship disappear when they factored in the effects of being overweight or obese (Petersmarck et al., 1999). The metabolic benefits of endurance training on risk factors for cardiovascular disease (triglycerides, total cholesterol, low-density and high-density lipoproteins, insulin, and lipoprotein lipase activity) do not persist with detraining (Petibois, Cassaigne, Gin, & Deleris, 2004).

Most of the information that exists on the health effects of weight cycling in athletes comes from wrestlers. During their competitive season, wrestlers restrict their food intake and exercise to "make weight," often losing 5% to 10% of their normal weight. Several studies examined how body composition and metabolic rate change as wrestlers lose and regain weight (McCargar & Crawford, 1992; Melby, Schmidt, & Corrigan, 1990; Steen, Oppliger, & Brownell, 1988). Although the results are equivocal, resting metabolic rate decreases during the weight loss phase. Thyroid hormone levels also decrease during weight loss, suggesting that the brain perceives an energy shortage and sends out signals for the body to conserve energy (Loucks et al., 1992). Once the competitive season is over and wrestlers are no longer in a chronic energy deficit, metabolic rate returns to normal.

## Eating Disorders and the Female Athlete Triad

In an effort to achieve or maintain an unrealistically low body weight or percentage of body fat, some athletes restrict their food intake so severely that endocrine function is disrupted. The hypothalamus senses the energy deficit, and the secretion of gonadotropin-releasing hormone, thyrotropin-releasing hormone, corticotropin-releasing hormone, and growth-hormone-releasing hormone are altered at different thresholds of energy availability (Laughlin & Yen, 1996; Loucks, 2004; Loucks et al., 1992; Loucks & Verdun, 1998). As a result, secretion of luteinizing hormone, follicle-stimulating hormone, thyroid-stimulating hormone, and growth hormone (GH) is abnormal and production of reproductive hormones, thyroid hormone, and insulin-like growth factor-I (IGF-I) is reduced (Laughlin & Yen, 1996; Rickenlund, Thoren, Carlstrom, von Schoultz, & Hirschberg, 2004; Waters, Qualls, Dorin, Veldhuis, & Baumgartner, 2001).

The sex hormones are decreased in both men (testosterone) and women (estrogen and progesterone). Women experience irregular menstrual cycles (**oligomenorrhea**) or absent menstrual cycles (**amenorrhea**); men may notice a decline in sex drive (Rickenlund, Thoren, et al., 2004). Thyroid hormone (triiodothyromine) is decreased in both genders, resulting the signs and symptoms of decreased metabolic rate, such as **bradycardia**, hypotension, slowed respiration rate, and delayed reflexes. GH secretion is increased, but because of the energy-deficient state, the liver and other tissues are resistant to GH. As a consequence, hepatic production of IGF-I is decreased. In addition, the secretion of adrenocorticotropic hormone is increased, and serum cortisol concentrations are elevated.

**oligomenorrhea** Irregular or infrequent menstruation in menarchal females; cycles occur at an interval of 35 days or greater, resulting in four to nine cycles per year.

**amenorrhea** Absence of three consecutive menstrual cycles in a menarchal female.

**bradycardia** Abnormally slow heart rate (< 60 beats/minute) that does not meet the body's metabolic demands.

Each of these hormonal changes has a negative impact on bone mineral content and density. Bone turnover becomes imbalanced (i.e., bone resorption exceeds bone formation) when energy availability is 10 to 20 kcal/kg lean body mass per day (Ihle & Loucks, 2004). Hypoestrogenemia and hypercortisolemia accelerate bone resorption. Bone formation is reduced with low testosterone, thyroid hormone, and IGF-I. Osteopenia, osteoporosis, and fractures are consequences of chronic low energy intake. Measurable loss of bone mineral density is evident after missing only six consecutive menstrual cycles. Loss of bone mass is insidious because it is irreversible and often proceeds undetected for long periods of time (Keen & Drinkwater, 1997). In women, the constellation of disordered eating, amenorrhea, and osteopenia/osteoporosis has been labeled the Female Athlete Triad (Loucks, 2004).

There are other negative consequences of chronic low energy availability. Protein turnover is slowed, making athletes more susceptible to injury, illness, and overtraining syndrome. In response to inadequate energy intake from the diet, the body has to rely on other energy sources. The body uses energy that is stored as body fat and breaks down muscle, converting the protein into glucose. Proteolysis of skeletal muscle results in a loss of strength and power, with negative effects on performance.

## Childhood Through Adolescence

To support growth children and adolescents have higher energy and protein needs per kilogram of body weight than do adults. Growth is relatively constant at a rate of approximately 5 to 6 cm and 2.5 kg per year from age 4 years until puberty. At puberty, androgens, estrogens, GH, and IGF-I cause increases in bone mineral, muscle mass, and sex-specific deposition of body fat (Rogol, 2000). The RDA for protein is 0.95 g/kg BW for children ages 9 to 13 years and 0.85 g/kg BW for teenagers, compared with 0.8 g/kg BW for adults (Institute of Medicine, Food and Nutrition Board, 2003). More than 90% of peak bone mass is acquired by age 18 years, and bone mass doubles between the onset of puberty and young adulthood. Thus, children and adolescents require more calcium than adults do; the adequate intake for calcium is 1,300 mg/day for ages 9 to 18 years (Institute of Medicine, Food and Nutrition Board, 2010). Iron and zinc needs also are increased during adolescence to support an increase in skeletal muscle mass and an expansion in red blood cell number (Institute of Medicine, Food and Nutrition Board, 2001).

### Special Focus on Children and Adolescents

Weight-bearing physical activity has a greater effect on bone mass in children and adolescents compared with adults. The effects of exercise on growth and development are closely related to the energy drain created by physical activity. Growth and sexual maturation can be delayed by intense physical training before puberty. Weight and height growth velocities can be decreased in elite female athletes and menarche delayed 1 to 2 years compared with a population reference menarchal age of 13.0 years. Delayed menarche and hypoestrogenism compromise acquisition of bone mass and bone mineral. Exercise duration and training intensity were negatively associated with bone mass density (BMD) in elite female adolescent gymnasts, and the younger the athlete started training, the greater the effect on bone (Markou et al., 2004). A cross-sectional study of 5,461 girls aged 11 to 17 years found that girls who participated in at least 16 hours of exercise per week were at greater risk for stress fractures than those who participated in less than 4 hours per week. Each hour per week spent running, performing gymnastics, or cheerleading also increased the risk of stress fracture (Loud, Gordon, Micheli, & Field, 2005). Some catch-up growth may be possible if an athlete's training load is reduced. Because there are few longitudinal growth studies that follow athletes before the onset of training, it is difficult to determine the effects of slowed growth and delayed maturation on adult body size.

Normal growth and development in males are less affected by training than in females. This may be, in large part, because of selection bias. Physical maturity is an asset in most male sports where strength and power confer a competitive advantage. Wrestlers are unique because of the ubiquitous practice of losing weight through dietary restriction and excessive exercise to compete in lower competitive weight classes. Growth velocity is slowed during the season and catch-up growth occurs during the off season. It is not clear if the short stature of wrestlers is the result of growth restriction, selection bias, or a combination (Rogol, Clark, & Roemmich, 2000).

Children and adolescents also are more susceptible to heat-related illnesses because of a greater impairment of thermal regulation with dehydration (Bar-Or, 2001). Until puberty, children and adolescents sweat less than adults do. In addition, children have a greater surface area to body weight ratio than do adults and therefore receive greater exposure to solar radiation. Similar to adult athletes, children and adolescents do not consume adequate fluids during exercise to remain euhydrated. Provision of a flavored carbohydrate–electrolyte drink increases fluid consumption and reduces voluntary dehydration in children (Rivera-Brown, Gutierrez, Gutierrez, Frontera, & Bar-Or, 1999).

Oral contraceptive agents (OCAs) have benefits for female athletes: timing of the menstrual cycle around important competitions, reduced risk of iron-deficiency anemia resulting from decreased

blood loss via menstrual bleeding, and reduced loss of bone mass in estrogen-deficient athletes with amenorrhea (Bennell, White, & Crossley, 1999). Despite these potential benefits, plus the obvious one of birth control, many athletes (and their coaches) worry that taking the pill will cause weight gain, decrease aerobic capacity, and negatively affect fuel metabolism. However, there is very little evidence to support these fears (Bonen, Haynes, & Graham, 1991; Bryner, Toffle, Ullrich, & Yeater, 1996; Casazza, Suh, Miller, Navazio, & Brooks, 2002; Jankowski, Ben-Ezra, Gozansky, & Scheaffer, 2004). Only a few studies have investigated the effects of oral contraceptives on athletic performance and only a fraction of these used highly trained athletes as subjects. It is important to recognize that the effects of OCAs in trained women may differ from those observed in sedentary women.

A study of endurance-trained athletes and sedentary control subjects examined the effects of 10 months of OCA treatment (both estrogen and progesterone) on body weight and composition, aerobic capacity (maximal oxygen consumption), muscular strength, and bone mineral density. Half of the athletes were regularly menstruating and the other half were either oligomenorrheic (cycles at intervals of > 6 weeks) or amenorrheic (absence of menstrual cycles for at least 3 consecutive months) (Rickenlund, Carlstrom, et al., 2004). The women with irregular menstrual cycles gained weight (body weight increase from 124 to 128 lbs, on average) and increased their percentage of body fat (from 17% to 20%, on average) after OCA treatment. Despite this increase in body weight, relative maximal oxygen consumption did not change with OCA treatment (56.7 vs. 55.6 mL/kg/min). The bone mineral density of the women with oligomenorrhea and amenorrhea significantly increased with OCAs, and the improvement in BMD was greatest in individuals with the lowest initial BMD. Ten months of OCAs did not alter body weight, body composition, aerobic capacity, strength, or bone mineral density in the regularly menstruating women or in the sedentary control subjects.

## Dietary Supplements as Ergogenic Aids

### SUPPLEMENT LABELING AND PERFORMANCE CLAIMS

The laws regarding supplements put the burden of investigating the benefits and harms of dietary supplements on the consumer. Because of the way dietary supplements are regulated, at least in the United States, manufacturers may put essentially any performance-related claim on the label, even if it is unsubstantiated. Unlike food and drugs, the U.S. Food and Drug Administration does not monitor supplements for ingredient content or purity. In other words, the ingredients on the label may or may not be present and additional compounds not listed on the label may be included. For example, small amounts of anabolic steroids have been found in supplements that claim to increase muscle mass. Many supplements that are evaluated by consumer protection groups are found to contain much less of the active ingredient than what is listed on the label. Both practices are illegal, but, without quality control of the supplement industry, they take place nonetheless. Therefore, safety of dietary supplements is a concern. In the first 6 months that adverse events associated with dietary supplements were required to be reported by law, there were 604 "adverse events," including five deaths reported (Perez, 2008). It is estimated that this represents only 1% of the actual adverse events. A good source of reliable information on dietary supplements is the National Center for Complementary and Alternative Medicine of the National Institutes of Health (http://www.nccam.nih.gov/health). Information on whether a supplement contains what is on the ingredient list can be found at www.consumerlabs.com.

### PLACEBO EFFECT

The power of the mind cannot be underestimated. If an athlete believes that a supplement is going to help his or her performance, it probably will. However, if the ergogenic effects of that supplement were studied in a double-blind placebo-controlled trial (meaning subjects randomly receive the supplement or an identical-looking placebo treatment and neither the subjects nor the investigators know who gets what), a significant difference between the supplement and placebo may not be detected.

Dietary supplements range from the conventional (multivitamins) to the everyday (caffeine), exotic (Chinese herbs), weird (caterpillar fungus), disgusting (pituitary extract), and dangerous (ephedra). In general, supplements taken to correct a nutrient deficiency will benefit a deficient individual. With a few exceptions, there is little evidence from double-blind placebo-controlled studies to support the use of dietary supplements to enhance athletic performance. A search of PubMed, a database of peer-reviewed journal articles, using the phrase "supplements and sports" returned more than 1,600 entries, with most of these studies finding no effect of supplements on performance.

Even among the few supplements where a performance-enhancing effect has been repeatedly demonstrated, the magnitude of the effect is relatively small compared with the sum of the improvements that could be obtained from increased training or better equipment (Jeukendrup & Martin, 2001).

### VITAMIN AND MINERAL SUPPLEMENTS

Consuming excessive amounts of any nutrient (even water) can have detrimental effects. Vitamin and mineral excesses produce negative health consequences. Potential toxicity is greater for fat-soluble vitamins than for water-soluble vitamins because excess fat-soluble vitamins are stored in the liver and body fat, accumulating to toxic levels over time. For example, consuming just three to four times the RDA for vitamin A results in toxicity symptoms of loss of appetite, hair loss, and bone and muscle pain. Excess vitamin D causes calcification of the organs, high blood pressure, and kidney dysfunction. Large doses of water-soluble vitamins also can be harmful. For example, vitamin C normally acts as an antioxidant, preventing damage to the cell membrane by reacting with harmful molecules. However, at high

levels (>50 times the RDA), vitamin C can act as a pro-oxidant, reacting with iron or copper to generate compounds that can cause cell damage. Megadoses of vitamin $B_6$ can cause degeneration of nerves, resulting in unsteady gait, numbness in the extremities, and impaired tendon reflexes. Consumption of iron in excess of the RDA can result in iron toxicity. Because iron is a pro-oxidant, it damages cell membranes and can ultimately result in loss of organ function.

Consuming too much of one nutrient often creates a deficiency of another. This can happen in several ways. If two or more nutrients are absorbed by the same pathway (i.e., transport proteins) in the intestine, there is competition among those nutrients for absorption. For example, zinc, copper, and iron are absorbed by the same pathway. So, if large amounts of one of these minerals are consumed, that mineral monopolizes the transport system and less of the other two minerals is absorbed. If two nutrients have similar chemical properties, they can interfere with each other's function in biochemical reactions. For example, calcium and magnesium are similar chemically but have opposing actions on blood clot formation. Calcium is needed for the clotting process. At high doses, magnesium substitutes for calcium, inhibiting blood clot formation. Similarly, at very high doses, vitamin E interferes with the actions of vitamin K.

Consumption of supplemental vitamins and minerals in excess of needs is a waste of money. Absorption and excretion of vitamins and minerals are highly regulated. In general, the more the body needs, the more is absorbed from the intestine. By limiting nutrient absorption to what is needed, the chances of toxicity are reduced. Likewise, if the vitamin or mineral is already adequate, the excess that is consumed is either stored or excreted. However, these built-in safety mechanisms of absorption and excretion can be overwhelmed if nutrients are consumed in large amounts.

## CREATINE

Creatine is a nitrogen-containing compound that is made in the liver and kidneys from the amino acids arginine and glycine. About 1 to 2 g of creatine is synthesized in the human body per day, and the typical diet provides another 1 to 2 g/day from meat and fish. Skeletal muscle contains most of the body's creatine, but there is an upper limit to the amount of creatine that the muscle will retain. Any excess creatine consumed in the diet or from supplements is excreted by the kidneys. For this reason, individuals who consume adequate amounts of creatine in their diet are less likely to derive any benefit from taking additional creatine than are vegetarians whose diets are lacking creatine. The effects of creatine supplementation on oxygen consumption during exercise depends on the exercise intensity, fitness of the subjects, and the proportions of type I and type II muscle fibers (which is related to cardiovascular fitness but also influenced by genetics) (Jones, Carter, Pringle, & Campbell, 2002; Wyss & Kaddurah-Daouk, 2000).

In individuals who respond to supplementation, the amount of creatine in skeletal muscle typically increases 15% to 20% and body weight increases 1 to 3 kg during the first 5 to 7 days of supplementation. Creatine is not anabolic; it does not stimulate muscle protein synthesis or muscle growth. The rapid weight gain associated with creatine supplementation is to the result of water retention in the muscle cells. When the muscle cells take in more creatine, they also have to retain more water to maintain the correct intracellular fluid pressure. The extra water not only decreases the power to weight ratio (by increasing weight and not power), but it may impair muscle function and cause muscle stiffness and cramping. Because body water will shift from the blood to the muscle cells, sweating and thermoregulation may be impaired. Athletes who supplement with creatine must be especially careful not to become dehydrated—they should consume copious fluids and refrain from strenuous exercise, especially in the heat.

Creatine is used by skeletal muscle to make phosphocreatine. During maximal anaerobic efforts, phosphocreatine allows the muscle to generate large amounts of ATP very rapidly. However, there is only enough phosphocreatine to last 30 seconds. The idea behind "creatine loading" is that by increasing the amount of creatine in the muscle, there will be more phosphocreatine on hand to generate ATP. If there is more ATP available for use during maximal anaerobic efforts, the muscle will have more energy and performance will improve. Creatine supplementation has been shown to improve performance in repeated cycling sprints and maximal weight lifting efforts. In other words, creatine does not increase maximal power or strength but allows an individual to achieve his or her maximum potential during repetitive tests separated by short rest intervals. Creatine does not increase endurance performance because ATP is derived from aerobic metabolism of glucose and fat and not from phosphocreatine. There is not enough evidence supporting an ergogenic effect of creatine on endurance performance to endorse its use for that purpose; this position is consistent with that published by the American College of Sports Medicine (Terjung et al., 2000).

## GLUTAMINE

Glutamine is a conditionally essential amino acid, meaning that under normal circumstances the body can make what it needs. Skeletal muscle is the most significant source of glutamine in the body. When muscle is broken down in response to the stress hormone cortisol, glutamine is released from the muscle into the blood. During times of extraordinary stress, such as severe trauma, burns, or sepsis, the body's demand for glutamine exceeds its ability to make the amino acid and it becomes essential (Castell, 2003). In clinical studies of critically ill patients, providing supplemental glutamine improved patient outcome and survival. This is in large part because cells of the immune system use glutamine for energy. During times of stress, the immune system is activated, requiring more energy. Although not nearly as stressful as a burn injury or sepsis, exhaustive exercise (e.g., running a marathon) causes a short-term (< 12 hours) decrease in blood glutamine concentrations. Overtraining syndrome also produces a decrease in blood glutamine concentrations (Halson, Lancaster, Jeukendrup, & Gleeson, 2003).

Because athletes competing in endurance events often experience a mild decrease in immune function and an increase in upper respiratory tract infections after the event, the effect of glutamine supplementation on immune response has been studied in endurance athletes (Krieger, Crowe, & Blank, 2004; Krzywkowski, Petersen, Ostrowski, Kristensen, et al., 2001; Krzywkowski, Petersen, Ostrowski, Link-Amster, et al., 2001). One study found that runners who consumed glutamine immediately and 2 hours after a marathon had fewer self-reported upper respiratory tract infections in the week after the race. Consuming glutamine for 3 to 4 weeks before competition also reduced self-reported infections in marathon runners and triathletes. However, numerous other studies investigating the mechanism behind the beneficial effect of glutamine found no effect of glutamine supplementation on immune cell function.

It should be noted that glutamine is not stable in solution and exposure to ultraviolet light degrades it as well. The effective dose (0.1 g glutamine/kg BW) should not be exceeded. Amino acids compete with each other for absorption in the intestine and consuming an excess of one amino acid can create a deficiency of another. Excessive protein consumption unnecessarily stresses the kidneys, and excess amino acids that are not used in protein synthesis will be stored as adipose tissue.

## OTHER SUPPLEMENTS

Nitric oxide is made in the body from l-arginine and oxygen. This gaseous signal molecule plays an important role in many physiologic processes, including smooth muscle contraction, immune function, and nervous system activity. Because nitric oxide is a potent vasodilator, it plays an important role in oxygen use during exercise. Exercise stimulates nitric oxide production in blood vessels that perfuse skeletal muscle and causes increased blood flow to the working muscle (Krzywkowski, Petersen, Ostrowski, Kristensen, et al., 2001; Krzywkowski, Petersen, Ostrowski, Link-Amster, et al.,

2001; Miyauchi et al., 2003). Interestingly, nitric oxide decreases mitochondrial oxygen consumption by inhibiting several key enzymes involved in synthesis of ATP (Stamler & Meissner, 2001). The net result is that nitric oxide release acts to increase the oxygen available to muscle by increasing delivery and decreasing the rate at which it is used. Because nitric oxide is a gas, it is not commercially available in supplement form. Dietary supplements that claim to increase nitric oxide levels contain arginine.

*Cordyceps sinensis*, also known as Chinese caterpillar fungus, was popularized by the sudden success of the Chinese female distance runners in the early 1990s. When Wang Junxia shattered the world record in the 10,000 m, her coach attributed her performance to a diet of turtle blood and caterpillar fungus. Needless to say, the success and running careers of the Chinese women were short-lived. *Cordyceps* is a black fungus that is a parasite for several species of caterpillar. The fungus kills the caterpillar and uses it for nutrients as it grows. Because *Cordyceps* in nature is rare, a strain (Cs-4) that contains the active components is now cultivated for commercial purposes (Zhu, Halpern, & Jones, 1998a, 1998b). Other than the success of the Chinese runners, there is very little evidence to support the claim that *Cordyceps* improves performance. In fact, two recent studies of trained male cyclists found no effect of *Cordyceps* supplementation on maximal oxygen consumption, ventilatory threshold, or performance compared with placebo (Parcell, Smith, Schulthies, Myrer, & Fellingham, 2004).

## Sports During Pregnancy, Lactation, and Menopause

### PREGNANCY AND SPORTS

The American College of Obstetrics and Gynecology recommends that all women with uncomplicated pregnancies participate in moderate physical activity for at least 30 minutes most, if not all, days of the week (American College of Obstetricians and Gynecologists, 2002). Regular exercise has been shown to reduce the occurrence of physical complaints during pregnancy, to improve mood, and to shorten delivery time (Clapp, 2000). Women who are accustomed to exercising at high intensity and female athletes may continue to train vigorously during pregnancy, but they should be aware that some of the physiologic changes associated with pregnancy may require them to lower the intensity or modify the type of activity. Resting metabolic rate is increased during pregnancy, meaning the body requires more oxygen at rest. As the uterus grows during the second and third trimesters, it puts pressure on the diaphragm, increasing the work required to breathe. Because the body requires more oxygen and has to work harder to obtain that oxygen, less oxygen is available for exercise. For this reason, maximal workload may be reduced and exercise may feel more difficult at a given intensity during pregnancy. Another consequence of the elevated metabolic rate is an increased susceptibility to overheating and dehydration. There is some evidence that maternal overheating (core temperature above 39.2°C) during the first trimester may increase the risk of birth defects. Therefore, pregnant women should avoid exercising in hot humid weather and consume adequate fluids in the event of fever. The weight gain associated with pregnancy changes a woman's center of gravity, which may preclude activities that require balance to avoid abdominal trauma.

Pregnant women require an additional 300 kcal/day for growth of the fetus and maternal tissues (Institute of Medicine, Food and Nutrition Board, 2003). Inadequate energy consumption during pregnancy results in a low-birth-weight infant, which increases the chances of neonatal complications. Hormones produced by the placenta (human placental lactogen, estrogen, and progesterone) cause changes in maternal metabolism such that the mother preferentially uses fat for energy, which allows glucose to be used by the fetus. These metabolic changes cause pregnant women to have lower fasting glucose levels and to use more glucose during exercise than nonpregnant women do. Therefore, adequate carbohydrate consumption especially is important during exercise.

### LACTATION

The American Academy of Pediatrics (2005) recommends that infants be breastfed for at least 12 months. Breastfeeding not only benefits the infant but is advantageous for the mother as well. Exercise during lactation enhances cardiovascular fitness, decreases postpartum weight retention, improves postprandial insulin response, and increases high-density lipoprotein (Larson-Meyer, 2002). Lactating women may exercise without any adverse effect on milk volume or composition, infant feeding, or growth. Maximal, but not submaximal, exercise increases the acidity of breast milk for 0.5 to 1.0 hours after exercise. Whether the increased acid content reduces milk consumption is unclear. Wright, Quinn, and Carey (2002) in a well-controlled study suggested that the increase in acid has no effect on milk consumption.

### MENOPAUSE

Menopause is defined as the absence of regular menstrual cycles for 12 consecutive months. Typically, women experience menstrual cycle irregularity before cycles stop completely; changes in cycle length and frequency are common. During this perimenopausal period, estrogen concentrations also may be highly variable, and after 3 months of missed periods, estrogen declines significantly. Menopause, like puberty, is a time of hormonally driven changes in body shape and composition. During middle age, women gain an average of 0.5 kg (~1 lb) per year, and menopause does not seem to increase this rate of weight gain. Women lose muscle mass and increase fat mass during menopause, but these changes seem to be to the result of a decrease in physical activity as women get older rather than an inevitable consequence of menopause. Even if total body fat does not increase after menopause, there is a shift in body fat distribution from the hips and thighs to the abdomen.

The accumulation of fat around the internal organs located in the abdomen probably causes changes in fat and glucose metabolism. Postmenopausal women have higher total cholesterol, low-density lipoprotein (bad) cholesterol, and triglycerides (fat) and lower high-density lipoprotein (good) concentrations in blood than do premenopausal women. These changes explain why the risk of cardiovascular disease increases in women after menopause. Women, especially those who gain abdominal fat after menopause, may become insulin resistant. These changes in body composition and metabolism result not only from the direct effects of lower estrogen but also from estrogen-mediated changes in other hormones. GH and IGF-I exert anabolic effects on bone and muscle, and GH increases fat use as an energy source by stimulating release of fatty acids from body fat stores. Hypoestrogenemia reduces pulsatile release of GH, consequently lowering IGF-I secretion.

Exercise counteracts many of the unfavorable metabolic changes that occur after menopause by reducing weight gain, increasing fat utilization, maintaining skeletal muscle mass, improving cholesterol and triglycerides, and increasing the body's response to insulin. Strength training helps to prevent the decline in muscle mass and offsets the decline in metabolic rate. Strength training with weights also benefits the skeleton by stimulating bone growth because of the mechanical stress placed on the skeleton.

Adequate calcium intake is especially important for postmenopausal women. In addition to dairy products, some vegetables, fish with bones, and fortified foods (e.g., orange juice, cereal) are good sources of calcium. During the first 5 to 7 years of menopause women lose up to 20% of their skeletal mass (U.S. DHHS, n.d.). Consuming 1,200 mg calcium/day may minimize that loss.

# Pediatric Diabetes

*Karen Chapman-Novakofski, RD, LDN, PhD*

## Incidence and Prevalence

Type 1 diabetes is one of the most common chronic diseases of childhood. The incidence of type 1 diabetes peaks in children between 5 and 7 years of age and at puberty (Haller, Atkinson, & Schatz, 2005). The United States has an incidence rate of approximately 17 per 100,000/year for type 1 diabetes in youth, with a higher incidence in the northern regions as compared to the southern regions. Non-Hispanic white children have a higher incidence than do other racial groups (Stanescu, Lord, & Lipman, 2012). The number of children who have **type 1 diabetes** in the United States is expected to rise from 166,018 in 2010 to 203,382 by 2050. With this increase, more ethnicities will be affected, accounting for approximately half of the cases in 2050 (Imperatore et al., 2012).

**type 1 diabetes** Diabetes caused by a lack of endogenous insulin.

Children are also being diagnosed with **type 2 diabetes**, with an incidence rate of approximately 7.3 per 100,000/year (Stanescu et al., 2012). The prevalence of type 2 diabetes in youth is highest among Hispanic and non-Hispanic black children. Similar to type 1 diabetes, the projected prevalence rates are expected to increase from 22,820 in 2010 to 84,131 in 2050 (Imperatore et al., 2012). One study found type 2 diabetes in youth to be more prevalent in girls and among children with a family history of the disease (Copeland et al., 2011).

**type 2 diabetes** Diabetes caused by ineffective or low levels of insulin.

## Diagnosis of Diabetes in Children

The diagnosis of diabetes in children does not differ from diagnosis in adults. Symptoms of diabetes plus a random (nonfasting) blood glucose level of 200 mg/dL (11.1 mmol/L) or greater; fasting (fasting for 8 hours) plasma glucose level of 126 mg/dL (7 mmol/L) or greater; a 2-hour plasma glucose level of 200 mg/dL (11.1 mmol/L) or greater during an oral glucose tolerance test (75 g of anhydrous glucose dissolved in water); and a hemoglobin A1c value greater than or equal to 6.5% are all diagnostic of diabetes. However, these criteria should be repeated on a different day to confirm results (American Diabetes Association, 2013). These measurements may be less reliable in children, especially during puberty, although different criteria or tests have not been universally endorsed (Mizokami-Stout, Cree-Green, & Nadeau, 2012).

Screening for type 2 diabetes with any of the measures described previously is recommended for high-risk children. In general, this includes children who are overweight as defined by a body mass index (BMI) greater than the 85th percentile for age and sex, weight for height greater than 85th percentile, or weight greater than 120% of ideal for height. This group of children is further refined to those who also have two or more of the following risk factors: a family history of type 2 diabetes; of Native American, African American, Latino, Asian American, or Pacific Islander ethnicity; clinical symptoms of insulin resistance; or a mother who has had gestational diabetes. Although measurement of islet autoantibodies can identify children who are at risk for developing type 1 diabetes, this screening is generally only conducted within a clinical study (American Diabetes Association, 2013).

### Pathogenesis of Type 1 Diabetes in Children

Although there remain many unknowns about the pathogenesis of type 1 diabetes in children, in general, the genetically susceptible child is exposed to an environmental trigger that results in pancreatic beta cell autoimmunity and eventual self-destruction (Bending, Zaccone, & Cooke, 2012). The autoimmunity is specific to the pancreatic beta cells, but the mechanisms have yet to be identified. Typically, it is believed that symptoms do not occur until 80% to 90% of the beta cells have been destroyed, although evidence suggests that symptoms may present when only 40% to 50% have been destroyed (Haller et al., 2005). During the progression of type 1 diabetes, both the number of cells and the function of remaining cells are reduced (Bending et al., 2012).

**Learning Point** The classic symptoms of type 1 diabetes are polyuria, polydipsia, polyphagia, and weight loss, with children often brought to the hospital with diabetic ketoacidosis.

### Diabetes and Cultural Diversity

The incidence and prevalence of type 1 diabetes varies dramatically around the globe. Type 1 diabetes is uncommon in China, India, and Venezuela. The incidence of type 1 diabetes in children is highest in Canada and the Scandinavian countries of Norway, Sweden, and Finland, followed by Greenland and Australia. However, the prevalence of type 1 diabetes is strongest in Southeast Asia followed by European countries. Although type 2 diabetes in children is increasing, data are very limited on a global scale. It is known that type 2 diabetes incidence is steeply increasing in the United States and Japan (Kitagawa, Owada, Urakami, & Yamauchi, 1998). The variations in incidence and prevalence rates worldwide may reflect differences in gene susceptibility, environmental risk factors, or differences in the quality of data collection.

In the United States, the rates of type 2 diabetes are approximately 1.7 times higher for girls than for boys and may be linked to obesity (Artz, Haqq, & Freemark, 2005). Ethnic youth comprise a substantial number of cases of type 2 diabetes. Among teenage youth, Pima Indians have a very high prevalence of almost 5.1% (Fagot-Campagna et al., 2000). The prevalence of impaired fasting glucose levels has been reported to be significantly different among ethnic groups for adolescents: 13% Hispanic-American, 7% non-Hispanic American white, and 4% non-Hispanic African American youth (Williams et al., 2005).

Type 1 diabetes is a complex disease despite responding to genetic influences. Although having a first-degree family member with type 1 diabetes increases the risk of developing the condition, 85% of those diagnosed with type 1 diabetes have no family history of the disease (Hamalainen & Knip, 2002). The genetic predisposition includes human leukocyte antigen (HLA) class I and II genes (Christen, Bender, & von Herrath, 2012).

Clearly, environmental influences are also important. Among those environmental factors, both viral and nonviral agents have been suggested. Prenatal rubella, maternal enterovirus infection, and common childhood diseases such as mumps, chickenpox, measles, and rotavirus have possible associations with autoimmunity and type 1 diabetes (Haller et al., 2005). Some of the strongest epidemiological evidence involves enterovirus, which has also been isolated from the pancreas, gastrointestinal tract, and blood of those with type 1 diabetes (Christen et al., 2012). Although breastfeeding seems to exert a protective effect (Patelarou et al., 2012), the early introduction of cow's milk as a potential environmental trigger remains controversial (Clemens, 2011; Lempainen et al., 2012). The role of possible allergens such as wheat, and specifically gluten, during the first 3 months of life has been suggested but requires additional research (Hummel, Pflüger, Hummel, Bonifacio, & Ziegler, 2011; Hummel & Ziegler, 2011). Many other perinatal factors have been proposed, including maternal-child blood group incompatibility, maternal preeclampsia, and maternal age (Haller et al., 2005). Additional research is needed to define the role of these factors in the pathogenesis of type 1 diabetes.

Type 1 diabetes is at times further classified as type 1A, which is classic type 1 diabetes, and type 1B, which is diabetes with no known etiology. In type 1B, there is no evidence of autoimmunity (Concannon, Rich, & Nepom, 2009).

### Pathogenesis of Insulin Resistance in Children

Insulin resistance is characterized by **hyperinsulinemia**. The increased levels of insulin attempt to compensate for a declining peripheral sensitivity to insulin to maintain blood glucose levels within a normal range. The hyperinsulinemia is often accompanied by an increase in pancreatic islet cell size and beta cell mass (Artz & Freemark, 2004). Insulin resistance may be defined as the inability of circulating insulin levels to promote peripheral glucose disposal and to suppress hepatic glucose production. It is difficult to provide diagnostic classifications for insulin resistance. Fasting insulin levels may be used, but often differ according to diagnostic laboratory and may not relate to insulin resistance in children (Levy-Marchal et al., 2010). The homeostatic model assessment (HOMA) also is used to identify insulin resistance (Reaven, 2005). Although the HOMA is the most widely used method to evaluate insulin resistance in children, it may not offer more information than fasting insulin alone and can also vary with BMI (Schwartz et al., 2008).

**hyperinsulinemia** Blood levels of insulin above the normal range.

Insulin resistance often manifests during puberty, but may resolve after puberty. Children of ethnic minorities are at greater risk of insulin resistance, as are those children who are obese (Levy-Marchal et al., 2010). The consequences of insulin resistance in youth are increased risk for prediabetes and type 2 diabetes as well as for metabolic syndrome and cardiovascular disease. However, data from which to draw conclusions as to the degree of risk associated with insulin resistance and these diseases are limited (Levy-Marchal et al., 2010).

### Other Diabetes Classifications in Children

Maturity onset diabetes of the young (MODY) is a group of disorders characterized by beta cell

dysfunction. It is difficult to distinguish MODY from types 1 or 2 diabetes, and so prevalence rates are not reliable. In MODY, some beta cell function is maintained, whereas in type 1 diabetes all beta cell function is gone after 3 to 5 years. Therefore, a child thought to have type 1 diabetes but who does not exhibit ketoacidosis when insulin is not given may have MODY rather than type 1 diabetes. Children with type 2 diabetes but without insulin resistance may also have MODY (Thanabalasingham & Owen, 2011).

Double diabetes, or diabetes type 1.5, comprises those children with characteristics of both types of diabetes. That is, a child with type 2 diabetes who also has autoantibodies to beta cells, or a child with type 1 diabetes who becomes obese (Pozzilli, Guglielmi, Caprio, & Buzzetti, 2011).

### Risk Factors for Diabetes

Genetics plays a significant role in the development of both type 1 and type 2 diabetes. In type 1 diabetes, the risk increases from a risk of 0.4% in the general population to 3–6% for those with a sibling or parent with type 1 diabetes (Stanescu et al., 2012). In type 2 diabetes, the genetic component is stronger, with 74% to 100% of children with type 2 diabetes having a first- or second-degree relative with the condition (Morgan, 2012).

In addition to genetics, the development of diabetes is influenced by a number of environmental factors associated with the incidence of diabetes. These have been discussed in relation to type 1 diabetes development. In addition, however, an increase in obesity has been associated with type 1 diabetes in youth, as well as with type 2 diabetes (Badaru & Pihoker, 2012). Insulin resistance in type 2 diabetes is linked to hyperinsulinemia and beta cell dysfunction (D'Adamo & Caprio, 2011).

The dietary factors that have been shown to be associated with adiposity in children include dietary fat (moderate evidence), total energy intake (moderately strong evidence), sugar-sweetened beverage intake (strong evidence), and energy density (moderately strong evidence), according to the Nutrition Evidence Library of the USDA. Intake of 100% fruit juice, fruits and vegetables, and dietary fiber had limited evidence of being associated with adiposity in children. There was moderately strong evidence that intake of calcium and/or dairy had no relationship to adiposity in children (USDA Nutrition Evidence Library, 2010).

Effective strategies for preventing obesity in children have included school curriculum for healthy eating and physical activity; physical activity in the schools; healthy lunches in the schools; and activities at home that promote healthy eating and physical activity. However, it has been noted that there is great heterogeneity among studies investigating childhood obesity prevention, and the contribution of various components cannot be measured (Waters et al., 2011).

### Associated Clinical Conditions

Youth with type 2 diabetes may present with **acanthosis nigricans**, a velvety skin texture often found in intertriginous areas such as skinfolds of the groin, axilla, and breasts as well as the nape of the neck. The skin condition is often associated with obesity and is more prevalent in the African American population (Hermanns-Lê, Scheen, & Pierard, 2004) but has been reported to be closely associated with insulin resistance in Native American children as well (Copeland et al., 2006). Although many youth with type 2 diabetes have acanthosis nigricans, not all youth with acanthosis nigricans develop diabetes (Brickman, Huang, Silverman, & Metzger, 2010).

**acanthosis nigricans**
A velvety skin texture often found in skinfold areas that is frequently associated with obesity and type 2 diabetes.

The mechanism by which hyperinsulinemia may cause acanthosis nigricans is complex and not completely understood. The process probably involves excess insulin binding to IGF receptors that increase fibroblast and keratinocyte proliferation. This increase may lead to subsequent defects in the skin tissue (Hermanns-Lê et al., 2004).

**Polycystic ovarian syndrome (PCOS)** is associated with obesity and insulin resistance in young women, which increases the risk for metabolic syndrome and type 2 diabetes (Connor, 2012). The diagnosis is difficult because PCOS is a clinical syndrome, but generally the presence of two of the following three conditions is diagnostic: oligo- or anovulation, hyperandrogenism, and polycystic ovaries. There is concern that requiring only two rather than all three criteria may overdiagnose PCOS in adolescents (Rackow, 2012). Usually manifested during adolescence, PCOS may originate in intrauterine development (Xita & Tsatsoulis, 2010). Clinical symptoms can include menstrual irregularities, acne, hirsutism,

**polycystic ovarian syndrome (PCOS)**
A clinical syndrome in women consisting of two of the following three conditions: oligo- or anovulation, hyperandrogenism, and polycystic ovaries.

and alopecia (Brewer, Pawelczak, Kessler, & Shah, 2010). For overweight adolescents with PCOS, weight loss through diet and physical activity is recommended for improvement in insulin sensitivity and decreasing androgens. For those with hirsutism, hair removal treatments may improve this clinical symptom, although pharmacological treatment is needed to prevent new hair growth. Oral contraceptives containing both estrogen and progestin also may help manage hirsutism as well as menses and acne. Metformin is often used for those with insulin resistance (Bremer, 2010).

### Management

For children with type 1 diabetes, management should be guided by a multidisciplinary team trained in pediatric diabetes. The individual child and family should be considered when responsibilities are suggested to parent and child. The child's age should guide the degree of **glycemic control** that is targeted (see **Table 8.3**). The benefits of tighter control and delay of chronic conditions must be weighed against the risk of hypoglycemia. When the child is 10 years old, screening for microalbuminemia and ophthalmologic changes should occur. If hyperlipidemia is present in the family, the child should also be screened for this disorder. Additional screening for celiac disease and hypothyroidism should occur soon after diagnosis with diabetes, and blood pressure should be monitored. These recommended screenings also apply to children with type 2 diabetes (American Diabetes Association, 2013).

**glycemic control**
Maintaining blood glucose within the target range.

The general guideline for those with type 1 diabetes is treatment with multiple daily injections (MDIs) of basal and prandial insulin or continuous subcutaneous insulin infusion (CSII) (American Diabetes Association, 2013). For preschoolers with type 1 diabetes, insulin pumps are often begun rather than insulin injections (Kordonouri et al., 2011).

Medical nutrition therapy is an important factor in achieving both glycemic control and normal growth and development. Macronutrient distribution is based on adult recommendations but should be tailored to meet individual needs. Total calories should be balanced between needs for growth and prevention of obesity. The Dietary Reference Intakes listed in **Table 8.4** are guidelines

**TABLE 8.3 Plasma Blood Glucose and Glycosylated Hemoglobin (A1c) Goals for Type 1 Diabetes by Age Group**

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Plasma Blood Glucose Goal Range (mg/dL)** | | | | | | | | | | | | | | | | | | | |
| Before Meals | 100–180 | | | | | | 90–180 | | | | | | 90–130 | | | | | | |
| Overnight | 110–120 | | | | | | 100–180 | | | | | | 90–150 | | | | | | |
| **Targeted Glycemic Control** | | | | | | | | | | | | | | | | | | | |
| A1c(%) | < 8.5 | | | | | | < 8 | | | | | | < 7.5 | | | | | | |
| Years of Age | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |

Data from American Diabetes Association. (2013). Standards of Medical Care in Diabetes—2013. *Diabetes Care*, 36(Supp 1), S11–S66.

**TABLE 8.4 Estimated Energy Requirements for Children and Adolescents**

| | |
|---|---|
| **Children 3–8 Years** | |
| Boys | 88.5 – (61.9 × age [yr]) + PA × (26.7 × weight [kg] + 903 × height [m]) + 20 kcal |
| Girls | 135.3 – (30.8 × age [yr]) + PA × (10.0 × weight [kg] + 934 × height [m]) + 20 kcal |
| **Children 9–18 Years** | |
| Boys | 88.5 – (61.9 × age [yr]) + PA × (26.7 × weight [kg] + 903 × height [m]) + 25 kcal |
| Girls | 135.3 – (30.8 × age [yr]) + PA × (10.0 × weight [kg] + 934 × height [m]) + 25 kcal |

PA, physical activity coefficient: 1.0 if sedentary; 1.13 if low active; 1.26 if active; and 1.42 if very active

Data from Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: National Academies Press.

for estimating caloric needs (Institute of Medicine, 2005). For youth with type 2 diabetes, portion control, elimination of sugar-sweetened beverages, reduction of high-fat and high-calorie foods, and maintaining regular meals should be goals of medical nutrition therapy (Flint & Arslanian, 2011). Children with or without diabetes should be encouraged to have at least 60 minutes of physical activity each day (American Diabetes Association, 2013).

### The Role of Exercise

Exercise is an important part of a healthy lifestyle. National guidelines suggest that adolescents engage in physical activity for a minimum of 60 minutes most days (U.S. DHHS, 2008). Increasing the amount ofmoderate to vigorous physical activity is supported by evidence-based data for disease prevention in youth (Strong et al., 2005). However, few intervention studies have been conducted on the effects of physical activity on prevention or treatment of chronic disease in youth (Cruz et al., 2005).

For the child with type 2 diabetes, regular physical activity may help to achieve a healthier body weight. For the child with type 1 diabetes, care should be taken to prevent hypoglycemia (U.S. DHHS & National Diabetes Education Program, 2006). Hypoglycemia may occur after prolonged moderate-intensity aerobic exercise, as might occur in an after-school program, and may require more carbohydrate than a traditional 15 g for treatment (Diabetes Research in Children Network Study Group, 2006). Children on continuous subcutaneous insulin infusion may need to use a variety of strategies depending on the physical activity. The pump can be removed for intense exercise such as in organized sports, but it must be recognized that blood glucose may drop as a result of the intense exercise or rise because of the excitement of the game. There also may be a risk of nocturnal hypoglycemia after an eventful, full day of physical activity such as a soccer or basketball tournament. For prolonged yet even-exertion activities such as biking or hiking, the pump may be set at half the usual rate of infusion, although this should also be individually calibrated (Tamborlane, Fredrickson, & Ahern, 2003).

To reach national guidelines of physical activity for youth in general, families, schools, after- school programs, and youth sports and recreation programs should all be involved at the individual and community levels. To promote physical activity in youth, media campaigns and the communities' structural environment should be engaged (Centers for Disease Control and Prevention, n.d.). Although the American Diabetes Association (1999) and the American College of Sports Medicine (Pollock et al., 2000) recommend a combination of both strength training and aerobic exercise for those adults with diabetes, guidelines for youth are yet to be disseminated.

Initial education for the child with type 1 diabetes and his or her family may be in the medical center. Topics discussed are generally "survival strategies," such as how to store and inject insulin. After hospitalization, in the case of the child with type 1 diabetes and certainly for the child with type 2 diabetes, outpatient education should be multidisciplinary and similar to that for adults. For the child with type 1 diabetes, carbohydrate-counting education is essential for both the child and caregiver(s) if continuous subcutaneous insulin infusion or basal-bolus insulin regimens are to be successful. Insulin therapies need to be accompanied by self-management of blood glucose training and home glucose monitoring.

For the child with type 2 diabetes, body weight reduction must be a goal without compromising normal growth and development. Increased physical activity and healthy eating patterns and amounts are preferable to very restricted diets. The scientific literature supporting particular interventions for overweight and obese youth are lacking. Multicomponent interventions for families of children aged 5 to 12 years and school-based interventions for older children are recommended for treating obesity (American Dietetic Association, 2006). If lifestyle modifications are not successful, medication may be prescribed.

Currently, only insulin and metformin are approved by the U.S. Food and Drug Administration for children with type 2 diabetes. Metformin is an oral hypoglycemic agent whose main site of action is the liver, where it decreases hepatic gluconeogenesis and increases hepatic glucose uptake. Action at the peripheral tissues may also increase glucose utilization. Because metformin does not affect insulin levels, the risk of hypoglycemia is minimized. If metformin is not tolerated, or does not contribute to achieving desired glycemic control, insulin is initiated in the child with type 2 diabetes (Vaidyanathan, Choe, & Sahajwalla, 2012). As with adults, diabetes self-management education is essential for optimal care. However, in pediatric diabetes care, additional supportive measures are usually required to manage social issues of adolescents and the subsequent effect on glycemic control and general health (Berry, Urban, & Grey, 2006).

## *Disordered Eating*

*Pamela S. Hinton, PhD*

Eating disorders are psychiatric disorders with significant medical and psychosocial complications that affect approximately 5 million Americans, primarily adolescent girls and young women. Three eating disorders have been identified by the American Psychiatric Association: anorexia

nervosa (AN), bulimia nervosa (BN), and eating disorder not otherwise specified (EDNOS). The diagnostic criteria are outlined in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (*DSM-IV-TR*; American Psychiatric Association, 2000). Although behaviorally distinct, with AN characterized by severe dietary restriction and BN by binge eating and compensatory purging behaviors, the eating disorders share an excessive importance given to body weight and shape in relation to self-concept and self-esteem. Eating disorder diagnoses are mutually exclusive; however, an individual may meet diagnostic criteria for more than one disorder during the course of the illness.

## Anorexia Nervosa

AN is characterized by a relentless pursuit of thinness (see **Table 8.5**). Individuals with AN may achieve very low body weights via severe restriction of the amount and types of food eaten and compulsive and excessive exercise. Patients with binge eating–purging subtype of AN occasionally use self-induced vomiting, laxatives, or diuretics to control their body weight. AN is a progressive condition in that the standard for "thin" is lowered over time and patients are never satisfied with their body weight or shape. As weight loss progresses, impaired cognitive function, depression, anxiety, and social isolation worsen. Academic and work performance and personal relationships are often sacrificed to spend time exercising or to avoid social situations that require eating.

## Bulimia Nervosa

Individuals with BN also desire thinness, but their chronic dieting is interspersed with recurrent episodes of binge eating, followed by compensatory purging behaviors (e.g., self-induced vomiting) to "undo" the effects of the binge (see **Table 8.6**). As a result, most patients with BN are of normal body weight. Feelings of guilt, shame, depression, anxiety, and negative ego-syntonicity increase in parallel with the frequency of binge–purge cycles. Poor impulse regulation often manifests as substance misuse and abuse and self-harming behavior in patients with BN. For some individuals, BN also is a progressive condition with the need to binge–purge interfering with academic or work performance and personal relationships.

## Eating Disorder Not Otherwise Specified

EDNOS diagnoses should not imply a less serious condition or one that is not worthy of intervention. Typically, EDNOS is nearly identical to AN or BN

**TABLE 8.5** Diagnostic Criteria for Anorexia Nervosa

| Criterion | Description |
|---|---|
| A | Refusal to maintain body weight at or above a minimally normal weight for age and height. |
| B | Intense fear of gaining weight or becoming fat, even though underweight. |
| C | Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight. |
| D | In postmenarchal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only after hormone [e.g., estrogen] administration.) |
| **AN Subtypes** | |
| Restricting type | During the current episode of anorexia nervosa, the person has not regularly engaged in binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). |
| Binge eating–purging type | During the current episode of anorexia nervosa, the person has regularly engaged in binge eating or purging behaviors (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). |

Data from U.S. Department of Health and Human Services, National Institute of Mental Health (2001).

**TABLE 8.6** Diagnostic Criteria for Bulimia Nervosa

| Criterion | Description |
|---|---|
| A | Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:<br>1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances<br>2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating) |
| B | Recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise. |
| C | The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months. |
| D | Self-evaluation is unduly influenced by body shape and weight. |
| E | The disturbance does not occur exclusively during episodes of anorexia nervosa. |
| **BN Subtypes** | |
| Purging type | During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas. |
| Nonpurging type | During the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas. |

Data from U.S. Department of Health and Human Services, National Institute of Mental Health (2001).

but without meeting all the diagnostic criteria for either AN or BN (Henig, 2004). All the criteria for AN are met except the following:

- The individual has regular menses.
- The individual's current weight is in the normal range.

All the criteria for BN are met except these:

- Binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months.
- Regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food.
- Chewing and spitting out, but not swallowing, large amounts of food.
- Purging disorder.
- Binge eating disorder.

For example, an individual may meet all the diagnostic criteria for AN except amenorrhea. As in AN and BN, undue influence of body weight or shape on self-worth is the core psychopathology of most individuals who receive an EDNOS diagnosis. Binge eating disorder (BED) is a subtype of EDNOS, defined by criteria that are for research purposes only (see **Table 8.7**).

## Psychiatric Comorbidity

**psychiatric comorbidity** Presence of additional mental disorders, for example, substance abuse or personality disorder.

**Psychiatric comorbidity** is high among individuals with eating disorders (American Psychiatric Association, 2000; Becker, Grinspoon, Klibanski, & Herzog, 1999; Fairburn & Harrison, 2003), but as demonstrated in the Minnesota Starvation Study, starvation causes significant changes in mood, personality, and cognition and behavior (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). Thus, it is sometimes difficult to differentiate between primary and secondary psychological disturbances. Depression is common in both AN and BN, but affect improves significantly with refeeding and reduction in binge–purge symptomology. Obsessive-compulsive disorder and anxiety disorders are common among individuals with eating disorders, but the obsessions (thoughts),

**TABLE 8.7 Research Criteria for Binge Eating**

| Criterion | Description |
|---|---|
| A | Recurrent episodes of binge eating characterized by both of the following:<br>1. Eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat during a similar period of time under similar circumstances. Not continual snacking or grazing. Objective definition of quantity<br>2. A sense of lack of control over eating during the episode |
| B | Binge eating episodes are associated with three or more of the following:<br>• Eating much more rapidly than usual<br>• Eating until uncomfortably full<br>• Eating large amounts when not feeling physically hungry<br>• Eating alone because embarrassed about quantity<br>• Feeling disgusted with oneself, depressed, or very guilty after overeating |
| C | Marked distress regarding binge eating is present because of loss of control and its implications for weight and health. |
| D | Binge eating occurs, on average, at least 2 days a week for 6 months. |
| E | Binge eating is not associated with the regular use of inappropriate compensatory behaviors and does not occur exclusively during the course of anorexia nervosa. |

Data from U.S. Department of Health and Human Services, National Institute of Mental Health (2001).

compulsions (behaviors), and fears often are related to the eating disorder.

A significant proportion of individuals with eating disorders also suffer from personality disorders, and failure to treat comorbid personality disorders may preclude recovery from eating disorders. Cluster B (Antisocial, Borderline, Histrionic, Narcissistic) personality disorders are more common in individuals with BN; patients with AN are more likely to have Cluster C personality disorders (Avoidant, Dependent, Obsessive-Compulsive). Self-harming behaviors, substance misuse and abuse, and suicide are present at higher rates in patients with eating disorders compared with the general population. If psychological comorbidity is present, it is important to treat all pathologies; otherwise, diminution of the eating disorder may exacerbate symptoms of the remaining disorders.

## Etiology and Course of Eating Disorders

Eating disorders have multifactorial etiologies. Sociocultural, genetic, family, and personality factors predispose an individual to engage in behavioral risk factors, which are dieting and excessive exercise, which universally precede a clinical eating disorder. The estimated genetic heritability of eating disorders is 50–83% (Treasure, Claudino, & Zucker, 2010). Negative life events, role transitions, developmental milestones, sexual and physical abuse, and criticism of physical appearance often precipitate the progression from dieting to eating disorder (American Psychiatric Association, 2000; Fairburn & Harrison, 2003).

Of individuals who survive anorexia nervosa, 45% achieve full recovery, which is usually defined based on physical and behavioral criteria (i.e., normal body weight, regular menses, and normalization of eating patterns). An additional 30% of anorexia nervosa patients improve, and for the remaining 20% of affected individuals the eating disorder becomes chronic. For individuals with bulimia nervosa, the outcome is only slightly better (Steinhausen, 2002). Protection, control, and positive social reinforcement are benefits that individuals with eating disorder derive from their disorder, thus contributing to the chronicity of the conditions. AN has the highest mortality rate of any psychiatric illness with a

crude mortality rate of 5% (Steinhausen, 2002); the standardized mortality rate ranges from 5.9% to 12.8% and the suicide rate is 56.9% (Agras et al., 2004; Arcelus, Mitchell, Wales, & Nielsen, 2011).

The single most important positive prognostic factor is a short interval between onset of the eating disorder and intervention; thus, less severe weight loss and symptomology also are positive prognostic indicators (Agras et al., 2004; American Psychiatric Association, 2000; Fairburn & Harrison, 2003). Younger age at onset of the eating disorder also is associated with increased likelihood of recovery. Recovery is very difficult after the eating disorder has become **ego-syntonic** and the behaviors entrenched. Psychiatric comorbidity, high vomiting frequency, self-harming behaviors, and substance abuse disorders are negative prognostic indicators (Fairburn & Harrison, 2003).

**ego-syntonic** Consistent with self-concept.

## Epidemiology

Eating disorders are disorders of Western societies, although there are isolated reports of self-imposed starvation in adolescents from non-Western societies. Food refusal and thinness have little meaning where food is scarce and malnutrition is endemic. Eating disorders are more common in whites than in ethnic and racial minorities, and 90% of individuals with AN and BN are female. The disparity between whites and African Americans is less for BED than for AN and BN. The onset of AN typically occurs during adolescence, compared with young adulthood for BN and middle age for BED. Worldwide, the estimated lifetime prevalence of eating disorders is about 0.6% for AN, 1% for bulimia nervosa, and 3% for binge eating disorder (Treasure et al., 2010). Disordered eating is common among American adolescents, affecting 7% of boys and 13% of girls aged 9–14 years (Treasure et al., 2010). The incidence of AN is 19 females and 2 males per 100,000 per year compared with 29 females and 1 male for BN (Fairburn & Harrison, 2003). There is very little epidemiologic data regarding EDNOS, although these atypical eating disorders seem to affect primarily adolescents and young adult women (Fairburn & Bohn, 2005). However, compared with AN and BN, BED is more common among males—approximately 40% of patients with BED are male. The lifetime prevalence of binge eating disorder in adolescents in the United States is 2.3% for girls and 0.8% for boys, and the incidence of BED appears to be increasing over time. It is thought that the incidence of both AN and BN has increased in the recent past. However, it is very difficult to accurately determine the frequency of eating disorders in the general population (Agras et al., 2004). Because individuals with these disorders are reluctant to seek treatment, many eating disorders go undiagnosed in the community. In addition, there is bias in access to treatment, which in turn biases the reported prevalence of eating disorders among different ethnic and racial groups and between the genders.

## Medical Consequences of Eating Disorders

Routine laboratory tests include measurements of serum electrolytes (potassium, sodium, and chloride), glucose, blood urea nitrogen, creatinine, and a complete blood count (American Dietetic Association, 2001; American Psychiatric Association, 2000). However, normal laboratory findings are common in patients with eating disorders. Additional laboratory tests, such as serum calcium, magnesium, and phosphorus, liver and kidney function tests, electrocardiogram, bone density assessment, thyroid hormone, and estradiol or testosterone levels, are warranted if the patient exhibits signs and symptoms of malnutrition (American Psychiatric Association, 2000; Fairburn & Harrison, 2003; Winston, 2012).

Most of the medical complications of AN result from inadequate energy intake and thus are reversed upon refeeding (American Psychiatric Association, 2000; Fairburn & Harrison, 2003). The exception is loss of bone mineral density, which may persist after restoration of body weight (Heer, Mika, Grzella, Heussen, & Herpertz-Dahlmann, 2004). Fifty percent of women with AN have BMD that is more than 2 standard deviations below the average value for young adult women, and 40% have BMD that is one standard deviation below the young adult mean (Becker et al., 1999; Miller et al., 2004). In adolescents with AN, both bone formation and breakdown are reduced, resulting in bone structural abnormalities that are evident before reductions in BMD. The fracture rate is 30% in adolescents with AN and fracture risk is seven-fold greater than in age- and sex-matched controls. There is no proven

drug or hormonal treatment for loss of bone in adolescents with AN (Miller, 2004). Physical signs and symptoms and clinical abnormalities associated with AN are presented in **Table 8.8**.

Apparently, low energy availability is sensed in the brain; leptin and ghrelin (De Souza, Leidy, O'Donnell, Lasley, & Williams, 2004) may be signals from the periphery to the brain of energy status. Hypothalamic release of thyrotropin-releasing hormone and gonadotropin-releasing hormone are suppressed, resulting in decreased production of thyroid hormone (triiodothyronine, thyroxine), estrogen, and progesterone. In contrast, hypothalamic secretion of growth hormone–releasing hormone and corticotropin-releasing hormone is elevated and, subsequently, so are GH and cortisol. Although GH secretion is increased, the liver and extrahepatic tissues become GH resistant and systemic and local production of IGF-I is decreased. These hormonal alterations produce many of the physical signs and symptoms associated with AN:

- Low triiodothyronine results in cold intolerance, bradycardia, **orthostatic hypotension**, **acrocyanosis**, and delayed reflexes.

**orthostatic hypotension** A sudden fall in blood pressure that occurs when a person assumes a standing position, causing dizziness, lightheadedness, blurred vision, and syncope.

**acrocyanosis** Blueness of the extremities (the hands and feet), caused by narrowing (constriction) of small arterioles (tiny arteries) toward the end of the arms and legs.

**TABLE 8.8 Medical Consequences of Eating Disorders**

Physical symptoms
- Cold intolerance
- Gastrointestinal complaints
- Dizziness
- Amenorrhea
- Poor sleep with early morning waking
- Apathy, poor concentration
- Muscle pain
- Weakness, lassitude

Physical signs
- Emaciation; stunted growth and failure/regression of secondary sex characteristics
- Dry skin
- Lanugo
- Orange discoloration of palms and soles with hypercarotenemia
- Swelling of parotid and submandibular glands (vomiting)
- Erosion of inner surface of front teeth (perimylolysis, caused by vomiting)
- Scarring on dorsum of hand (Russell's sign, caused by self-induced vomiting)
- Esophagitis, gastroesophageal reflux, erythema of pharynx
- Cold hands and feet, hypothermia, acrocyanosis
- Bradycardia, orthostatic hypotension, cardiac arrhythmias
- Edema
- Muscle weakness
- Cognitive impairment, depressed irritable mood

Abnormalities on physical investigation
- Endocrine
  - ➤ Low luteinizing hormone, follicle-stimulating hormone, estradiol
  - ➤ Low triiodothyronine, thyroxine in normal range, normal thyroid-stimulating hormone
  - ➤ Mild hypercortisolemia
  - ➤ Elevated GH
  - ➤ Hypoglycemia
  - ➤ Low leptin
- Cardiovascular
  - ➤ Electrocardiographic abnormalities (especially prolonged Q–T interval)
- Gastrointestinal
  - ➤ Delayed gastric emptying
  - ➤ Decreased colonic motility
  - ➤ Acute gastric dilation
- Hematologic
  - ➤ Moderate normocytic normochromic anemia
  - ➤ Mild leucopenia with relative lymphocytosis
  - ➤ Thrombocytopenia
- Other metabolic abnormalities
  - ➤ Hypercholesterolemia
  - ➤ Hypercarotenemia
  - ➤ Hypophosphatemia
  - ➤ Dehydration
  - ➤ Electrolyte disturbance: metabolic alkalosis and hypokalemia (vomiting); metabolic acidosis, hypokalemia, and hyponatremia (laxative abuse)
- Other abnormalities
  - ➤ Osteopenia and osteoporosis
  - ➤ Enlarged cerebral ventricles and external cerebrospinal fluid spaces

Data from U.S. Department of Health and Human Services, National Institute of Mental Health (2001).

**leucopenia** A decrease in the number of white blood cells circulating in the blood.

**lanugo** Downy, very fine, soft, and usually unpigmented hair.

- Low estradiol causes anovulatory amenorrhea, loss of BMD, hypertriglyceridemia and hypercholesterolemia, elevated adhesion molecule expression, and impaired flow-mediated dilation.
- GH resistance and low IGF-I results in muscle atrophy, loss of BMD, stunting in growing adolescents, and, possibly, **leucopenia**.
- Hypercortisolemia may contribute to loss of muscle mass and BMD as well as to immune modulation.

Growth of fine downy hair (**lanugo**), dry skin, brittle hair, and electrocardiographic abnormalities, usually prolongation of the Q–T interval (Swenne, 2000), are characteristic signs of malnutrition.

The focus of eating disorder treatment should be correcting the self-imposed starvation rather than correction hormone abnormalities via replacement therapy (Fairburn & Harrison, 2003). However, because of the irreversible nature of the loss of BMD, use of anabolic and antiresorptive pharmacologic interventions may be warranted to treat osteopenia and osteoporosis (Miller et al., 2004). There is limited evidence that the combination of rhIGF-I and estradiol may ameliorate loss of BMD during AN (Grinspoon, Friedman, et al., 2003; Grinspoon, Miller, Herzog, Clemmons, & Klibanski, 2003; Grinspoon, Miller, Herzog, Grieco, & Klibanski, 2004).

The primary medical complications of BN result from electrolyte and fluid imbalances caused by self-induced vomiting and laxative and dieretic misuse. Frequent vomiting results in metabolic acidosis and hypokalemia; laxative misuse creates metabolic acidosis, hypekalemia, and hypnoatremia. Electrolyte abnormalities cause cardiac arrhythmias that are potentially life-threatening in severely underweight individuals. Peripheral edema and dehydration also result from electrolyte imbalances. Chronic laxative use creates colonic dependence; constipation results when laxative use is stopped because of decreased intestinal motility. Physical symptoms resulting from self-induced vomiting include callous on the dorsum of the hand (Russell's sign), swelling of the submandibular and parotid glands, and erosion of dental enamel on the occlusal and lingual surfaces of the teeth.

Individuals with EDNOS experience medical complications of AN and BN, depending on the nature of their disordered eating behaviors. A diagnosis of EDNOS does not mean that physical health is not jeopardized. Patients with BED are typically overweight and at risk for chronic diseases associated with excess adiposity.

## Assessment

Diagnosis of an eating disorder requires assessment of the patient's weight history, physical symptoms, eating, exercise, compensatory behaviors, and mental status. In addition, core beliefs about weight, body shape, and eating must be evaluated along with screening for other psychiatric disorders. Several standardized self-report measures can be used to evaluate symptoms (American Psychiatric Association, 2006). For adolescents, assessment should involve parents and possibly other adults who routinely interact with the teenager (e.g., teachers).

General physical assessment should include measurement of height and weight and evaluation of growth pattern and sexual development; vital signs, including heart rate and postural changes in blood pressure; dermatologic and dental signs; and evidence of self-harming behaviors (American Psychiatric Association, 2006).

## Treatment

Unfortunately, treatment of eating disorders, especially AN, often is dictated by insurance reimbursement rather than need or long-term efficacy. Treatment of AN is costly: Approximately 50% of patients require hospitalization, 50% are prescribed pharmacotherapy, and all require outpatient therapy. In the United States, short-term hospitalization to achieve nutritional stabilization or avert a medical crisis is the norm. However, in other countries, long-term hospitalization with weight restoration and psychotherapy is the standard (Agras et al., 2004). For adolescents with AN, family involvement and treatment are necessary (American Psychiatric Association, 2006).

### Treatment Goals

Ideally, treatment should achieve three objectives (American Dietetic Association, 2001; Fairburn & Harrison, 2003). First, effective management of eating disorders fosters motivation to change

disordered eating behaviors. The second goal is normalization of body weight, which not only improves physical health but also facilitates improvements in mood and responsiveness to psychotherapy. Malnourished individuals have impaired cognitive function, which limits the efficacy of psychotherapy. Patients with AN cannot respond to serotonin reuptake inhibitors during semistarvation because serotonin production is impaired by malnutrition. The third treatment goal is to help patients accord body weight and shape an appropriate level of significance in their self-evaluation, to reduce disordered eating and exercise patterns, and to improve psychosocial functioning. In the case of chronic eating disorders that are resistant to treatment, the goal of therapy is to minimize the negative physical, social, and emotional consequences of the eating disorder. Other treatment goals may include treatment of psychiatric comorbidities and family counseling when appropriate (American Psychiatric Association, 2006).

Eating disorder treatment programs generally use a multidisciplinary treatment team to achieve these therapeutic goals (American Psychiatric Association, 2000, 2006; Stewart & Williamson, 2004). A physician oversees the treatment plan and monitors the physical health of the patient. Psychotherapy, by a psychologist, psychiatrist, psychiatric nurse, or social worker trained in eating disorders, helps the patient address unresolved emotional issues, dysfunctional cognitive styles, problematic relationships, and psychiatric comorbidity. A nutritionist or registered dietitian with eating disorder expertise counsels the patient regarding healthful diet and modification of eating habits (American Dietetic Association, 2001).

### Inpatient Treatment

Outpatient therapy is preferable to inpatient whenever possible because of the high monetary cost of hospitalization and the perception by the patient that inpatient treatment is punitive (Agras et al., 2004; Stewart & Williamson, 2004). However, certain circumstances necessitate inpatient treatment: significant medical risk, very low body weight (< 75% ideal body weight), dehydration, electrolyte disturbances, cardiac dysrhythmia, physiologic instability, arrested growth, acute medical complications of malnutrition (**syncope**, seizures, cardiac failure, pancreatitis), acute psychiatric emergencies (suicidal ideation, self-harming behaviors), uncontrollable binge eating and purging, substance abuse, and circumstances that interfere with treatment (dysfunctional family, abusive relationship). To prevent lasting negative effects on growth and development, adolescents require inpatient medical treatment with less severe weight loss as that necessitating hospitalization in adults. Some patients requiring inpatient treatment may need 24-hour care because of their extreme medical instability (American Psychiatric Association, 2000; Becker et al., 1999; Fairburn & Harrison, 2003; Stewart & Williamson, 2004). Although body weight often is the primary admission criterion, it should not be the single determinant of inpatient treatment. Individuals with eating disorders are adept at artificially elevating their body weight via hyperhydration to avoid hospitalization, which can result in acute hyponatremia. The rate of readmission is relatively high: Approximately 20% of individuals treated in hospital settings will be admitted a second time (Stewart & Williamson, 2004).

**syncope** Temporary loss of consciousness.

### Outpatient Treatment

Outpatient therapy for eating disorders may be associated with an eating disorder treatment program or may be physician monitoring of body weight and physical health, psychotherapy, and nutritional counseling. Outpatient day care facilities are an alternative to inpatient treatment and also serve as a transition from inpatient care (American Psychiatric Association, 2000).

### Therapeutic Alliance

Successful treatment of eating disorders depends on the relationship between the treatment team and the affected individual (Stewart & Williamson, 2004), referred to as the **therapeutic alliance**. Unconditional positive regard, genuineness, warmth, and empathy foster a positive relationship. Validation of the patient's feelings and thoughts by the treatment team increases the patient's self-trust. The treatment plan always should encourage the patient's sense of responsibility and autonomy.

**therapeutic alliance** Cooperation between the patient and the therapist to engage in treatment; dependent on good rapport between patient and therapist.

### Medical Nutrition Therapy

#### Weight Gain Goal

Although low body weight is only a symptom of underlying psychopathology, it is the focus of

nutritional interventions. Because body weight often is used as the criterion for admission to or release from inpatient treatment programs and because low body weight is associated with increased morbidity and mortality, it frequently is monitored. The perceived focus on weight gain and measurement of body weight can cause significant distress for individuals with eating disorders because of their intense fear of becoming fat. In addition, patients may believe the cause of their eating disorder is going to be ignored and go untreated if they perceive the primary treatment focus is restoration of body weight. Because the eating disorder often arises out of an unmet need to be "taken seriously," patients need reassurance that they will continue to receive the psychological treatment and therapeutic support they need even if they gain weight.

To minimize the distress associated with determination of body weight, patients are often weighed with their back to the scale. It also is helpful to focus on improving overall health during refeeding and weight restoration. Monitoring clinical signs and symptoms associated with malnutrition can provide patients empirical evidence that they are becoming "healthy" rather than "fat."

Target body weight usually is determined as 92% of ideal body weight, 90% of previous highest weight, and weight at which menstruation resumes (Stewart & Williamson, 2004). Often, patients are initially not told their target body weight or the energy prescription required to achieve their goal weight.

#### Refeeding

The desired rate of weight gain is 0.5 to 1 lb per week in outpatient therapy and 2 to 3 lbs for inpatient treatment (American Dietetic Association, 2001; American Psychiatric Association, 2006). Initially, energy intake is typically 30–40 kcal/kg/day. Registered dietitians either provide a structured meal plan or help patients choose foods to ensure nutritional adequacy and consumption of all food groups. Paradoxically, initial weight gain often is difficult for patients with AN. Although resting metabolic rate is suppressed even when expressed relative to lean body mass in AN, there is evidence that patients become hypermetabolic during refeeding (de Zwaan, Aslam, & Mitchell, 2002). Up to 70 to 100 kcal/kg BW a day may be needed for individuals with AN to gain weight (American Psychiatric Association, 2006). However, because of the risk of refeeding syndrome, energy intake should be increased gradually, that is, by about 200 kcal/day for 1 week (American Dietetic Association, 2001). Refeeding syndrome is characterized by hypophosphatemia (Fisher, Simpser, & Schneider, 2000), hypomagnesemia (Birmingham, Puddicombe, & Hlynsky, 2004), hypokalemia, glucose intolerance, pancreatitis (Morris, Stephenson, Herring, & Marti, 2004), gastrointestinal dysfunction, cardiac arrhythmias, and congestive heart failure. Fluid retention and edema are common and complicate accurate determination of changes in body cell mass. The risk of refeeding syndrome is greater with enteral and parenteral tube feeding.

The goal is to first achieve an energy intake of 2,500 kcal/day for 1 week, regardless of weight changes. This is followed by an incremental increase over an additional week to 3,500 kcal/day. At this point energy intake is adjusted to achieve a rate of weight gain of at least 2 lbs per week. If the patient does not gain a minimum of 2 lbs per week after 2 weeks of inpatient therapy, bed rest or nasogastric tube feeding may be implemented (Stewart & Williamson, 2004).

#### Micronutrient Deficiencies

Dietary intake of many micronutrients is decreased in individuals with AN. Deficiencies of iron, zinc, calcium, copper, vitamin C, thiamine, riboflavin, and vitamin $B_6$ have been reported in AN (Winston, 2012). There also is evidence of poor folate and vitamin $B_{12}$ status, as evidenced by low red blood cell folate in patients with AN. Because of the increased risk of micronutrient deficiencies, AN patients should be treated with a complete micronutrient supplement. Abnormalities in carotenoid metabolism result in elevated serum carotene concentrations. To minimize loss of bone mineral, it is important to ensure that patients with eating disorders consume adequate calcium and vitamin D (American Dietetic Association, 2001).

#### Normalization of Eating and Exercise Patterns

A goal of medical nutrition therapy is to enable the patient to eat regular meals that meet nutrient needs and include a variety of foods (American Dietetic Association, 2001). In addition, individuals recovering from eating disorders need to learn to be sensitive to hunger and satiety cues. This often involves learning to "trust" one's body, that is, that eating when hungry will not lead to excessive weight gain. Individuals with eating disorders often believe their bodies are somehow defective or

different and that they will become obese if they eat like "normal people." Normalization of eating patterns often requires giving up "forbidden" foods and idiosyncratic rituals around food preparation and eating. It is unrealistic to expect an individual recovering from an eating disorder to give up all their disordered eating behaviors at once. Change seems possible if it is incremental.

### Bulimia Nervosa

Treatment goals for patients with BN include (1) reduce binge eating and purging; (2) treat physical complication of BN; (3) provide education regarding healthy nutrition and eating patterns; (4) help patient reassess and change core dysfunctional thoughts, beliefs, conflicts, and feelings related to the eating disorders; (5) treat comorbid psychiatric conditions; (6) enlist family support and provide family therapy; and (7) prevent relapse (American Psychiatric Association, 2006).

Patients with BN often present wanting to lose weight. Key to recovery from BN is the recognition that weight loss cannot be achieved by "dieting" and that dieting initiates the bing eating–purging cycle. Indiviuals recovering from BN must learn that eating regular meals will not "make them fat" but will, in fact, facilitate weight control by minimizing binge eating.

Nutritionists should assist patients with BN in structuring their eating patterns to three regular meals and one to three snacks per day. Initially, energy intake should not be determined by hunger because hunger is positively associated with binge eating and purging. Normalization of eating patterns and cessation of vomiting and laxative abuse may cause significant fluid retention and edema, which may be troubling to patients. Chronic laxative users benefit from a high-fiber diet and adequate fluid intake to minimize constipation (American Dietetic Association, 2001).

Individuals recovering from eating disorders should be educated on benefits of exercise in addition to weight control, and they should be encouraged to view exercise as pleasurable rather than compulsory. Trying new activities or sports may foster adoption of this positive view of physical activity. As with hunger and satiety, individuals with eating disorders often ignore feelings of fatigue and pain, exercising when sick or injured. They should be encouraged to have an integrated view of their bodies, rather than viewing the body as something to be mastered or controlled.

### Psychotherapy

A variety of psychological treatments has been used in the treatment of eating disorders: psychodynamic, interpersonal, cognitive behavioral (CBT), cognitive analytical, behavioral, and family psychotherapy (Fairburn & Harrison, 2003). There are very few controlled outpatient treatment studies comparing modes of psychotherapy for AN. The existing data are limited by small sample sizes, high rates of attrition, lack of standardized treatment protocols, and variation in outcome variables. In early onset AN, family therapy appears to be effective, and thus it is used in treatment of adolescents with eating disorders. There is some support for modest efficacy of psychodynamic therapy, cognitive analytical therapy, and CBT in AN (Agras et al., 2004; Becker et al., 1999).

The evidence in support of the efficacy of CBT for BN is strong. CBT is short-term (20 weekly sessions), structured, and manual-based therapy that focuses on changing dysfunctional cognitive styles that lead to disordered eating and exercise behaviors. About one-third to one-half of patients who participate in CBT experience significant and sustained improvements in bulimic behaviors. Interpersonal therapy, which focuses on problematic relationships, was shown to be as effective as CBT in two short-term trials (Agras et al., 2004; Becker et al., 1999).

### Psychotropic Medications

Antidepressants are the most common pharmacologic intervention for eating disorders (Becker et al., 1999; Fairburn & Harrison, 2003). Patients with AN and BN often experience significant depressive symptoms, and there is increased prevalence of depression in families of individuals with eating disorders. Recently, genetic linkage studies examined the association between variants of the serotonin receptor gene and AN. There is limited evidence that one variation of a serotonin receptor gene (*5-HTR2A*) is associated with AN (Kaye, Frank, Bailer, & Henry, 2005; Kaye, Frank, Bailer, Henry, & Meltzer, et al., 2005; Klump & Gobrogge, 2005). Serotonin reuptake inhibitors increase the availability of serotonin within the brain. There is no empirical evidence supporting the use of antidepressants in treatment of AN, especially during semistarvation. Serotonin synthesis is sensitive to nutritional status, rendering serotonin reuptake inhibitors ineffective in malnourished individuals. Fluoxetine may prevent relapse in weight-restored

individuals with AN (Becker et al., 1999). These antidepressants significantly improve mood and bulimic symptoms acutely, and they are effective as one component of initial treatment for BN. However, the positive effect of selective serotonin reuptake inhibitors (SSRIs) is not sustained. Furthermore, antidepressants are neither as effective as CBT nor do they consistently augment the effects of CBT.

#### Eating Disorder Not Otherwise Specified

Patients who do not meet full definitional criteria for AN or BN, yet present with eating pathology should be treated as patients who meet all of the diagnostic criteria. For patients with binge eating disorder (BED), there is strong evidence to support CBT to treat the behavioral and psychological symptoms. SSRIs appear to reduce binge eating in the short term, but do not result in significant weight loss. The appetite-suppressant sibutramine both reduces binge eating and facilitates weight loss. There is limited evidence for additional benefits of the combination of CBT and antidepressants (American Psychiatric Association, 2006).

## Special Populations

### Males

Eating disorders are less common in men than in women. It is estimated that the ratio of male to female cases of AN is 1:6 and for BN, 1:20. However, the true gender ratio is unknown because eating disorders are more likely to go undiagnosed in men than in women. Moreover, although eating disorders are more common in women, males with eating disorders are not rare; the most prevalent eating disorder among males is EDNOS. Males with eating disorders exhibit more psychiatric comorbidity and more sever psychosocial consequences than do females with eating disorders (American Psychiatric Association, 2006). Males with BN are less likely to use vomiting and laxatives as compensatory behaviors and more likely to engage in excessive exercise.

Eating disorders are more common in homosexual compared with heterosexual males because of the increased social value of thinness in the homosexual community. Except in certain subcultures (e.g., homosexual community, wrestlers, and jockeys) there is no positive reinforcement of thinness for males. In males with eating disorders, the etiology of the eating disturbance is more likely to be related to improving athletic performance, avoiding medical disease, and avoiding peer teasing. There are subtle differences in the body dissatisfaction experienced between the sexes. Women with AN are preoccupied with body weight, and men generally are dissatisfied with their body shape or size, rather than weight per se. Males with AN experience the same medical complications as females, including a decline in testosterone and loss of bone mineral density. Limited evidence has shown that treatment outcome is improved if therapy is limited to all-male patient groups and the treatment team includes some male members. Males with AN have significantly higher energy needs than their female counterparts and may require 4,000–5,000 kcal/day during weight gain. There is some evidence that testosterone treatment during refeeding may help increase muscle mass in patients with AN (American Psychiatric Association, 2006). Strength training, in combination with nutrition counseling, improves body dissatisfaction in males who are concerned with their body shape or size (Andersen, 1999).

### Pregnancy

Although fertility is reduced in women with AN, ovulation may still occur in some individuals. Women with eating disorders have increased complications during pregnancy and are more likely to have a low-birth-weight infant and to deliver prematurely (Helgstrand & Andersen, 2005; Kouba, Hallstrom, Lindholm, & Hirschberg, 2005). BN is associated with increased risk of miscarriage. Prenatal death is more common in infants born to mothers with AN (Becker et al., 1999; Fairburn & Harrison, 2003; Kouba et al., 2005). Pregnancy is a time when women are receptive to making significant lifestyle changes to increase their chances of delivering a healthy baby. Women with eating disorders experience a decrease in the severity of their illness during pregnancy. Unfortunately, the symptoms often recur after delivery when the developing infant is no longer in jeopardy (Rocco et al., 2005). Likewise, among women who were recovered from an eating disorder, the rate of relapse increased during pregnancy.

### Athletes

As discussed previously, athletes may be at an increased risk for eating disorders compared with

nonathletes. Some studies have reported increased body dissatisfaction, drive for thinness, and disordered eating in athletes compared with nonathletes. Other investigations, however, found that athletes have a more positive body image than nonathletes do and are not at greater risk for eating disorders than nonathletes are. The prevalence of eating disorders may vary with the nature of the sport; endurance and aesthetic sports have higher rates of eating disorders than do sports where body weight, composition, and appearance do not play a central role in performance (Goodman, 2005; Sanford et al., 2005).

When counseling athletes with eating disorders, it is important that the nutritionist be trained in sports nutrition. Athletes discredit diet and exercise recommendations that are not made by a professional who understands sports. Athletes often believe they are "special" and have unique nutrient requirements. This is especially important regarding exercise counseling. For example, an athlete who has been participating in rigorous training will be highly critical of any suggestion that 20 to 30 minutes of moderate exercise 5 days per week is "normal" or adequate. When the health of an athlete involved in scholastic sports is compromised, the team physician may determine that participation in organized practice and competition is contingent on maintenance of a minimal weight or percentage of body fat or on behavioral criteria. Athletes may be reluctant to recognize that they have an eating disorder and to seek help because they fear losing their scholarship and disappointing their coach and teammates. Some coaches facilitate intervention and treatment, whereas others resist, believing their authority has been overstepped. It is helpful if sports medicine professionals and coaches view eating disorders in athletes as they would any other injury or illness that requires treatment and physician release before return to practice and competition (Goodman, 2005).

### Insulin-Dependent Diabetes Mellitus

There is limited evidence that insulin-dependent diabetes mellitus increases the risk for eating disorders. It has been hypothesized that dietary interventions and weight gain associated with insulin treatment are etiologic factors (Verrotti, Catino, De Luca, Morgese, & Chiarelli, 1999). Individuals with insulin-dependent diabetes mellitus may not comply with insulin therapy to prevent weight gain, which may be considered a compensatory behavior. Poor glycemic control is associated with rapid progression of complications from diabetes and should be considered a warning sign of an eating disorder. Medical complications associated with diabetes are increased when the diabetes occurs with EDNOS or BN, and the mortality rate in individuals who suffer from both AN and insulin-dependent diabetes is significantly elevated (American Psychiatric Association, 2006).

## Childhood Eating Disorders

Disordered eating is problematic in pediatric and adolescent populations. Neither the American Psychiatric Association nor the World Health Organization provides diagnostic criteria for the classification of problematic eating in children. However, clinicians and researchers have defined several types of disordered eating in children based on psychological and behavioral characteristics (Watkins, 2002).

*Pervasive refusal syndrome* is characterized by refusal to eat, drink, talk, walk, and perform self-care (Lask, 2004). Social withdrawal and resistance to treatment are remarkable and further aggravate a serious and potentially life-threatening condition. The average age at onset is 8 to 16 years, and far more girls are affected than boys. Affected children are usually high achievers with high self-expectations, fear of failure, and difficulty dealing with failure to achieve personal standards. There is limited evidence that family violence and sexual abuse also are causal factors. The onset of pervasive refusal syndrome is usually acute. Illness and injury are the most common precipitating factors. Treatment is multidisciplinary, and hospitalization in a child psychiatric unit usually is required. The goal of medical nutrition therapy is to reverse dehydration and electrolyte imbalances and to improve energy intake and nutritional status. Nasogastric tube feedings are almost always needed; resistance to the nasogastric tube feeding is not uncommon. Because affected children are immobile, they must be moved frequently to prevent bedsores and infections. Passive stretching and hydrotherapy prevent muscle stiffness and contractions. Psychotherapy, which often involves nonverbal communication, helps patients identify and express emotions and deal with concerns about returning to normal

life. Family therapy may help resolve dysfunctional family interactions. Recovery from pervasive refusal syndrome is slow, typically requiring 1 year after identification and entering treatment, but most children have a complete recovery.

**Learning Point** The goal of medical nutrition therapy is to reverse dehydration and electrolyte imbalances and to improve energy intake and nutritional status.

Children with *food avoidance emotional disorder* present with symptoms similar to AN, depression, or anxiety disorders. The apparent lack of appetite and inadequate food intake result from negative affect; these children are not preoccupied with body weight or shape.

Children with *functional dysphagia* and *food phobias* are fearful of certain foods or textures—usually lumpy or solid foods. Their fears are rooted in beliefs that the food is poisonous or will make them gag, choke, or vomit. The origin of the phobia may be a traumatic event or a misplaced association between the food and expected negative consequence of eating the feared food.

Another type of disordered eating in children is *selective eating* or *extreme faddism*. It is normal for preschool children to go on "food jags." During this phase, children resist trying new foods and may gag if forced to eat a novel food. It is thought that children with selective eating have not progressed beyond the preschool stage characterized by food faddism. Because affected children consume their preferred foods they are usually of normal height and weight. This problem, which is most common in children ages 7 to 11 years, improves with increased social interaction.

## Summary

To support growth children and adolescents have higher energy and protein needs per kilogram of body weight than do adults. Because of this fact, nutrition during athletic performance must be properly planned and supported by diet. At puberty, androgens, estrogens, GH, and IGF-I cause increases in bone mineral, muscle mass, and sex-specific deposition of body fat. The RDA for protein is 0.95 g/kg BW for children aged 9 to 13 years and 0.85 g/kg BW for teenagers, compared with 0.8 g/kg BW for adults. More than 90% of peak bone mass is acquired by age 18 years, and bone mass doubles between the onset of puberty and young adulthood. Thus, children and adolescents require more calcium than adults do; the adequate intake for calcium is 1,300 mg/day for children ages 9 to 18 years. Iron and zinc needs also are increased during adolescence to support an increase in skeletal muscle mass and an expansion in red blood cell number.

Both the prevalence and severity of subsequent complications place type 1 and type 2 diabetes as significant diseases among the pediatric population. Whereas type 1 diabetes pathogenesis is autoimmune dysfunction that is believed to be triggered by environmental factors, type 2 diabetes focuses on obesity development. Associated with type 2 diabetes in youth is acanthosis nigricans, and in young or adolescent females, polycystic ovarian syndrome.

Treatment for both type 1 and type 2 diabetes in youth involves appropriate calories for achieving and maintaining a healthy weight while preventing obesity and allowing for growth. Adolescence can be difficult emotionally for children without a chronic disease; for those with diabetes the transition to adulthood can be especially challenging. The healthcare team needs to work with the family and child not only to achieve glycemic control but also to maintain a healthy social and psychological environment.

Eating disorders are psychiatric disorders with significant medical and psychosocial complications that affect approximately 5 million Americans. Eating disorders have been identified by the American Psychiatric Association: AN, BN, and EDNOS. Although behaviorally distinct, with AN characterized by severe dietary restriction and BN by binge eating and compensatory purging behaviors, the eating disorders share an excessive importance of body weight and shape to self-concept and self-esteem. Eating disorder diagnoses are mutually exclusive; however, an individual may meet diagnostic criteria for more than one disorder during the course of the illness. Treatment is multidisciplinary, and hospitalization in a psychiatric unit usually is required. The goal of medical nutrition therapy is to reverse dehydration and electrolyte imbalances and to improve energy intake and nutritional status.

## Case Study 1

### Pete Is a Tired Athlete

Pamela S. Hinton, PhD

Pete is a 16-year-old who plays soccer on a club team during the spring and summer. He is also a running back for his high school football team. In addition to organized practice, Pete supplements his training with running workouts and weight training twice per week. Pete tries to follow a healthful diet by limiting high-fat, high-sugar foods and regularly eating fruits and vegetables. He avoids fast food and pizza and doesn't eat much red meat. A considerable amount of his dietary protein comes from protein supplements and milk. Pete frequently takes nonsteroidal anti-inflammatory drugs for muscle soreness after hard workouts or games. For the past month, Pete has been feeling tired and doesn't seem to recover well.

#### Questions

1. What do you suspect is wrong with Pete? What tests would you perform to confirm your diagnosis?
2. What do you think is the cause of Pete's fatigue and inability to recover?
3. What are your treatment recommendations?
4. What follow-up treatment would you recommend?

## Case Study 2

### Pediatric Type 1 Diabetes

Karen Chapman-Novakofski, RD, LDN, PhD

Hannah is a 10-year-old female who has a 2-week history of polyuria, polydipsia, and a 19-lb weight loss. Her parents say that they have noticed that she looks quite thin. Approximately 4 days prior to admission, she began to have abdominal pain, nausea, and vomiting, which the family initially attributed to gastroenteritis. She has been tired for the past several days and today her parents found her lethargic but responsive. She was admitted to the hospital for diabetic ketoacidosis and diagnosed with type 1 diabetes mellitus. Basal bolus insulin therapy was initiated on admission. Nutrition education for basal bolus insulin therapy began during hospitalization.

**Diet History:** Hannah typically grazes on snacks/small meals throughout the day. Her family has dinner from 6–7 p.m. and Hannah typically takes a full hour to consume her meal. Hannah usually has a snack of cookies or graham crackers before bed. She doesn't drink milk but does have milk when she has cereal in the morning.

**Physical Activity:** Hannah goes to gymnastics three times a week for 45 minutes in the evening and 2 hours on Saturday. She has gym class one time a week at school.

**Social History:** Hannah lives with her mother, father, and 12-year-old brother. Her parents are concerned about learning how to manage Hannah's diabetes.

**Anthropometric Data:** Wt.: 27.8 kg; Ht.: 138.5 cm; BMI: 14.5

#### Questions

1. Hannah has lost weight and is quite thin. What dietary intervention is indicated for this weight loss?
2. Summarize the key learning objectives of diabetic nutrition education.
3. What special considerations need to be taken on days that Hannah has gymnastics?
4. What questions would you ask to find out more about Hannah's calcium intake? How much calcium does a girl Hannah's age need?
5. What difficulties with basal bolus insulin therapy would you expect as a result of Hannah's grazing pattern of eating? What interventions might address these concerns?

## Case Study 3

### Eating Disorders

Ellen Glovsky, PhD, RD, LDN

**Social History:** SP is a 19-year-old WF in her second year of college. She is the younger of two children, whose parents are both professionals with flourishing careers. She lives at home with her parents, who have stated clearly that they expect her to be highly successful. Her parents have placed tight restraints on her so that she can be the best at everything she attempts in her life. SP says she has very few chances to make decisions for herself and does not see this as a sign of her parents' love, but rather views it as a means of controlling her and ruining her social life. SP says she feels she must perform to receive their love and acceptance. Her parents want her to become a physician, and she is in the pre-med program, but she wants to be a personal trainer.

SP has set very high goals for herself, both socially and academically. She is devastated because she recently received a B+ in a course, tarnishing her otherwise straight-A average. She blames herself for this failure and says she is depressed. Recently, SP began talking with her friends about how she needed to lose weight and "get into shape." She began following a weight loss diet she found online and attending the gym at school every day. SP reported that if she has to skip a day, she "feels fat" and is sure she will regain all of her lost weight. She recently added bicycling everywhere she goes to burn more calories.

SP found that eating with her parents was very difficult because they would nag her about how little she ate, so she avoids eating with them. If she must eat with them, she eats a "normal" amount and later forces herself to vomit. Her main concern is getting to the bathroom in time so that very little of the food is digested.

She has begun to avoid eating with her friends as well because they are starting to comment on how little she eats and that she's getting too thin. Her two best friends had an "intervention" in which they told her how worried they are.

To "shut them up" she ate with them in the dining hall and showed them they are wrong. She then stopped at the bathroom to get rid of the food.

SP reported that her parents are very concerned that she has lost too much weight. She feels weak, is having trouble concentrating, and amenorrhea and headaches are a problem. She collapsed at school recently after standing up rapidly and was brought to the emergency room of the local hospital, where her parents were called to meet her. Her father was furious and her mother was "ashamed" at the doctor's diagnosis of anorexia nervosa. The ER recommended a psychologist and a registered dietician, but the family refused to see them. Her mother insisted they would handle things "their way."

Finally, SP had to drop out of school because she was just too weak to continue. Her father demanded that she see the recommended professionals and just "get on with it."

**Objective Data:** Before the recent "diet," SP weighed 140 lbs and is 5 foot 4 inches. She now weighs 95 lbs and is continuing to lose weight. SP weighs herself twice a day and reported that her weight "decides whether it's a good day or not."

**Diet History:** The weight loss website she has been reading said that fat provides more calories than protein or carbohydrate, so SP decided to be on a "fat-free diet." She also read that white flour and sugar and red meat are "deadly," so she avoids those foods and has become a vegetarian. She will eat fat-free dairy foods, fruits and vegetables, some whole grains and occasionally eggs. She is very worried about carbohydrates, which she knows will make her fat. SP reported that she does not like to feel full and is sure she can feel even small amounts of food "inside me" and cannot tolerate that feeling.

#### Questions

1. Determine SP's pre- and postdiet BMI, and discuss these in relation to her overall health and diagnosis of an eating disorder. What percentage of weight did she lose?
2. What aspects of this history lead to the diagnosis of anorexia nervosa? Do you see signs of another eating disorder, and why?
3. What physical, social, mental, or psychological characteristics do people with eating disorders usually have? Cover all three categories of eating disorders.
4. Which health professionals should be involved in SP's care? State the role of each.
5. What are the warning signs of anorexia nervosa? Bulimia nervosa? Binge eating disorder?
6. Describe the typical progression of treatment for a patient with anorexia nervosa and very low weight. Use your course text, this case study, and any other materials to describe treatment for each of the three types of eating disorders.
7. Prepare a care plan for this patient with three categories as discussed in class:

   **Problem:** Identify the specific problems for this patient.
   **Goal(s):** What are the goals for weight, behavior, diet, and so forth that you would set for her?
   **Approaches/Intervention:** For each problem and goal, describe how you would go about working with this patient.

## Issues to Debate

1. Should coaches and schools be responsible for the proper nutrition of their child athletes?
2. Although several lawsuits and media attention have focused on the role of the fast food industry on the increasing obesity prevalence in the United States, others have professed that individuals have a right to eat where and what they want. With a strong link between obesity and type 2 diabetes, should children of parents who are obese and have type 2 diabetes receive health warnings about places to eat or particular foods they should avoid? Should the business world, the community, the parents, or the child be responsible for eating patterns?
3. Adolescents with type 1 diabetes take insulin and monitor their blood glucose. However, it is not uncommon for them to experience some episodes of hypoglycemia. Should these young adults be allowed to take driver's education? Is having blood glucose levels checked before driving an invasion of privacy?
4. Knowing the medical consequences of eating disorders, should children be placed in a medical facility for treatment against their will?

## References


Acheson, K. J., Gremaud, G., Meirim, I., Montigon, F., Krebs, Y., Fay, L. B., … Tappy, L. (2004). Metabolic effects of caffeine in humans: Lipid oxidation or futile cycling? *American Journal of Clinical Nutrition, 79*, 40–46.

Agras, W. S., Brandt, H. A., Bulik, C. M., Dolan-Sewell, R., Fairburn, C. G., Halmi, K. A., … Wilfley, D. E. (2004). Report of the National Institutes of Health workshop on overcoming barriers to treatment research in anorexia nervosa. *International Journal of Eating Disorders, 35*, 509–521.

Ainsworth, B. E., Haskell, W. L., Herrmann, S. D., Meckes, N., Bassett, D. R., Jr., Tudor-Locke, C., … Leon, A. S. (2011). 2011 Compendium of Physical Activities: A second update of codes and MET values. *Medicine and Science in Sports and Exercise*, *43*, 1575–1581.

Ainsworth, B. E., Jacobs, D. R., Jr., Leon, A. S., Richardson, M. T., & Montoye, H. J. (1993). Assessment of the accuracy of physical activity questionnaire occupational data. *Journal of Occupational Medicine, 35*, 1017–1027.

American Academy of Pediatrics. (2005). Breastfeeding and the use of human milk. *Pediatrics, 115*, 496–450.

American College of Obstetricians and Gynecologists. (2002). ACOG committee opinion. Exercise during pregnancy and the postpartum period. *International Journal of Gynaecology and Obstetrics, 77*, 79–81.

American Diabetes Association. (1999). Diabetes mellitus and exercise. *Diabetes Care*, S49–S53.

American Diabetes Association. (2013). Standards of medical care in diabetes. *Diabetes Care*, *36*, S11-66.

American Dietetic Association. (2001). Position paper: Nutrition intervention in the treatment of anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified (EDNOS). *Journal of the American Dietetic Association, 101*, 810–819.

American Dietetic Association, Dietitians of Canada, & American College of Sports Medicine. (2000). Position paper: Nutrition and athletic performance. *Journal of the American Dietetic Association, 100*, 1543–1556.

American Dietetic Association. (2006). Position of the American Dietetic Association: Individual-, family-, school-, and community-based interventions for pediatric overweight. *Journal of the American Dietetic Association, 1*(6), 925–945.

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). Washington, DC: Author.

American Psychiatric Association. (2006). Treatment of patients with eating disorders, third edition. American Psychiatric Association. *American Journal of Psychiatry*, *163*, 4–54.

Andersen, A. E. (1999). Gender-related aspects of eating disorders: A guide to practice. *Journal of Gender Specific Medicine, 2*, 47–54.

Anderson, M. J., Cotter, J. D., Garnham, A. P., Casley, D. J., & Febbraio, M. A. (2001). Effect of glycerol-induced hyperhydration on thermoregulation and metabolism during exercise in heat. *International Journal of Sport Nutrition and Exercise Metabolism, 11*, 315–333. Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry*, *68*, 724–731.

Arcelus, J., Mitchell, A., Wales, J. & Nielsen, S. (2011). Mortality rayes in patients with anorexia nervosa and other eating disorders: A meta-anaylsis of 36 studies. *Arch Gen Psychiatry, 68*(11), 724–731.

Atherton, P. J., Babraj, J., Smith, K., Singh, J., Rennie, M. J., & Wackerhage, H. (2005). Selective activation of AMPK-PGC-1alpha or PKB-TSC2-mTOR signaling can explain specific adaptive responses to endurance or resistance training-like electrical muscle stimulation. *FASEB Journal*, *19*, 786–788.

Artz, E., Haqq, A., & Freemark, M. (2005). Hormonal and metabolic consequences of childhood obesity. *Endocrinology and Metabolism Clinics of North America, 34*, 643–658.

Artz, E., & Freemark, M. (2004). The pathogenesis of insulin resistance in children: Metabolic complications and the roles of diet, exercise and pharmacotherapy in the prevention of type 2 diabetes. *Pediatric Endocrinology Reviews, 1*, 296–309.

Bar-Or, O. (2001). Nutritional considerations for the child athlete. *Canadian Journal of Applied Physiology*, *26*(Suppl.), S186–S191.

Badaru, A., & Pihoker, C. (2012). Type 2 diabetes in childhood: Clinical characteristics and role of β-cell autoimmunity. *Current Diabetes Reports*, *12*, 75–81.

Barry, D. W., & Kohrt, W. M. (2008). BMD decreases over the course of a year in competitive male cyclists. *Journal of Bone and Mineral Research*, 23, 484–491.

Becker, A. E., Grinspoon, S. K., Klibanski, A., & Herzog, D. B. (1999). Eating disorders. *New England Journal of Medicine, 340*, 1092–1098.

Bending, D., Zaccone, P., & Cooke, A. (2012). Inflammation and type one diabetes. *International Immunology*, *24*, 339–346.

Bennell, K., White, S., & Crossley, K. (1999). The oral contraceptive pill: A revolution for sportswomen? *British Journal of Sports Medicine*, *33*, 231–238.

Berry, D., Urban, A., & Grey, M. (2006). Management of type 2 diabetes in youth (part 2). *Journal of Pediatric Health Care, 20*, 88–97.

Birmingham, C. L., Puddicombe, D., & Hlynsky, J. (2004). Hypomagnesemia during refeeding in anorexia nervosa. *Eating and Weight Disorders*, *9*, 236–237.

Bonen, A., Haynes, F. W., & Graham, T. E. (1991). Substrate and hormonal responses to exercise in women using oral contraceptives. *Journal of Applied Physiology*, *70*, 1917–1927.

Borsheim, E., Aarsland, A., & Wolfe, R. R. (2004). Effect of an amino acid, protein, and carbohydrate mixture on net muscle protein balance after resistance exercise. *International Journal of Sport Nutrition and Exercise Metabolism, 14*, 255–271.

Borsheim, E., Cree, M. G., Tipton, K. D., Elliott, T. A., Aarsland, A., & Wolfe, R. R. (2004). Effect of carbohydrate intake on net muscle protein synthesis during recovery from resistance exercise. *Journal of Applied Physiology, 96*, 674–678.

Borsheim, E., Tipton, K. D., Wolf, S. E., & Wolfe, R. R. (2002). Essential amino acids and muscle protein recovery from resistance exercise. *American Journal of Physiology. Endocrinology and Metabolism, 283*, E648–E657.

Bremer, A. A. (2010). Polycystic ovary syndrome in the pediatric population. *Metabolic Syndrome and Related Disorders*, *8*, 375–394.

Brewer, M., Pawelczak, M., Kessler, M., & Shah, B. (2010). A review of polycystic ovarian syndrome in adolescents. *Minerva Pediatrics*, *62*, 459–473.

Brickman, W. J., Huang, J., Silverman, B. L., & Metzger, B. E. (2010). Acanthosis nigricans identifies youth at high risk for metabolic abnormalities. *Journal of Pediatrics*, *156*, 87–92.

Brownlie, T., Utermohlen, V., Hinton, P. S., Giordano, C., & Haas, J. D. (2002). Marginal iron deficiency without anemia impairs aerobic adaptation among previously untrained women. *American Journal of Clinical Nutrition, 75*, 734–742.

Brownlie, T., Utermohlen, V., Hinton, P. S., & Haas, J. D. (2004). Tissue iron deficiency without anemia impairs adaptation in endurance capacity after aerobic training in previously untrained women. *American Journal of Clinical Nutrition, 79*, 437–443.

Brutsaert, T. D., Hernandez-Cordero, S., Rivera, J., Viola, T., Hughes, G., & Haas, J. D. (2003). Iron supplementation improves progressive fatigue resistance during dynamic knee extensor exercise in iron-depleted, nonanemic women. *American Journal of Clinical Nutrition, 77*, 441–448.

Bryner, R. W., Toffle, R. C., Ullrich, I. H., & Yeater, R. A. (1996). Effect of low dose oral contraceptives on exercise performance. *British Journal of Sports Medicine, 30*, 36–40.

Burke, L. M., Collier, G. R., Broad, E. M., Davis, P. G., Martin, D. T., Sanigorski, A. J., & Hargreaves, M. (2003). Effect of alcohol intake on muscle glycogen storage after prolonged exercise. *Journal of Applied Physiology, 95*, 983–990.

Cannell, J. J., Hollis, B. W., Sorenson, M. B., Taft, T. N., & Anderson, J. J. (2009). Athletic performance and vitamin D. *Medicine and Science in Sports and Exercise*, *41*, 1102–1110.

Casazza, G. A., Suh, S. H., Miller, B. F., Navazio, F. M., & Brooks, G. A. (2002). Effects of oral contraceptives on peak exercise capacity. *Journal of Applied Physiology, 93*, 1698–1702.

Castell, L. (2003). Glutamine supplementation in vitro and in vivo, in exercise and in immunodepression. *Sports Medicine, 33*, 323–345.

Celsing, F., Ekblom, B., Sylven, C., Everett, J., & Astrand, P. O. (1988). Effects of chronic iron deficiency anaemia on myoglobin content, enzyme activity, and capillary density in the human skeletal muscle. *Acta Medica Scandinavica, 223*, 451–457.

Centers for Disease Control and Prevention. (n.d.). National Center for Chronic Disease Prevention and Health Promotion. Healthy youth! Physical activity, promoting better health. Retrieved from www.cdc.gov/healthyyouth/physicalactivity/promoting_health

Christen, U., Bender, C., & von Herrath, M. G. (2012). Infection as a cause of type 1 diabetes? *Current Opinions in Rheumatology*, *24*, 417–423.

Clapp, J. F., 3rd. (2000). Exercise during pregnancy. A clinical update. *Clinical Sports Medicine, 19*, 273–286.

Clemens, R. A. (2011). Milk A1 and A2 peptides and diabetes. *Nestle Nutrition Workshop Series Pediatrics Program*, *67*,187–195.

Concannon, P., Rich, S. S., & Nepom, G. T. (2009). Genetics of type 1A_diabetes. *New England Journal of Medicine*, *360*, 1646–1654.

Connor, E. L. (2012). Adolescent polycystic ovary syndrome. *Adolescent Medicine State of the Art Reviews*, *23*,164–177.

Connor, W. E. (2000). Importance of n-3 fatty acids in health and disease. *American Journal of Clinical Nutrition, 71* (1 Suppl.), 171S–175S.

Convertino, V. A., Armstrong, L. E., Coyle, E. F., Mack, G. W., Sawka, M. N., Senay, L. C., Jr., … Sherman, W. M. (1996). American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine and Science in Sports and Exercise, 28*, i–vii.

Copeland, K., Pankratz, K., Cathey, V., Immohotichey, P., Maddox, J., Felton, B., … Blackett, P. (2006). Acanthosis nigricans, insulin resistance (HOMA) and dyslipidemia among Native American children. *Journal of the Oklahoma State Medical Association, 99*, 19–24.

Copeland, K. C., Zeitler, P., Geffner, M., Guandalini, C., Higgins, J., Hirst, K., … TODAY Study Group. (2011). Characteristics of adolescents and youth with recent-onset type 2 diabetes: The TODAY cohort at baseline. *Journal of Clinical Endocrinology and Metabolism*, *96*, 159–167.

Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R., & Park, S. H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine and Science in Sports and Exercise, 20*, 249–254.

Couture, S., Massicotte, D., Lavoie, C., Hillaire-Marcel, C., & Peronnet, F. (2002). Oral [(13)C] glucose and endogenous energy substrate oxidation during prolonged treadmill running. *Journal of Applied Physiology, 92*, 1255–1260.

Cruz, M. L., Shaibi, G. Q., Weigensberg, M. J., Spruijt-Metz, D., Ball, G. D., & Goran, M. I. (2005). Pediatric obesity and insulin resistance: Chronic disease risk and implications for treatment and prevention beyond body weight modification. *Annual Reviews of Nutrition, 25*, 435–468.

D'Adamo, E., & Caprio, S. (2011). Type 2 diabetes in youth: epidemiology and pathophysiology. *Diabetes Care*, *34*, S161–S165.

Davies, K. J., Donovan, C. M., Refino, C. J., Brooks, G. A., Packer, L., & Dallman, P. R. (1984). Distinguishing effects of anemia and muscle iron deficiency on exercise bioenergetics in the rat. *American Journal of Physiology, 246*(6 Pt. 1), E535–E543.

Deldicque, L., Theisen, D., & Francaux, M. (2005). Regulation of mTOR by amino acids and resistance exercise in skeletal muscle. *European Journal of Applied Physiology, 94*, 1–10.

De Souza, M. J., Leidy, H. J., O'Donnell, E., Lasley, B., & Williams, N. I. (2004). Fasting ghrelin levels in physically active women: Relationship with menstrual disturbances and metabolic hormones. *Journal of Clinical Endocrinology and Metabolism, 89*, 3536–3542.

de Zwaan, M., Aslam, Z., & Mitchell, J. E. (2002). Research on energy expenditure in individuals with eating disorders: A review. *International Journal of Eating Disorders, 32*, 127–134.

Diabetes Research in Children Network Study Group. (2006). Prevention of hypoglycemia during exercise in children with type 1 diabetes by suspending basal insulin. *Diabetes Care, 29*(10), 2200 –2204.

Fagot-Campagna, A., Perritt, D. J., Engelgau, M. M., Burrows, N. R., Geiss, L. S., Valdez, R., … Narayan, K. M. (2000). Type 2 diabetes among North American children and adolescents: An epidemiologic review and a public health perspective. *Journal of Pediatrics, 136*, 664–672.

Fairburn, C. G., & Bohn, K. (2005). Eating disorder NOS (EDNOS): An example of the troublesome "not otherwise specified" (NOS) category in *DSM-IV*. *Behaviour Research and Therapy, 43*, 691–701.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *Lancet, 361*, 407–416.

Finch, C. A., Miller, L. R., Inamdar, A. R., Person, R., Seiler, K., & Mackler, B. (1976). Iron deficiency in the rat. Physiological and biochemical studies of muscle dysfunction. *Journal of Clinical Investigations, 58*, 447–453.

Fisher, M., Simpser, E., & Schneider, M. (2000). Hypophosphatemia secondary to oral refeeding in anorexia nervosa. *International Journal of Eating Disorders, 28*, 181–187.

Flint, A. & Arslanian, S. (2011). Treatment of type 2 diabetes in youth. *Diabetes Care, 34*(Supp 2), S177–S183.

Foster-Powell, K., Holt, S. H., & Brand-Miller, J. C. (2002). International table of glycemic index and glycemic load values: 2002. *American Journal of Clinical Nutrition, 76*, 5–56.

Goodman, L. R. (2005). The female athlete and menstrual function. *Current Opinion in Obstetrics and Gynecology, 17*, 466–470.

Girgis, C. M., Clifton-Bligh, R. J., Hamrick, M. W., Holick, M. F., & Gunton, J. E. (2012). The roles of vitamin D in skeletal muscle: Form, function, and metabolism. *Endocrine Reviews*, *34*(1), 33–83.

Grinspoon, S. K., Friedman, A. J., Miller, K. K., Lippman, J., Olson, W. H., & Warren, M. P. (2003). Effects of a triphasic combination oral contraceptive containing norgestimate/ethinyl estradiol on biochemical markers of bone metabolism in young women with osteopenia secondary to hypothalamic amenorrhea. *Journal of Clinical Endocrinology and Metabolism, 88*, 3651–3656.

Grinspoon, S., Miller, K., Herzog, D., Clemmons, D., & Klibanski, A. (2003). Effects of recombinant human insulin-like growth factor (IGF)-I and estrogen administration on IGF-I, IGF binding protein (IGFBP)-2, and IGFBP-3 in anorexia nervosa: A randomized-controlled study. *Journal of Clinical Endocrinology and Metabolism, 88*, 1142–1149.

Grinspoon, S., Miller, K. K., Herzog, D. B., Grieco, K. A., & Klibanski, A. (2004). Effects of estrogen and recombinant human insulin-like growth factor-I on ghrelin secretion in severe undernutrition. *Journal of Clinical Endocrinology and Metabolism, 89*, 3988–3993.

Guzmán, J., Esteve, H., Pablos, C., Pablos, A., Blasco, C., & Villegas, J. (2011). DHA- rich fish oil improves complex reaction time in female elite soccer players. *Journal of Sports Science & Medicine, 10*(2), 301–305.

Haller, M. J., Atkinson, M. A., & Schatz, D. (2005). Type 1 diabetes mellitus: Etiology, presentation, and management. *Pediatric Clinics of North America, 52*, 1553–1578.

Halliday, T. M., Peterson, N. J., Thomas, J. J., Kleppinger, K., Hollis, B. W., & Larson-Meyer, D. E. (2011). Vitamin D status relative to diet, lifestyle, injury, and illness in college athletes. *Medicine and Science in Sports and Exercise*, *43*, 335–343.

Halson, S. L., Lancaster, G. I., Jeukendrup, A. E., & Gleeson, M. (2003). Immunological responses to overreaching in cyclists. *Medicine and Science in Sports and Exercise, 35*, 854–861.

Hamalainen, A. M., & Knip, M. (2002). Autoimmunity and familial risk of type I diabetes. *Current Diabetes Reports, 2*, 347–353.

Hamilton, B. (2010). Vitamin D and human skeletal muscle. *Scandinavian Journal of Medicine and Science in Sports*, *20*, 182–190.

Heaney, R. P., & Rafferty, K. (2001). Carbonated beverages and urinary calcium excretion. *American Journal of Clinical Nutrition, 74*, 343–347.

Heer, M., Mika, C., Grzella, I., Heussen, N., & Herpertz-Dahlmann, B. (2004). Bone turnover during inpatient nutritional therapy and outpatient follow-up in patients with anorexia nervosa compared with that in healthy control subjects. *American Journal of Clinical Nutrition, 80*, 774–781.

Helgstrand, S., & Andersen, A. M. (2005). Maternal underweight and the risk of spontaneous abortion. *Acta Obstetricia Et Gynecologica Scandinavica, 84*, 1197–1201.

Henig, R. M. (2004, November 30). Sorry. Your eating disorder doesn't meet our criteria. *New York Times*. Retrieved from www.nytimes.com/2004/11/30/health/psychology/30eat.html

Hermanns-Lê, T., Scheen, A., & Pierard, G. E. (2004). Acanthosis nigricans associated with insulin resistance: Pathophysiology and management. *American Journal of Clinical Dermatology, 5*, 199–203.

Hildebrandt, A. L., Pilegaard, H., & Neufer, P. D. (2003). Differential transcriptional activation of select metabolic genes in response to variations in exercise intensity and duration. *American Journal of Physiology, Endocrinology and Metabolism, 285*, E1021–E1027.

Hinton, P. S., Giordano, C., Brownlie, T., & Haas, J. D. (2000). Iron supplementation improves endurance after training in iron-depleted, nonanemic women. *Journal of Applied Physiology, 88*, 1103–1111.

Hummel, S., Pflüger, M., Hummel, M., Bonifacio, E., & Ziegler, A. G. (2011). Primary dietary intervention study to reduce the risk of islet autoimmunity in children at increased risk for type 1 diabetes: The BABYDIET study. *Diabetes Care*, *34*, 1301–1305.

Hummel, S., & Ziegler, A. G. (2011). Early determinants of type 1 diabetes: Experience from the BABYDIAB and BABYDIET studies. *American Journal of Clinical Nutrition*, *94*, 1821S–1823S.

Ihle, R., & Loucks, A. B. (2004). Dose-response relationships between energy availability and bone turnover in young exercising women. *Journal of Bone and Mineral Research*, *19*, 1231–1240.

Imperatore, G., Boyle, J. P., Thompson, T. J., Case, D., Dabelea, D., Hamman, R. F., … Standiford, D., for the SEARCH for Diabetes in Youth Study Group. (2012). Projections of type 1 and type 2 diabetes burden in the U.S. population aged < 20 years through 2050: Dynamic modeling of incidence, mortality, and population growth. *Diabetes Care*, *35*, 2515–2520.

Institute of Medicine, Food and Nutrition Board. (1997). *Dietary Reference Intakes for calcium, phosphorous, magnesium, vitamin D, and fluoride*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2001). *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, nickel, silicon, vanadium, and zinc*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2003). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, protein and amino acids*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for water, potassium, sodium, chloride, and sulfate*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Washington, DC: National Academies Press.

Institute of Medicine, Food and Nutrition Board. (2010). *Dietary Reference Intakes for calcium and vitamin D*. Washington, DC: National Academies Press.

Ivy, J. L., Goforth, H. W., Jr., Damon, B. M., McCauley, T. R., Parsons, E. C., & Price, T. B. (2002). Early postexercise muscle

glycogen recovery is enhanced with a carbohydrate-protein supplement. *Journal of Applied Physiology, 93*, 1337–1344.

Ivy, J. L., Res, P. T., Sprague, R. C., & Widzer, M. O. (2003). Effect of a carbohydrate–protein supplement on endurance performance during exercise of varying intensity. *International Journal of Sport Nutrition and Exercise Metabolism, 13*, 382–395.

James, M. J., Gibson, R. A., & Cleland, L. G. (2000). Dietary polyunsaturated fatty acids and inflammatory mediator production. *American Journal of Clinical Nutrition, 71*(1 Suppl.), 343S–348S.

Jankowski, C. M., Ben-Ezra, V., Gozansky, W. S., & Scheaffer, S. E. (2004). Effects of oral contraceptives on glucoregulatory responses to exercise. *Metabolism, 53*, 348–352.

Jentjens, R. L., Achten, J., & Jeukendrup, A. E. (2004). High oxidation rates from combined carbohydrates ingested during exercise. *Medicine and Science in Sports and Exercise, 36*, 1551–1558.

Jentjens, R. L., Moseley, L., Waring, R. H., Harding, L. K., & Jeukendrup, A. E. (2004). Oxidation of combined ingestion of glucose and fructose during exercise. *Journal of Applied Physiology, 96*, 1277–1284.

Jentjens, R. L., van Loon, L. J., Mann, C. H., Wagenmakers, A. J., & Jeukendrup, A. E. (2001). Addition of protein and amino acids to carbohydrates does not enhance postexercise muscle glycogen synthesis. *Journal of Applied Physiology, 91*, 839–846.

Jentjens, R. L., Venables, M. C., & Jeukendrup, A. E. (2004). Oxidation of exogenous glucose, sucrose, and maltose during prolonged cycling exercise. *Journal of Applied Physiology, 96*, 1285–1291.

Jeukendrup, A. E., & Martin, J. (2001). Improving cycling performance: How should we spend our time and money? *Sports Medicine, 31*, 559–569.

Johnson, N. A., Stannard, S. R., Mehalski, K., Trenell, M. I., Sachinwalla, T., Thompson, C. H., & Thompson, M. W. (2003). Intramyocellular triacylglycerol in prolonged cycling with high- and low-carbohydrate availability. *Journal of Applied Physiology, 94*, 1365–1372.

Jones, A. M., Carter, H., Pringle, J. S., & Campbell, I. T. (2002). Effect of creatine supplementation on oxygen uptake kinetics during submaximal cycle exercise. *Journal of Applied Physiology, 92*, 2571–2577.

Kaye, W. H., Frank, G. K., Bailer, U. F., & Henry, S. E. (2005). Neurobiology of anorexia nervosa: Clinical implications of alterations of the function of serotonin and other neuronal systems. *International Journal of Eating Disorders, 37* (Suppl.), S15–S19; discussion S20–S11.

Kaye, W. H., Frank, G. K., Bailer, U. F., Henry, S. E., Meltzer, C. C., Price, J. C., … Wagner, A. (2005). Serotonin alterations in anorexia and bulimia nervosa: New insights from imaging studies. *Physiology and Behavior, 85*, 73–81.

Keen, A. D., & Drinkwater, B. L. (1997). Irreversible bone loss in former amenorrheic athletes. *Osteoporosis International, 7*, 311–315.

Keesey, R. E., & Corbett, S. W. (1984). Metabolic defense of the body weight set-point. *Association for Research in Nervous and Mental Disease, 62*, 87–96.

Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The biology of human starvation I-II*. Minneapolis: University of Minnesota Press.

Kitagawa, T., Owada, M., Urakami, T., & Yamauchi, K. (1998). Increased incidence of non-insulin dependent diabetes mellitus among Japanese schoolchildren correlates with an increased intake of animal protein and fat. *Clinical Pediatrics, 27*, 111–115.

Klump, K. L., & Gobrogge, K. L. (2005). A review and primer of molecular genetic studies of anorexia nervosa. *International Journal of Eating Disorders, 37* (Suppl.), S43–S48; discussion S87–S49.

Kordonouri, O., Hartman, R. & Danne, T. (2011). Treatment of type 1 diabetes in children and adolescents using modern insulin pumps. *Diabetes Research and Clinical Practice, 93*(Supp 1), S118–24.

Kouba, S., Hallstrom, T., Lindholm, C., & Hirschberg, A. L. (2005). Pregnancy and neonatal outcomes in women with eating disorders. *Obstetrics and Gynecology, 105*, 255–260.

Krieger, J. W., Crowe, M., & Blank, S. E. (2004). Chronic glutamine supplementation increases nasal but not salivary IgA during 9 days of interval training. *Journal of Applied Physiology, 97*, 585–591.

Krzywkowski, K., Petersen, E. W., Ostrowski, K., Kristensen, J. H., Boza, J., & Pedersen, B. K. (2001). Effect of glutamine supplementation on exercise-induced changes in lymphocyte function. *American Journal of Physiology—Cell Physiology, 281*, C1259–C1265.

Krzywkowski, K., Petersen, E. W., Ostrowski, K., Link-Amster, H., Boza, J., Halkjaer-Kristensen, J., & Pedersen, B. K. (2001). Effect of glutamine and protein supplementation on exercise-induced decreases in salivary IgA. *Journal of Applied Physiology, 91*, 832–838.

Lambert, G. P., Bleiler, T. L., Chang, R. T., Johnson, A. K., & Gisolfi, C. V. (1993). Effects of carbonated and noncarbonated beverages at specific intervals during treadmill running in the heat. *International Journal of Sport Nutrition, 3*, 177–193.

Larson-Meyer, D. E. (2002). Effect of postpartum exercise on mothers and their offspring: A review of the literature. *Obesity Research, 10*, 841–853.

Lask, B. (2004). Pervasive refusal syndrome. *Advances in Psychiatric Treatment, 10*, 153–159.

Laughlin, G. A., & Yen, S. S. (1996). Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. *Journal of Clinical Endocrinology and Metabolism, 81*, 4301–4309.

Leachman Slawson, D., McClanahan, B. S., Clemens, L. H., Ward, K. D., Klesges, R. C., Vukadinovich, C. M., & Cantler, E. D. (2001). Food sources of calcium in a sample of African-American and Euro-American collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism, 11*, 199–208.

Lempainen, J., Tauriainen, S., Vaarala, O., Mäkelä, M., Honkanen, H., Marttila, J., … Ilonen, J. (2012). Interaction of enterovirus infection and cow's milk–based formula nutrition in type 1 diabetes-associated autoimmunity. *Diabetes Metabolism Research and Reviews*, *28*, 177–185.

Levy-Marchal, C., Arslanian, S., Cutfield, W., Sinaiko, A., Druet, C., Marcovecchio, M. L.,… Insulin Resistance in Children Consensus Conference Group. (2010). Insulin resistance in children: Consensus, perspective, and future directions. *Journal of Clinical Endocrinology and Metabolism*, *95*, 5189–5198.

Looker, A. C., Dallman, P. R., Carroll, M. D., Gunter, E. W., & Johnson, C. L. (1997). Prevalence of iron deficiency in the United States. *Journal of the American Medical Association, 277*, 973–976.

Loucks, A. B. (2004). Energy balance and body composition in sports and exercise. *Journal of Sports Science, 22*, 1–14.

Loucks, A. B., Laughlin, G. A., Mortola, J. F., Girton, L., Nelson, J. C., & Yen, S. S. (1992). Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. *Journal of Clinical Endocrinology and Metabolism, 75*, 514–518.

Loucks, A. B., & Verdun, M. (1998). Slow restoration of LH pulsatility by refeeding in energetically disrupted women. *American Journal of Physiology, 275*(4 Pt. 2), R1218–R1226.

Loud, K. J., Gordon, C. M., Micheli, L. J., & Field, A. E. (2005). Correlates of stress fractures among preadolescent and adolescent girls. *Pediatrics, 115*, e399–e406.

Lovallo, W. R., Wilson, M. F., Vincent, A. S., Sung, B. H., McKey, B. S., & Whitsett, T. L. (2004). Blood pressure response to caffeine shows incomplete tolerance after short-term regular consumption. *Hypertension, 43*, 760–765.

Marino, F. E., Kay, D., & Cannon, J. (2003). Glycerol hyperhydration fails to improve endurance performance and thermoregulation in humans in a warm humid environment. *Pflügers Archiv—European Journal of Physiology, 446*, 455–462.

Markou, K. B., Mylonas, P., Theodoropoulou, A., Kontogiannis, A., Leglise, M., Vagenakis, A. G., & Georgopoulos, N. A. (2004). The influence of intensive physical exercise on bone acquisition in adolescent elite female and male artistic gymnasts. *Journal of Clinical Endocrinology and Metabolism, 89*, 4383–4387.

McCargar, L. J., & Crawford, S. M. (1992). Metabolic and anthropometric changes with weight cycling in wrestlers. *Medicine and Science in Sports and Exercise, 24*, 1270–1275.

Melby, C. L., Schmidt, W. D., & Corrigan, D. (1990). Resting metabolic rate in weight-cycling collegiate wrestlers compared with physically active, noncycling control subjects. *American Journal of Clinical Nutrition, 52*, 409–414.

Miller, K. K., Grieco, K. A., Mulder, J., Grinspoon, S., Mickley, D., Yehezkel, R., … Klibanski, A. (2004). Effects of risedronate on bone density in anorexia nervosa. *Journal of Clinical Endocrinology and Metabolism, 89*, 3903–3906.

Miller, K. K. (2011). Endocrine dysregulation in anorexia nervosa update. *Journal of Clinical Endocrinology and Metabolism*, *96*, 2939–2949.

Miyauchi, T., Maeda, S., Iemitsu, M., Kobayashi, T., Kumagai, Y., Yamaguchi, I., & Matsuda, M. (2003). Exercise causes a tissue-specific change of NO production in the kidney and lung. *Journal of Applied Physiology, 94*, 60–68.

Mizokami-Stout, K., Cree-Green, M., & Nadeau, K. J. (2012). Insulin resistance in type 2 diabetic youth. *Current Opinions in Endocrinology, Diabetes, and Obesity, 19*, 255–262.

Morgan, A. R. (2012). Determining genetic risk factors for pediatric type 2 diabetes. *Current Diabetes Reports*, *12*, 88–92.

Morris, L. G., Stephenson, K. E., Herring, S., & Marti, J. L. (2004). Recurrent acute pancreatitis in anorexia and bulimia. *Journal of the Pancreas*, *5*, 231–234.

Murphy, S. P., & Johnson, R. K. (2003). The scientific basis of recent US guidance on sugars intake. *American Journal of Clinical Nutrition, 78*, 827S–833S.

Otis, C. L., Drinkwater, B., Johnson, M., Loucks, A., & Wilmore, J. (1997). American College of Sports Medicine position stand. The female athlete triad. *Medicine and Science in Sports and Exercise, 29*, i–ix.

Parcell, A. C., Smith, J. M., Schulthies, S. S., Myrer, J. W., & Fellingham, G. (2004). *Cordyceps sinensis* (CordyMax Cs-4) supplementation does not improve endurance exercise performance. *International Journal of Sport Nutrition and Exercise Metabolism, 14*, 236–242.

Park, Y., & Harris, W. S. (2003). Omega-3 fatty acid supplementation accelerates chylomicron triglyceride clearance. *Journal of Lipid Research, 44*, 455–463.

Patelarou, E., Girvalaki, C., Brokalaki, H., Patelarou, A., Androulaki, Z., & Vardavas, C. (2012). Current evidence on the associations of breastfeeding, infant formula, and cow's milk introduction with type 1 diabetes mellitus: A systematic review. *Nutrition Reviews*, *70*, 509–519.

Peeling, P., Dawson, B., Goodman, C., Landers, G. & Trinder, D. (2008). Athletic induced iron deficiency: New insights into the role of inflammation, cytokines and hormones. *European Journal of Applied Physiology*, *103*, 381–391.

Peeling, P., Dawson, B., Goodman, C., Landers, G., Wiegerinck, E. T., Swinkels, D. W., & Trinder, D. (2009a). Cumulative effects of consecutive running sessions on hemolysis, inflammation and hepcidin activity. *European Journal of Applied Physiology*, *106*, 51–59.

Peeling, P., Dawson, B., Goodman, C., Landers, G., Wiegerinck, E. T., Swinkels, D.W., & Trinder, D. (2009b). Effects of exercise on hepcidin response and iron metabolism during recovery. *International Journal of Sport Nutrition and Exercise Metabolism*, *19*, 583–597.

Peeling, P., & Landers, G. (2009). Swimming intensity during triathlon: A review of current research and strategies to enhance race performance. *Journal of Sports Sciences*, *27*, 1079–1085.

Perez, A. J. (2008, September 22). Dietary supplements cause 600 "adverse events." *USA Today*. Retrieved from http://usatoday30.usatoday.com/news/health/2008-09-22-supplements-adverse-events_N.htm

Petersmarck, K. A., Teitelbaum, H. S., Bond, J. T., Bianchi, L., Hoerr, S. M., & Sowers, M. F. (1999). The effect of weight cycling on blood lipids and blood pressure in the Multiple Risk Factor Intervention Trial Special Intervention Group. *International Journal of Obesity and Related Metabolic Disorders, 23*, 1246–1255.

Petibois, C., Cassaigne, A., Gin, H., & Deleris, G. (2004). Lipid profile disorders induced by long-term cessation of physical activity in previously highly endurance-trained subjects. *Journal of Clinical Endocrinology and Metabolism, 89*, 3377–3384.

Phillips, S. M., & Van Loon, L. J. (2011). Dietary protein for athletes: From requirements to optimum adaptation. *Journal of Sports Sciences*, *29*(Suppl. 1), S29–38.

Pollock, M. L., Franklin, B. A., Balady, G. J., Chaitman, B. L., Fleg, J. L., Fletcher, B., … Bazzarre, T. (2000). Resistance exercise in individuals with and without cardiovascular disease: Benefits, rationale, safety, and prescription. An advisory from the Committee on Exercise, Rehabilitation,

and Prevention, Council on Clinical Cardiology, and American Heart Association. Position paper endorsed by the American College of Sports Medicine. *Circulation, 101*, 828–833.

Pozzilli, P., Guglielmi, C., Caprio, S., & Buzzetti, R. (2011). Obesity, autoimmunity, and double diabetes in youth. *Diabetes Care*, *34*, S166–170.

Rackow, B. W. (2012). Polycystic ovary syndrome in adolescents. *Current Opinions in Obstetrics and Gynecology*, *24*, 281–287.

Rasmussen, B. B., Hancock, C. R., & Winder, W. W. (1998). Postexercise recovery of skeletal muscle malonyl-CoA, acetyl-CoA carboxylase, and AMP-activated protein kinase. *Journal of Applied Physiology, 85*, 1629–1634.

Reaven, G. M. (2005). The insulin resistance syndrome: Definition and dietary approaches to treatment. *Annual Reviews of Nutrition*, *25*, 391–406.

Rickenlund, A., Carlstrom, K., Ekblom, B., Brismar, T. B., Von Schoultz, B., & Hirschberg, A. L. (2004). Effects of oral contraceptives on body composition and physical performance in female athletes. *Journal of Clinical Endocrinology and Metabolism, 89*, 4364–4370.

Rickenlund, A., Thoren, M., Carlstrom, K., von Schoultz, B., & Hirschberg, A. L. (2004). Diurnal profiles of testosterone and pituitary hormones suggest different mechanisms for menstrual disturbances in endurance athletes. *Journal of Clinical Endocrinology and Metabolism, 89*, 702–707.

Rivera-Brown, A. M., Gutierrez, R., Gutierrez, J. C., Frontera, W. R., & Bar-Or, O. (1999). Drink composition, voluntary drinking, and fluid balance in exercising, trained, heat-acclimatized boys. *Journal of Applied Physiology, 86*, 78–84.

Rocco, P. L., Orbitello, B., Perini, L., Pera, V., Ciano, R. P., & Balestrieri, M. (2005). Effects of pregnancy on eating attitudes and disorders: A prospective study. *Journal of Psychosomatic Research, 59*, 175–179.

Rodriguez, N. R., DiMarco, N. M., & Langley, S. (2009). Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: Nutrition and athletic performance. *Journal of the American Dietetic Association*, *109*, 509–527.

Roecker, L., Meier-Buttermilch, R., Brechtel, L., Nemeth, E., & Ganz, T. (2005). Iron-regulatory protein hepcidin is increased in female athletes after a marathon. *European Journal of Applied Physiology*, *95*, 569–571.

Rogol, A. D. (2000). Sex steroid and growth hormone supplementation to enhance performance in adolescent athletes. *Current Opinion on Pediatrics, 12*, 382–387.

Rogol, A. D., Clark, P. A., & Roemmich, J. N. (2000). Growth and pubertal development in children and adolescents: Effects of diet and physical activity. *American Journal of Clinical Nutrition, 72*(2 Suppl.), 521S–528S.

Ruderman, N. B., Park, H., Kaushik, V. K., Dean, D., Constant, S., Prentki, M., & Saha, A. K. (2003). AMPK as a metabolic switch in rat muscle, liver and adipose tissue after exercise. *Acta Physiologica Scandinavica, 178*, 435–442.

Sanford, T. C., Davidson, M. M., Yakushoko, O. F, Martens, M. P., Hinton, P. S., & Beck, N. C. (2005). Clinical and subclinical eating disorders: An examination of collegiate athletes. *Journal of Applied Sport Psychology*, *17*, 79–86.

Saris, W. H., van Erp-Baart, M. A., Brouns, F., Westerterp, K. R., & ten Hoor, F. (1989). Study on food intake and energy expenditure during extreme sustained exercise: The Tour de France. *International Journal of Sports Medicine, 10*(Suppl. 1), S26–S31.

Saunders, M. J., Kane, M. D., & Todd, M. K. (2004). Effects of a carbohydrate–protein beverage on cycling endurance and muscle damage. *Medicine and Science in Sports and Exercise, 36*, 1233–1238.

Sawka, M. N., Burke, L. M., Eichner, E. R., Maughan, R. J., Montain, S. J., & Stachenfeld, N. S. (2007). American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine and Science in Sports and Exercise*, *39*, 377–390.

Schwartz, B., Jacobs, D. R., Jr., Moran, A., Steinberger, J., Hong, C. P., & Sinaiko, A. R. (2008). Measurement of insulin sensitivity in children: Comparison between the euglycemic-hyperinsulinemic clamp and surrogate measures. *Diabetes Care*, *31*, 783–788.

Sea, M. M., Fong, W. P., Huang, Y., & Chen, Z. Y. (2000). Weight cycling-induced alteration in fatty acid metabolism. *American Journal of Physiology*, *279*, R1145–R1155.

Sherman, W. M., Doyle, J. A., Lamb, D. R., & Strauss, R. H. (1993). Dietary carbohydrate, muscle glycogen, and exercise performance during 7 d of training. *American Journal of Clinical Nutrition, 57*, 27–31.

Shirreffs, S. M., & Maughan, R. J. (1998). Volume repletion after exercise-induced volume depletion in humans: Replacement of water and sodium losses. *American Journal of Physiology, 274*(5 Pt. 2), F868–F875.

Sinclair, L. M., & Hinton, P. S. (2005). Prevalence of iron deficiency with and without anemia in recreationally active men and women. *Journal of the American Dietetic Association, 105*, 975–978.

Stanescu, D., Lord, K., & Lipman, T. (2012). Epidemiology of type 1 diabetes in children. *Endocrinology and Metabolism Clinics of North America*, *41*, 679–694.

Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry, 159*(8), 1284–1293.

Stewart, T. M., & Williamson, D. A. (2004). Multidisciplinary treatment of eating disorders. Part 1. Structure and costs of treatment. *Behavior Modification*, *28*, 812–830.

Strong, W., Malina, R., Blimkie, C., Daniels, S., Dishman, R .... Trudeau, F. (2005). Evidence-based physical activity for school aged youth. *Journal of Pediatrics, 146*(6), 732–737.

Swenne, I. (2000). Heart risk associated with weight loss in anorexia nervosa and eating disorders: Electrocardiographic changes during the early phase of refeeding. *Acta Paediatrica, 89*, 447–452.

Stamler, J. S., & Meissner, G. (2001). Physiology of nitric oxide in skeletal muscle. *Physiology Review, 81*, 209–237.

Steen, S. N., Oppliger, R. A., & Brownell, K. D. (1988). Metabolic effects of repeated weight loss and regain in adolescent wrestlers. *Journal of the American Medical Association, 260*, 47–50.

Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, *159*, 1284–1293.

Tamborlane, W., Fredrickson, L. P., & Ahern, J. H. (2003). Insulin pump therapy in childhood diabetes mellitus. *Treatments in Endocrinology*, *2*, 11–21.

Terjung, R. L., Clarkson, P., Eichner, E. R., Greenhaff, P. L., Hespel, P. J., Israel, R. G., … Williams, M. H. (2000). American College of Sports Medicine roundtable. The physiological and health effects of oral creatine supplementation. *Medicine and Science in Sports and Exercise, 32*, 706–717.

Thanabalasingham, G., & Owen, K. R. (2011). Diagnosis and management of maturity onset diabetes of the young (MODY). *British Medical Journal*, *343*, d6044.

Tipton, K. D., Borsheim, E., Wolf, S. E., Sanford, A. P., & Wolfe, R. R. (2003). Acute response of net muscle protein balance reflects 24-h balance after exercise and amino acid ingestion. *American Journal of Physiology: Endocrinology and Metabolism, 284*, E76–E89.

Tipton, K. D., Ferrando, A. A., Phillips, S. M., Doyle, D., Jr., & Wolfe, R. R. (1999). Postexercise net protein synthesis in human muscle from orally administered amino acids. *American Journal of Physiology, 276*(4 Pt. 1), E628–E634.

Treasure, J., Claudino, A. M., & Zucker, N. (2010). Eating disorders. *Lancet*, *375*, 583–593.

Turner, L. W., & Bass, M. A. (2001). Osteoporosis knowledge, attitudes, and behaviors of female collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism, 11*, 482–489.

Ukropec, J., Reseland, J. E., Gasperikova, D., Demcakova, E., Madsen, L., Berge, R. K., … Sebökova, E. (2003). The hypotriglyceridemic effect of dietary n-3 FA is associated with increased beta-oxidation and reduced leptin expression. *Lipids, 38*, 1023–1029.

USDA Nutrition Evidence Library. (2010). Adiposity, children. Retrieved from www.nel.gov/topic.cfm?cat=3068

U.S. Department of Health and Human Services. (2005). *Dietary Guidelines for Americans*. Retrieved from www.health.gov/dietaryguidelines/dga2005/document/pdf/dga2005.pdf

U.S. Department of Health and Human Services. (2008). *Physical Guidelines for Americans*. Retrieved from www.health.gov/paguidelines

U.S. Department of Health and Human Services. (n.d.). *Bone health and osteoporosis: A report of the Surgeon General*. Washington, DC: Office of the Surgeon General.

U.S. Department of Health and Human Services & National Diabetes Education Program. (2006). Overview of diabetes in children and adolescents. Retrieved from http://ndep.nih.gov/diabetes/pubs/Youth_FactSheet.pdf

Vaidyanathan, J., Choe, S., & Sahajwalla, C. G. (2012). Type 2 diabetes in pediatrics and adults: Thoughts from a clinical pharmacology perspective. *Journal of Pharmacological Sciences, 101*, 1659–1671.

Van Baak, M. A., & Saris, W. H. (2000). The effect of caffeine on endurance performance after nonselective beta-adrenergic blockade. *Medicine and Science in Sports and Exercise, 32*, 499–503.

van Loon, L. J., Schrauwen-Hinderling, V. B., Koopman, R., Wagenmakers, A. J., Hesselink, M. K., Schaart, G., … Saris, W. H. (2003). Influence of prolonged endurance cycling and recovery diet on intramuscular triglyceride content in trained males. *American Journal of Physiology: Endocrinology and Metabolism, 285*, E804–E811.

Venables, M. C., Shaw, L., Jeukendrup, A. E., Roedig-Penman, A., Finke, M., Newcombe, R. G., … Smith, A. J. (2005). Erosive effect of a new sports drink on dental enamel during exercise. *Medicine and Science in Sports and Exercise, 37*, 39–44.

Verrotti, A., Catino, M., De Luca, F. A., Morgese, G., & Chiarelli, F. (1999). Eating disorders in adolescents with type 1 diabetes mellitus. *Acta Diabetologica*, *36*, 21–25.

Waters, D. L., Qualls, C. R., Dorin, R., Veldhuis, J. D., & Baumgartner, R. N. (2001). Increased pulsatility, process irregularity, and nocturnal trough concentrations of growth hormone in amenorrheic compared to eumenorrheic athletes. *Journal of Clinical Endocrinology and Metabolism, 86*, 1013–1019.

Waters, E., de Silva-Sanigorski, A., Hall, B. J., Brown, T., Campbell, K. J., Gao, Y., … Summerbell, C. D. (2011). Interventions for preventing obesity in children. *Cochrane Database Systematic Reviews*, (12), CD001871.

Watkins, B. A., Li, Y., Lippman, H. E., & Seifert, M. F. (2001). Omega-3 polyunsaturated fatty acids and skeletal health. *Experimental Biology and Medicine, 226*, 485–497.

Watkins, B. L. B. (2002). Eating disorders in school-aged children. *Child and Adolescent Psychiatric Clinics of North America, 11*, 185–199.

Watson, J., Deary, I., & Kerr, D. (2002). Central and peripheral effects of sustained caffeine use: Tolerance is incomplete. *British Journal of Clinical Pharmacology, 54*, 400–406.

Williams, D. E., Cadwell, B. L., Cheng, Y. J., Cowie, C. C., Gregg, E. W., Geiss, L. S., … Imperatore, G. (2005). Prevalence of impaired fasting glucose and its relationship with cardiovascular disease risk factors US adolescents, 1999–2000. *Pediatrics, 116*, 1122–1126.

Williams, M. B., Raven, P. B., Fogt, D. L., & Ivy, J. L. (2003). Effects of recovery beverages on glycogen restoration and endurance exercise performance. *Journal of Strength and Conditioning Research/National Strength and Conditioning Association, 17*, 12–19.

Willis, W. T., Brooks, G. A., Henderson, S. A., & Dallman, P. R. (1987). Effects of iron deficiency and training on mitochondrial enzymes in skeletal muscle. *Journal of Applied Physiology, 62*, 2442–2446.

Winston, A. P. (2012). The clinical biochemistry of anorexia nervosa. *Annals of Clinical Biochemistry*, *49*, 132–143.

Wolfe, R. R. (2000). Protein supplements and exercise. *American Journal of Clinical Nutrition, 72*(2 Suppl.), 551S–557S.

Wolfe, R. R., Klein, S., Carraro, F., & Weber, J. M. (1990). Role of triglyceride-fatty acid cycle in controlling fat metabolism in humans during and after exercise. *American Journal of Physiology, 258*(2 Pt. 1), E382–E389.

World Anti-Doping Agency. (2013). *The World Anti-Doping code. The 2014 prohibited list: International standard*. Retrieved from www.wada-ama.org/Documents/World_Anti-Doping_Program/WADP-Prohibited-list/2014/WADA-prohibited-list-2014-EN.pdf

Wright, K. S., Quinn, T. J., & Carey, G. B. (2002). Infant acceptance of breast milk after maternal exercise. *Pediatrics, 109*, 585–589.

Wyss, M., & Kaddurah-Daouk, R. (2000). Creatine and creatinine metabolism. *Physiology Review, 80*, 1107–1213.

Xita, N., & Tsatsoulis, A. (2010). Fetal origins of the metabolic syndrome. *Annals of New York Academy Sciences*, *1205*, 148–155.

Yeager, K. K., Agostini, R., Nattiv, A., & Drinkwater, B. (1993). The female athlete triad: Disordered eating, amenorrhea, osteoporosis. *Medicine and Science in Sports and Exercise, 25*, 775–777.

Zhu, J. S., Halpern, G. M., & Jones, K. (1998a). The scientific rediscovery of an ancient Chinese herbal medicine: *Cordyceps sinensis*. Part I. *Journal of Alternative and Complementary Medicine*, *4*, 289–303.

Zhu, J. S., Halpern, G. M., & Jones, K. (1998b). The scientific rediscovery of a precious ancient Chinese herbal regimen: *Cordyceps sinensis*. Part II. *Journal of Alternative and Complementary Medicine*, *4*, 429–457.

SECTION 2

# Adult Evidence-Based Nutrition in the Life Cycle

# CHAPTER 9

# Special Topics in Adult Nutrition: Chronic Disease Nutritional Assessment

*Jennifer L. Bueche, PhD, RD, CDN*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Outline the metabolic changes associated with the aging process and their impact on the nutritional requirements of adults throughout adulthood.
2. Refer to the Dietary Reference Intake to determine nutritional requirements for an individual or life stage group (specifically ages 19 to 30, 31 to 50, and 51 to 70 years).
3. Describe how to perform a nutritional assessment of an adult based on the Academy of Nutrition and Dietetics Nutrition Care Process and Model.
4. Identify the chronic diseases in adulthood that are most related to diet, the resulting nutritional implications, and subsequent appropriate medical nutritional therapy.
5. Apply the Academy of Nutrition and Dietetics Nutrition Care Process and Model to the case study provided.
6. Discuss the nutritional issues of epidemic proportion in the adult population and the recommended nutrition intervention.

This chapter begins by defining adulthood and outlining the metabolic changes associated with the aging process. This is important to understand because the maturation process greatly affects the dietary recommendations and the nutritional requirements of adults throughout their life as they age. In 1997, the Recommended Dietary Allowances (RDAs) went through a major overhaul to incorporate not only prevention of disease but promotion of health. The Dietary Reference Intake (DRI) system replaced the RDA and has been issued in stages since then. This information is presented and discussed with regard to the life stage groups (i.e., ages 19 to 30, 31 to 50, and 51 to 70 years) that span most of the adult years. Next, this chapter reviews the components of the nutritional assessment process including the Academy of Nutrition and Dietetics Nutrition Care Process and Model. Given that this text takes an evidence-based approach, for each of the chronic diseases presented the research literature was reviewed to provide evidence-based practice guidelines as they exist related to the four steps in the Nutrition Care Process (nutrition assessment, nutrition diagnosis, nutrition intervention, and/or nutrition monitoring) and evaluation for each particular chronic disease state. In addition, overweight/obesity and osteoporosis have been highlighted, given their rise to epidemic proportions in the adult population.

## Definition of Adulthood

Adulthood can span 60+ years, depending on a person's genetic predisposition and the choices a person makes throughout his or her life, including how healthy he or she chooses to eat. Life expectancy in the United States is 78.5 years based on the latest data released by the National Center for Health Statistics, with women still living longer than men (80.9 years vs. 73.6 years, respectively) (National Center for Health Statistics [NCHS], 2011). In 2009, the average healthcare cost per person totaled $8,000, and national healthcare in the United States totaled $2.5 trillion (NCHS, 2011). Not surprising, given the obesity epidemic in this country and the ensuing health consequences, a potential decline in life expectancy has been predicted in the first half of this century (Olshansky et al., 2005). That is, for the first time in 1,000 years, children today will not live as long as their parents.

In this chapter the adult years are initially broken down into three phases, the *young* adult years, the *middle* adult years, and the *older* adult years because they coincide with the life stage groups outlined by the DRIs (i.e., ages 19 to 30, 31 to 50, and 51 to 70 years). This makes presenting nutritional requirements across such a large span of years much easier to manage.

### Young Adult Years: Ages 19 to 30 Years

The list below represents the challenges and growth indices that occur in the young adult years.

- Typically, growth and maturation are completed by early adulthood, although some males can grow slightly after age 20 years (Brown et al., 2005).
- At this point the focus shifts to maintaining health and physical fitness and avoiding weight gain. This may be particularly challenging for college students, especially freshman, given the requirement to purchase an "all you care to eat" dining plan, a lack of involvement in sports because of time constraints, and competing with the net average weight gain of 15 lbs (Kelly, 2003; Levitsky & Youn, 2004).
- Bone density continues to develop until ages 30 to 35 years.
- Muscle mass continues to grow as long as muscles are used.
- Young adults tend to be very involved in their careers. If they are married with children, both parents juggle the demands of work, family, and day care.
- There are many different definitions of *family*. Reproduction may or may not be seen as a priority. According to the latest reports from the Centers for Disease Control and Prevention, in 1970 the average age for a first-time mother was approximately 21 years. Forty years later (2010), the mean age for a first-time mother was reported to be 25.4 years (Martin et al., 2012).
- Women of reproductive age have special nutrition needs that must be addressed at each stage of reproduction: before conception, prenatal, and postnatal/lactation.
- Alcohol accounts for 16% of caloric intake for those who consume alcohol. Young men consume on average 175 kcal of alcohol versus 60 kcal for young women. Alcohol consumption decreases with age (Nielsen, Kit, Fakhouri, & Ogden, 2012).
- In 2008, the five leading causes of death in young adults younger than age 25 years were unintentional injuries (43.7%), homicide (16.3%), suicide (13.3%), malignant neoplasms (5.1%), and diseases of the heart (3.3%) (NCHS, 2011).

### Middle Adult Years: Ages 31 to 50 Years

The list below represents the challenges and growth indices that occur in the middle adult years.

- Beginning at approximately age 30 years, physiologic functions that affect mobility begin to decline at the rate of about 1% or more per year (Worthington-Roberts, 1996).
- Body composition begins to shift; weight may seem like it is harder to lose. Weight gain that occurs after age 40 years in women and men goes hand in hand with hormonal changes and less exercise.
- Hormonal changes in men and women differ:
  - In men, testosterone levels begin to decline around ages 40 to 50 years, although sperm can fertilize eggs until much later. Decreased sperm production is linked to underweight status; malnutrition is linked to declining libido. National Health and Nutrition Examination Survey data show that as men age their calorie consumption decreases. Weight gain after 40 years more than likely occurs because of less exercise.
  - In women, the reproductive cycle lasts approximately 40 years, with 13 menstrual cycles per year (minus those missed during pregnancy or for other reasons). Birth rates for women aged 35 to 39 years are higher than they have ever been. The birth rate for women aged 40 to 44 years has increased more than 51% since 1990 (Martin et al., 2012).
- In 2008, the five leading causes of death in middle-aged adults younger than age 45 years were unintentional injuries (25.8%), malignant neoplasms (13.6%), diseases of the heart (12.2%), suicide (10.1%), and homicide (6.3%) (NCHS, 2011).

### Older Adult Years: Ages 51 to 70 Years

The list below represents the challenges and growth indices that occur in the older adult years.

- Typically, adults in this age group have more time to enjoy life with less responsibility in terms of raising children.
- Typically, they have more disposable income as their careers peak.
- Many older adults enjoy the benefits of eating healthy and exercising. If they do not, more

than likely it is because they are dealing with one or more chronic diseases (i.e., heart disease, diabetes).

- Muscle mass and strength decrease with age, but exercise can offset this decline (Chin et al., 2001). Lack of exercise results in loss of muscle mass, which in turn decreases overall lean muscle mass, increases body fat, and decreases metabolic rates, leading to weight gain.
- The immune system weakens with age, and the ability to fight off infection declines. Immune system function can be further compromised by an inadequate intake of nutrients or underlying chronic disease.
- Posture begins to deteriorate, which could be a result of lack of exercise and poor muscle tone, bad habits, or bone loss.
- Major changes occur in the ability to taste and smell food. The senses are dulled; thus, foods prepared for older adults should have stronger smells and flavors so they are more appealing.
- Saliva decreases, gastric secretions decline, and constipation, gas, and bloating can become more of a problem. Often the causes of these problems are incorrectly attributed to certain foods and the diet can become more and more restrictive as perceived intolerances increase.
- According to the latest National Health and Nutrition Examination Survey data, alcohol intake accounts for 3% of calorie intake of adults between the ages 51 and 64 years (Kant, 2000).
- In 2008, the six leading causes of death in older adults younger than age 65 years were malignant neoplasms (32%), diseases of the heart (21.6%), suicide (10.1%), unintentional injuries (6.8%), chronic lower respiratory disease (3.8%), and diabetes mellitus (3.5%) (NCHS, 2011).

Delaying the onset of disabilities caused by chronic disease in adulthood is referred to as compression of morbidity (Harvard Health Letter, 2002). Perhaps the overall goal should not necessarily be the *number* of years lived but the number of *healthy, good-quality* years lived.

## Nutritional Requirements for the Adult

By combining the tools of nutrition assessment and nutrition research currently available, nutritional requirements for the adult can be determined with confidence. The current Food and Nutrition Board was formed in 1993 and has been working on the DRI system, which has been released in stages over the last few years. The DRI system has become the overarching framework that encompasses four sets of standards: Estimated Average Requirements (EARs), RDAs, Adequate Intakes (AIs), and Upper Tolerable Intake Levels (ULs).

### Energy

The standard used to express energy needs, called Estimated Energy Requirements (EERs), refers to the average needs based on height, age, gender, and activity level to promote weight maintenance. Excess energy consumed is not excreted like most vitamins and minerals are; thus, it is not desirable to consume excess kilocalories (kcal) in any form (i.e., protein, carbohydrate, fat, and/or alcohol).

**Learning Point** In 2002, the Food and Nutrition Board released the Estimated Energy Requirements for Men and Women 30 Years of Age, which allows for adjustment in kilocalories based on age. For each year a person is younger than age 30, add 7 kcal/day for women and 10 kcal/day for men to that person's energy requirements. For each year a person is older than 30 years, subtract 7 kcal/day for women and 10 kcal/day for men.

It is estimated that energy needs decrease by 6% between the ages of 51 and 74 years, and they decline a further 6% after the age of 74 years (Gary & Fleury, 2002). The doubly labeled water method revolutionized the understanding of energy requirements and energy balance in humans because for the first time a method could be applied to humans and energy balance could be measured. The doubly labeled water contains two stable isotopes, deuterium oxide and oxygen-18. After an individual ingests an oral dose of water labeled with both isotopes (thus the term *doubly labeled water*), the deuterium is eliminated from the body as water and the oxygen-18 is eliminated as water and carbon dioxide. Periodic sampling of body water (urine, saliva, and plasma) is taken. The two isotopes are measured for 10 to 14 days and the difference between the two elimination rates is the measure of carbon dioxide production. Carbon dioxide can then be equated to total energy expenditure (Schoeller, 1999).

Trends in intake of energy and macronutrients in adults from 1999–2000 through 2007–2008 were compared. No significant increases or decreases in average energy intake were noted (men = 2,504 kcal; women = 1,771 kcal reported

for 2007–2008); however, macronutrient intake changed significantly with a decrease in the average carbohydrate intake and an increase in the average protein intake (Wright & Wang, 2010).

### Macronutrients

Protein recommendations are based on the RDA for protein (0.8 g protein/kg body weight) listed on the DRI based on a reference weight for each life stage group. The amount of protein recommended for women is the same for all three life stages (i.e., ages 19 to 30, 31 to 50, and 51 to 70 years) and is 46 g/day. For men the recommended amount of protein for all three life stages is 56 g/day.

The DRI for carbohydrate is based on the RDA, which is 130 g for both men and women for all three life stages. In people older than age 65 years, fasting blood glucose rises to a mean of 140 mg/dL, resulting in an increased **incidence** of non-insulin-dependent diabetes, which can be attributed to age, obesity, and decreased physical activity levels. Thus, recommendations regarding amount and distribution of carbohydrate must be individualized (Fonesa & Wall, 1995).

**incidence** Rate of occurrence.

The grams of fat per day are not determinable but rather are recommended in terms of acceptable macronutrient distribution ranges. The acceptable macronutrient distribution ranges for fat are 20% to 35% kcal energy/day, for carbohydrate are 45% to 65% kcal energy/day, and for protein are 10% to 35% kcal energy/day. These recommendations meet the needs of nearly 98% of all *healthy* individuals of similar age and gender based on each life stage group.

### Micronutrients

Most vitamin and mineral requirements for healthy people remain the same as they move throughout each life stage group. In November 2010, the Institute of Medicine (IOM) released new Dietary Reference Intakes (DRIs) for calcium and vitamin D in response to the government's request to address conflicting consumer messages and reflect most current research. The research yielded higher quality studies that provided strong evidence to support a role for vitamin D and calcium in bone health, but not in other health conditions. Additionally, evidence emerged that too much calcium and/or vitamin D may be harmful, challenging the concept that "more is better" (Institute of Medicine [IOM] Committee to Review Dietary Reference Intakes for Vitamin D and Calcium et al., 2011). The RDA for calcium (1,000 mg/d) is the same for both females and males up until the age of 51 years, when females need an additional 200 mg/day. The RDA for calcium is set at 1,200 mg/day for all adults older than age 70 years. The RDA for vitamin D is 600 IU/day (15 µg/day) for all adults up until the age of 70 years, when it increases to 800 IU/day (20 µg/day).

### Water

Often overlooked as a nutrient, water should be considered one of the most important nutrients because humans can survive only days without it compared to weeks or months without food (Mahan, 2004). The DRI recommendations are based on adequate intakes for generally healthy individuals who are adequately hydrated. It is well known that water requirements vary from individual to individual. Water recommendations take into consideration water from all sources, including all beverages and food. The water content of many foods is quite high (i.e., an apple is 84% water). Moisture in foods can contribute 20% of the total water intake. For healthy men, the recommendation of 3.7 L/day of water is the same for all three life stage groups. For healthy women, the recommendation of 2.7 L/day of water is the same for all three life stage groups. No upper level has been set for water because of a lack of suitable data; however, caution is warranted in consuming levels above the recommended intakes. The RDA for an adult is approximately 1 mL water/kcal expended as long as kilocalorie needs are met (Food and Nutrition Board, National Research Council, & National Academy of Sciences, 1989).

Two other methods exist for determining water requirements in the clinical setting. The first method estimates fluid requirements starting with 100 mL/kg for the first 10 kg of body weight. For the second 10 kg of body weight, another 50 mL/kg of water is added. For the remaining body weight, 20 mL/kg is used if the person is younger than 50 years, and 15 mL/kg is used if the person is older than 50 years. The second method is expressed in milliliters per kilogram based on age: 40 mL/kg, 15 to 30 years old; 35 mL/kg, 25 to 55 years old; 30 mL/kg, 44 to 65 years old; and 25 mL/kg, older than 65 years.

### Fiber

Fiber recommendations based on the average daily dietary fiber recommendations for 2002 (Adequate Intake) are 25 g for women (ages 19 to 50 years)

and 38 g for men (ages 19 to 50 years), or 14 g of dietary fiber per 1,000 kcal. For women and men ages 51 to 70 years, average daily dietary fiber recommendations for 2002 (Adequate Intake) are 21 g and 28 g of fiber, respectively, per day. Daily median intakes are 12.1 to 13.8 g for women and 16.5 to 19.5 g for men (Brown et al., 2005).

## Nutritional Assessment in Chronic Disease

Relationship between nutrition and overall health and the prevention of chronic disease has been well documented (Food and Nutrition Board, Committee on Diet and Health, & National Research Council, 1989; Public Health Service, 1988). In the adult years nutrition plays an important role in maintaining wellness and reducing risk of chronic disease. According to the *Healthy People 2020* report issued by the U.S. Department of Health and Human Services, nutrition is one of the many factors affecting health and longevity of older people. This is especially true for older people who are minorities and of low income (U.S. Department of Health and Human Services [U.S. DHHS], 2011). It is estimated that up to 60% of older people admitted to the hospital have protein energy malnutrition on admission or their nutritional status becomes compromised while hospitalized (Berry & Braunschwig, 1998; Gallagher-Allred, Voss, Finn, & McCamish, 1996; Gary & Fleury, 2002).

The physiologic changes in the adult and the assessment of energy expenditure in adults with chronic disease, two important knowledge and skill areas, are reviewed first before the discussion of nutrition assessment in chronic disease.

### Physiologic Changes in the Adult: Nutritional Implications

Eighty-five percent of older adults have one or more nutrition-related problems (American Dietetic Association, 2000). The most common are obesity, diabetes, cardiovascular disease, hypertension, arthritis, osteoporosis, and malnutrition. Many of these disorders put people at nutritional risk. Two major metabolic changes occur as people age. There is a decrease in lean body mass, with a 10% decrease in lean body mass from ages 25 to 60 years, another 10% decrease from ages 60 to 75 years, and another 20–25% decrease after age 75 years (Chin et al., 2001). Basal metabolic rate also decreases with age and in response to the decrease in lean muscle mass and increase in adipose tissues. The challenge then becomes to make sure that nutrient needs are met with declining kilocalorie needs while maintaining weight. **Table 9.1** summarizes the numerous physiologic changes experienced by adults as they age. These physiologic changes individually or cumulatively over time could negatively affect overall nutritional health.

RDAs and related standards apply only to healthy people, but many older people may not be healthy, especially those with a chronic disease. In addition, many older people take medications that may negatively affect specific nutrients. Certain conditions that are more prevalent with age increase the need for specific vitamins and minerals. Some examples include decrease in immune function (increased need for vitamin $B_6$, vitamin E, and zinc), increase in gastric pH (increased need for vitamin $B_{12}$, folic acid, calcium, iron, and zinc), and increase in oxidative stress (increased need for beta-carotene, vitamin C, and vitamin E) (Wardlaw, Hampl, & DiSilvestro, 2004). In fact, because of these conditions many nutrition experts recommend a balanced multivitamin and mineral supplement for all older adults.

### Assessment of Energy Needs in Adults with Chronic Disease

The assessment of energy needs can be accomplished in a number of ways. Indirect calorimetry is a method for estimating energy production by measuring $O_2$ consumption and $CO_2$ production at a specific point in time, accurately determining resting metabolic rate (Mahan, 2004). Devices such as the Douglas bag or metabolic cart are the most useful at measuring resting metabolic rate. Indirect calorimetry data such as the respiratory quotient tell clinicians the net substrate oxidation of fat,

**TABLE 9.1** Physiological Changes Experienced with Aging

- Decline in dental health
- Reduced thirst sensation
- Fall in gastrointestinal function
- Changes in liver, gallbladder, and pancreatic function
- Decline in kidney function
- Reduced immune function
- Reduced lung function
- Reduced hearing and vision
- Reduced cardiovascular health

carbohydrate, and protein, allowing for precise changes in nutrition support when necessary (Brandi & Calafa, 1997). New technology allows the production of smaller handheld versions of the larger, costly, less accessible models. Indirect calorimetry, although still used, is not used as a routine assessment of energy expenditure in clinical dietetic practice. Assessment of energy needs is most often determined by using energy prediction equations because they are practical and their accuracy rate is enough to provide a starting point in which adjustments (increases or decreases) can be made based on the patient's metabolic response to total kilocalories and diet composition provided. The following equations are currently used in clinical practice to determine energy needs:

- *Harris–Benedict equation:* Harris and Benedict conducted a study in which 239 subjects (aged 15 to 74 years) had their basal metabolic rates measured. By today's standards these measurements would be referred to as resting metabolic rates. The Harris–Benedict equation (see **Table 9.2**) was derived from this study and is still used today. It is well known, however, that the Harris–Benedict equation can overestimate energy needs by as much as 20% to 30%, with an accuracy rate of 69% (Frankenfield, Roth-Yousey, & Compher, 2005).
- *Mifflin–St. Jeor equation:* Body composition has changed a great deal since 1919 when the Harris–Benedict equation first was derived. The Mifflin–St. Jeor equation (see **Table 9.3**) was developed in 1990 and has been shown to report resting metabolic rate 82% of the time in nonobese adults. The Mifflin–St. Jeor equation has an overestimation error of 15% and an underestimation error of 18%. Once resting metabolic rate is calculated, it is then multiplied by activity or injury and stress factors to determine the total energy required.

**TABLE 9.2 Harris–Benedict Equation**

**Men**
BEE = 66.47 + 13.75 (wt in kg) + 5 (ht in cm) – 6.76 (age in yr)

**Women**
BEE = 655.1 + 9.56 (wt in kg) + 1.85 (ht in cm) – 4.68 (age in yr)

- *Ireton-Jones equation:* The Ireton–Jones equation (see **Table 9.4**) provides estimated energy expenditure for hospitalized and critically ill patients who are either breathing spontaneously or are ventilator dependent. This equation takes into account age, weight (kg), and gender. The equation adjusts for trauma, burns, and/or obesity. The obesity adjustment should be used when the patient weighs more than 130% of the ideal body weight or has a **body mass index (BMI)** above 27 (Ireton-Jones, 1997).

**body mass index (BMI)** Calculated by dividing weight in kilograms by height in meters squared (BMI = kg/m$^2$); shown in the research literature to be correlated with adiposity and increased risk of chronic disease.

Depending on the illness, calorie needs can rise substantially in the hospitalized patient. For this reason, it is critically important to assess the energy needs of each patient individually (Klipstein-Grobusch, Reilly, Potter, Edwards, & Roberts, 1995).

**TABLE 9.3 Mifflin–St. Jeor Energy Estimation**

**Males**
RMR = (10 × wt [in kg]) + (6.25 × ht [in cm]) – (5 × age [in yr]) + 5

**Females**
RMR = (10 × wt [in kg]) + (6.25 × ht [in cm]) – (5 × age [in yr]) – 161

| Activity or Injury and Stress Factors | |
|---|---|
| Sedentary or weight maintenance | 1.2 |
| Light activity | 1.3 |
| Moderately active, infection, healing | 1.5 |
| Very active, extreme stress, burns | 2.0 |

RMR, resting metabolic rate.

**TABLE 9.4 Ireton-Jones Equation for Spontaneous and Ventilator-Dependent Breather**

**EEE Spontaneous Breather**
EEE(S) = 629 – 11(A) + 25W – (609 if obese)
EEE(S) = 629 – 11(A) + 25(W in kg) – (609 if obese)

**EEE Ventilator Dependent**
EEE(V) = 1,784 – 11(A) + 5(W) + 244(S) + 239(T) + 804(B)
EEE(V) = 1,784 – 11(A) + 5(W in kg) + 244 (if male) + 239 (if trauma) + 804 (if burn)

EEE(S), spontaneously breathing; EEE(V), ventilator dependent; S, sex (male, 1; female, 0); T, trauma; A, age; W, weight; B, burn.

## Nutritional Care Process and Model

Before the adoption of the Academy's final version of the Nutrition Care Process and Model, no standardized nutrition care process existed. Dietetic professionals strongly supported a standardized nutrition care process for use by registered dietitians and dietetic technicians because this standardized process would in effect promote the dietetic professional as the unique provider of nutrition care and enhance the practice of dietetics by improving outcomes and showing the value of our services (Lacey, 2003).

The Academy's Nutrition Care and Process and Model, a roadmap to quality nutrition care and outcomes, is circular in shape and looks very much like a dart board with a central core and middle and outer rings (see **Figure 9.1**). The central core depicts the relationships between patient/client/group and the dietetics professional. This central core sits inside a larger core, called the Nutrition Care Process, of which there are four steps: nutritional assessment, nutritional diagnosis, nutritional intervention, and nutrition monitoring and evaluation. Two outer rings surround the nutrition care process. The middle ring depicts the knowledge and skills that dietetic professionals bring to the process. The outer ring depicts the environmental factors that influence the process, such as the practice setting.

Two very important systems that are not considered part of the Nutrition Care Process and Model but that are critically important are the screening and referral system and the outcomes management system. Nutritional screening is not required to be done by a dietetic professional (registered dietitians or dietetic technicians); however, the process depends on accurate and timely nutritional screening. It is critically important that the nutritional screening process be monitored and

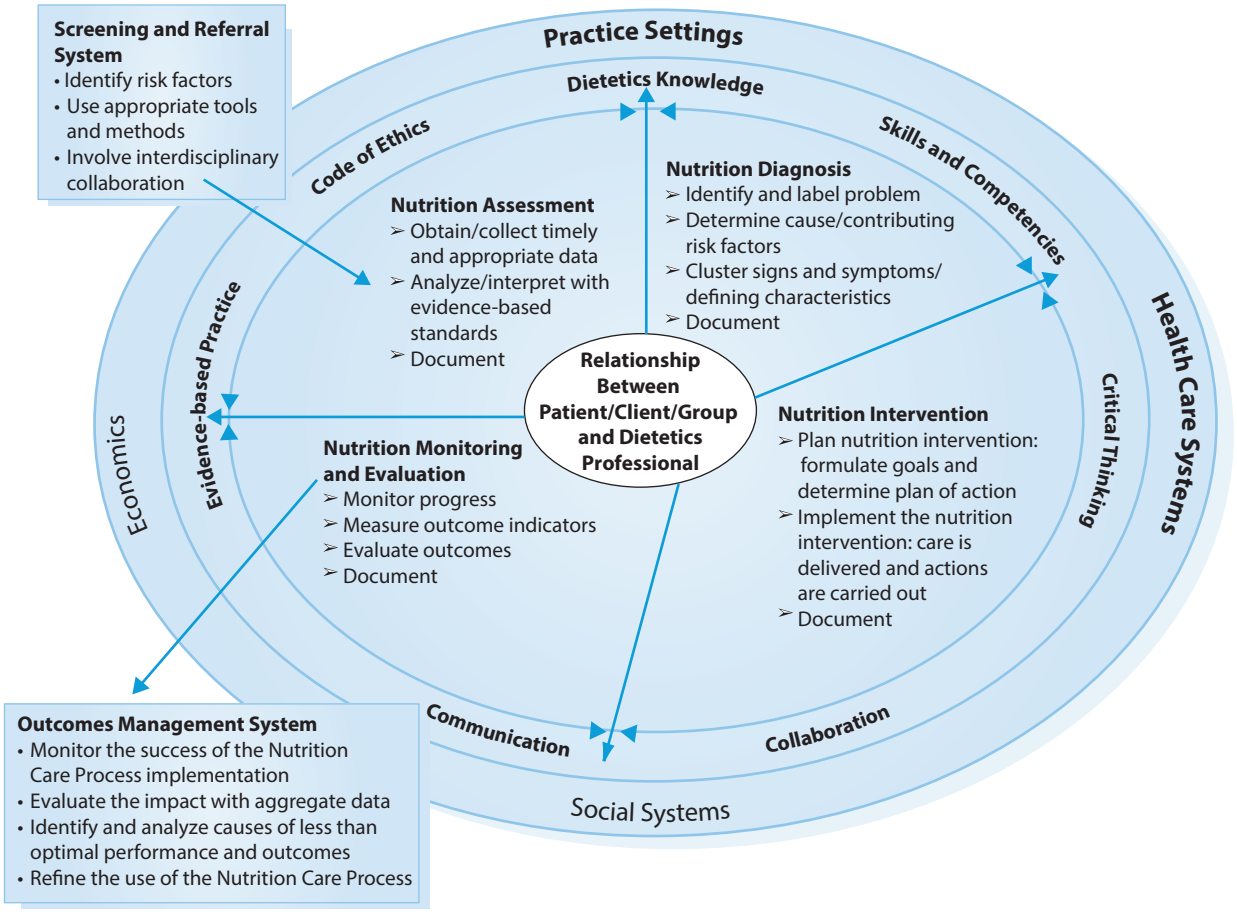



**Figure 9.1** ADA Nutrition Care Process and Model.

Reproduced from *The Journal of The American Dietetic Association*, vol 103(8): 1061–1072, Lacey et al.: "Nutrition Care Process and Model." © 2003, with permission from Elsevier.

evaluated as part of the outcomes management system to ensure that those who are at nutritional risk are identified. The outcomes management system allows data to be collected and analyzed to determine where improvements need to be made to improve overall quality of nutritional care provided.

It is important to distinguish between *standardized process* and *standardized care*. The Nutrition Care Process and Model does not attempt to standardize care (i.e., all patients receive the same care). Nutritional care still must be individualized. What the Nutrition Care Process and Model does do is provide a framework or guide based on a series of connected steps or actions in which nutrition care is provided. In a nutshell, the Nutrition Care Process and Model can be defined as "a systematic problem-solving method that dietetics professionals use to critically think and make decisions to address nutrition related problems and provide safe and effective quality nutrition care" (Writing Group of the Nutrition Care Process/Standardized Language Committee, 2008a, 2008b).

There are four steps in the Nutrition Care Process: (1) nutrition assessment, (2) nutrition diagnosis, (3) nutrition intervention, and (4) nutrition monitoring and evaluation. There is a difference between medical nutrition therapy and the Nutrition Care Process. Medical nutrition therapy was redefined as part of the 2001 Medicare MNT benefit legislation as "nutritional diagnostic, therapy, and counseling services for the purpose of disease management, which are furnished by a registered dietitian or nutrition professional" (Smith, 2003). Medical nutrition therapy is only one type of nutrition care. The Nutrition Care Process and Model is broader in scope and guides nutrition education and other preventative nutrition services.

In 2003, a task force of the American Dietetic Association began developing a standardized language for the Nutrition Care Process, which currently includes all four steps. A standardized nutrition language was determined to be necessary because of the lack of nutrition terms that existed in standardized languages serving other health professions and to allow the digital sources necessary for evidence-based practice within the profession of dietetics (Jenkins, Meyers, Charney, & Escott Stump, 2006). Evidence-based practice is needed to determine which practices support outcomes (Myers, Pritchett, & Johnson, 2002). The government is looking to evidence-based practice as a way to improve the quality of medical care for patients while managing the rising costs of an overburdened, nearly dysfunctional healthcare system (Smith, 2003). By using evidence-based practice as a part of the Nutrition Care Process and Model, practitioners and educators can apply the best research knowledge to dietetics practice. Demonstrating the science behind dietary recommendations and validating nutritional assessment tools utilizing evidence-based analysis to further dietetics practice strengthen the profession. The Academy has begun this process and has created evidence-based nutrition practice guidelines for numerous topic areas. All Academy members can access this information for free through the Academy Evidence Analysis Library on the Academy's website (http://www.eatright.org).

In 2009, the Academy of Nutrition and Dietetics (then known as the American Dietetic Association) published the first *International Dietetics and Nutrition Terminology Manual* (*IDNT*; 2nd edition) to describe the complete standardized language (American Dietetic Association, 2009). Because the standardized language has been revised based on validation studies and changes in practice, the Academy has published updated editions of the *IDNT* manual with the most recent being the fourth edition (Charney et al., 2006).

## Nutritional Assessment in Chronic Diseases

In this section we review the following chronic diseases: cardiovascular disease (coronary heart disease and cerebrovascular disease), cancer, chronic obstructive pulmonary disease (COPD), diabetes, chronic kidney disease, and HIV/AIDS. For each chronic disease reviewed, disease-specific considerations are addressed for conducting a nutritional assessment. Evidence analysis for evidence-based practice is presented for each chronic disease based on a thorough review of the current literature.

### Cardiovascular Disease

Cardiovascular disease affects one in four Americans and is still the number one leading cause of death in the United States. Death rates from heart disease are highest in the South and lowest in the West (Kochanek, Xu, Murphy, Miniño, & Kung, 2011). *Cardiovascular disease* is a broad term that encompasses a number of conditions, such as coronary heart disease (often called heart disease), stroke, and any other conditions that affect the structure or function of the heart. If the heart does not receive adequate blood, the heart becomes starved

of oxygen and the vital nutrients it needs to work properly. This could result in unstable angina (chest pain), damage to the heart muscle, and/or myocardial infarction; the severity and progression depends on the degree of blockage.

Symptoms are highly variable from person to person, more so when comparing symptoms for men versus women. Treatment for coronary artery disease involves an assessment of risk factors to determine the appropriate intervention as outlined by the latest report issued by the National Cholesterol Education Program (NCEP) Adult Treatment Panel III report recommendations (NCEP, 2001; National Heart, Lung, and Blood Institute & National Institutes of Health, 2002).

### Nutritional Assessment in Coronary Heart Disease

Nutritional assessment should include evaluation of the following:

- Excess weight for height: BMI > 25 (overweight), BMI > 30 (obese)
- Excess abdominal fat: Waist circumference of 35 inches for females, 40 inches or more for males, or waist-to-hip ratio of less than 0.8 for women and less than 1.0 for men
- Evidence of **hypertension** (blood pressure > 140/90 mm Hg)
- Complete lipid profile, fasting preferred (total cholesterol, low-density lipoprotein, and **high-density lipoprotein** cholesterol)
- Nonfasting total cholesterol, high-density lipoprotein if complete fasting lipid profile unavailable (proceed to lipoprotein profile if total cholesterol ≥ 200 mg/dL or high-density lipoprotein, 40 mg/dL)
- Plasma apolipoprotein B (an atherogenic lipoprotein)
- Homocysteine levels
- Evidence of diabetes, fasting blood sugar levels
- Evidence of atherogenic diet: Evaluate dietary intake (saturated fat and trans fatty acid intake, omega-3 fatty acid intake, overall diet quality particularly with regard to intake of fruits, vegetables, and whole grains)
- Physical activity level
- Smoking, tobacco use
- Family history of premature coronary heart disease
- Gender
- Age (men ≥ 45 years, women ≥ 55 years)

**hypertension** Systolic blood pressure above 140 mm Hg or diastolic blood pressure above 90 mm Hg.

**high-density lipoproteins** Often referred to as "good cholesterol," a lipoprotein that contains mostly protein with less cholesterol and triglyceride. High levels of high-density lipoproteins in the blood are associated with a decreased risk of coronary heart disease.

**homocysteine** An amino acid that can be measured in the blood that has been shown to be an independent risk factor for cardiovascular disease.

### Evidence Analysis for Evidence-Based Practice in Coronary Heart Disease

The original NCEP guidelines followed the recommendations of the American Heart Association's Step I and Step II diets (NCEP, 1988). The new NCEP guidelines released in 2002 are evidence-based guidelines that result in reduction of low-density lipoprotein cholesterol levels and reduce risk of coronary heart disease (National Heart, Lung, and Blood Institute & National Institutes of Health, 2002). The Therapeutic Lifestyle Changes diet lowers saturated fat and cholesterol intakes to levels of the previous Step II American Heart Association diet (see **Table 9.5**). If lipid levels are not achieved initially, plant stanols/sterols (2 g/day) and viscous (soluble) fiber (10 to 25 g/day), known to have low-density lipoprotein lipid-lowering effects, may be added to the diet. The new NCEP Adult Treatment Panel III guidelines increase the emphasis on weight management and physical activity:

- Consume less than 200 mg of dietary cholesterol per day.
- Limit sodium intake to less than 2,400 mg a day.
- Eat 25% to 35% or less of the day's total calories from fat.
- Consume less than 7% of the day's total calories from saturated fat.
- Eat just enough calories to achieve or maintain a healthy weight and reduce your blood cholesterol levels.
- The recommendations for cholesterol and sodium are the same for everyone on the Therapeutic Lifestyle Changes diet, regardless of caloric intake.
- The recommendations for saturated fat and total fat are based on the percentage of calories consumed and vary with intake.
- Cholesterol-lowering medications may be necessary if diet alone does not adequately lower levels.

Cerebrovascular disease, or stroke, strikes about 700,000 Americans each year, with most surviving. Nearly 5 million stroke survivors are managing their health today (www.webmd.com/

**TABLE 9.5** Therapeutic Lifestyle Changes Diet Guidelines

| Food Group | Number of Servings | Serving Size |
|---|---|---|
| Lean meat, fish, poultry, and dry beans | No more than 6 oz/day | • 6 ounces maximum per day lean meat, poultry, and fish<br>• ½ cup cooked dry peas or beans<br>• ½ cup tofu |
| Eggs | No more than four yolks per week | • Two egg whites = 1 whole egg in recipes |
| Egg substitute/egg whites | Unlimited | |
| Low-fat milk, yogurt, and cheese | 2–3 | • 1 cup fat-free or 1% milk<br>• 1 cup nonfat or low-fat yogurt<br>• 1 ounce of low-fat or fat-free cheese (< 3 g of fat in 1 ounce) |
| Fats and oils | No more than 6–8 | • 1 teaspoon soft margarine or vegetable oil<br>• 1 tablespoon salad dressing<br>• 1 ounce nuts |
| Fruits | 2–4 | • 1 piece of fruit<br>• ½ cup diced fruit<br>• ¾ cup juice |
| Vegetables | 3–5 | • 1 cup leafy or raw<br>• ½ cup cooked<br>• ¾ cup juice |
| Breads, cereals, pasta, rice, and other grains | 6–11 | • 1 slice of bread<br>• ½ bun, bagel, or muffin<br>• 1 ounce dry cereal<br>• ½ cup cooked cereal, potatoes, pasta, rice, or other grains |
| Sweets and snacks | Now and then | |

Data from National Cholesterol Education Program (ATP III). Serving sizes are based on the USDA's MyPyramid.

stroke/default.htm). There are two types of stroke based on cause: ischemic and hemorrhagic. An ischemic stroke is caused by a blood clot that blocks the supply of blood to the brain. Most often this is caused by atherosclerosis, which is caused by high blood pressure, diabetes, high cholesterol, or a combination of the three. Blood flow can also be interrupted by low blood pressure (hypotension), which may be a result of a heart attack, blood loss, or a severe infection. Clots can also break free as a result of a procedure or surgery and cause a stroke. A hemorrhagic stroke is the result of bleeding in or around the brain as a result most often of a ruptured blood vessel or uncontrolled blood pressure.

### Nutritional Assessment in Cerebrovascular Disease

Generally, the nutritional assessment here is the same as that for coronary heart disease except that a patient's functional status needs to be determined, especially as it relates to his or her ability to meet nutritional needs orally. **Table 9.6** shows the recommended intakes of total fat and saturated fat.

### Evidence Analysis for Evidence-Based Practice in Cerebrovascular Disease

Given the fact that evidence has already been presented for the treatment of coronary heart disease (with the underlying cause of atherosclerosis), the focus here is on the evidence available to support prevention and control of hypertension, the other major underlying cause of strokes. The Trials of Hypertension Prevention and the Dietary Approaches to Stop Hypertension studies clearly demonstrated that dietary intervention prevented hypertension or lowered blood pressure in persons with high to normal blood pressure (Appel et al., 1997; Trials of Hypertension Prevention Collaborative Research Group, 1997). The Dietary

**TABLE 9.6** **Maximum Daily Intake of Fat and Saturated Fatty Acid**

| | Calorie Level | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **1,600** | **1,800** | **2,000** | **2,200** | **2,400** | **2,600** | **2,800** | **3,000** |
| Total fat (g) | 44–62 | 50–70 | 56–78 | 61–86 | 67–93 | 72–101 | 78–109 | 83–117 |
| Saturated fat (g) | 12 | 14 | 16 | 17 | 19 | 20 | 22 | 23 |

Recommended intake of total fat 25% to 35% and saturated fat < 7%.

Data from National Education Cholesterol Program (ATP III).

Approaches to Stop Hypertension diet is used to prevent and control blood pressure and is based on a "heart healthy" diet with twice the average daily consumption of fruits and vegetables (available at http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf).

## Cancer

Cancer is a group of related diseases in which damage to the DNA of the cells causes uncontrolled growth and spread of abnormal cells. This abnormal cellular growth can have an effect on any one part of the body (breast, skin, or lungs) or the whole body (**metastases**). Cancer is the second leading cause of death in the United States, with 1.2 million new cases diagnosed annually and nearly 12 million Americans living with a history of cancer (National Cancer Institute [NCI], 2012). Given the fact that 35% of all cancers are diet related, cancer prevention depends to a large degree on the overall quality of diet and the ability to maintain a healthy weight.

**metastasis** Growth of malignant tissue that spreads to surrounding tissues or organs.

### Nutritional Assessment

One of the most important markers of nutritional status is assessment of body weight, which can be represented by an unplanned weight loss or a BMI less than 22, a serum albumin less than 3.5, or a cholesterol level less than 150 mg/dL. The diet history is evaluated to determine adequacy of caloric intake and overall diet quality, especially with regard to protein and vitamin and mineral intake. Particular attention is paid to the use of vitamins and minerals and complementary and alternative therapies.

### Evidence Analysis for Evidence-Based Practice

The main nutrition goal is to prevent or reverse nutrient deficiencies, to preserve lean body mass, to minimize nutrition-related side effects, and to maximize the quality of life. The role of nutrition is vitally important in the treatment of cancer patients. Much time is spent dealing with the common nutrition problems that occur as a result of the disease, treatment, or both. Common nutrition problems include anorexia/cachexia, nausea, vomiting, mucositis, esophagitis, xerostomia, dysgeusia, hypogeusia, diarrhea, and constipation. Recommendations for dealing with common nutrition problems are outlined in **Table 9.7**.

Nutritional recommendations for energy are highly variable and must be individualized. Protein requirements range from 1.2 to 2.0 g/kg with average protein requirements at 1.5 g/kg. Energy requirements can range between 21 and 40 kcal/kg/day depending on need for weight gain (30–40 kcal/kg/day), metabolic state (normal: 25–30 kcal/kg/day vs. hypermetabolic state: 35 kcal/kg/day), treatment (30–35 kcal/kg/day), presence of sepsis (25–30 kcal/kg/day), or need to adjust for obesity (21–25 kcal/kg/day) (Gottschlich, 2007). Patients with cancer typically exhibit weight loss, anorexia, and malnutrition as evidenced by the fact that more than 50% of people with cancer lose body weight and more than one-third lose more than 5% of their usual body weight (Goldman et al., 2006; Skipworth et al., 2007). Alterations occur in glucose, protein, and lipid metabolism because of the metabolic demands of the cancerous tumor(s). Tumors exert a constant demand for glucose and amino acids. To keep up with this constant need for glucose, gluconeogenesis and lipolysis occur at a high rate at the expense of skeletal muscle and adipose tissue, the fuel sources. Catabolism can be so great that visceral organ and other body proteins (albumin) are affected.

Antitumor therapy, such as chemotherapy, radiation, surgery, or immunotherapy, can contribute to nutritional alterations in patients by interfering

**TABLE 9.7** **Strategies to Improve Intake in Cancer Patients**

Strategies to address anorexia/cachexia:

- Identify factors contributing to poor appetite.
- Keep high calorie/high protein foods/liquids on hand for when appetite is good:
- Milkshakes, Carnation Instant Breakfast, peanut butter, fruited yogurt
- Discourage intake of high-fiber foods that can cause early satiety.
- Appetite stimulant if anorexia is longstanding or other strategies fail to improve condition

Strategies to address nausea:

- Sip liquids at frequent intervals separately from solid foods to maintain hydration.
- Discourage fasting because it may cause hypoglycemia and increase nausea.
- Cold or room temperature foods may be better tolerated.
- Avoid high-fat, high-fiber, spicy, or gas-producing foods that may be poorly tolerated.

Strategies to address vomiting:

- Encourage adequate fluids to prevent dehydration.
- Start with low-fat fluids and advance as tolerated.
- Introduce dry starchy foods first when advancing.
- Introduce high-fiber and high-fat foods last.
- Avoid eating 1½ to 2 hours pre- and posttreatment.

Strategies to address mucositis:

- Encourage good oral hygiene practices.
- Use oral baking soda rinse:
- 1 mL of baking soda to 250 mL of water
- Soft, moist, semisolid, or blended foods may be tolerated better than rough/crisp foods.
- Discourage intake of known irritants:
- Tart or acidic foods, spicy or salty foods, very hot or cold foods, tobacco, alcohol, and alcohol-based items
- Recommend dunking or moistening dry foods:
- Order extra gravy on trays.
- Alter temperature and consistency to individual tolerances.
- Reinforce use of analgesics as prescribed before meals to reduce pain associated with eating.

Strategies to address esophagitis:

- Same as strategies for mucositis plus the following:
  - Suggest the use of local anesthetic and analgesic before meals.
  - In cases of peptic esophagitis, antireflux and antacid therapy may be helpful.
  - Recommend regular antacids before and 1 hour after meals and before bed.

Strategies to address thick saliva/mucus:

- Beverages or foods that are slightly tart or carbonated may help to thin secretions.
- If milk products are found to affect mucus try soy-based items (such as soy milk) for tolerance.
- Mucous production may be minimized if clear fluids are consumed after milk products.
- Limit caffeine, alcohol, and spicy foods.
- Encourage a mouth rinse throughout the day.

Strategies to address xerostomia:

- Increased liquid consumption may provide symptomatic relief; however, liquids have no lubricating properties.
- Encourage consumption of foods such as gravies, sauces, and salad dressings (Italian).
- Mint or tart sugar-free gum/candy may stimulate saliva production.
- Discourage commercial mouthwashes and alcohol because they contribute to dryness.
- Citric-acid-containing beverages such as lemonade, orange-flavored soft drinks, frozen juice bars, and sherbets may help increase secretions.
- Recommend sugar-free or diet products to reduce the risk of dental decay and mouth infection.

Strategies to address dysgeusia:

- Determine specific taste or smell changes.
- Encourage fluids with meals to decrease unpleasant tastes.
- Reinforce proper oral care before and between meals.
- Tart foods can stimulate taste buds.
- Suggest mild-tasting foods such as biscuits, milk, pudding, and custards.
- If meat tastes bitter or metallic:
- Serve meat cold or at room temperature.
- Include meat in mixed dishes like casseroles.
- Choose alternative protein sources from dairy group: cottage cheese, yogurt, custard.
- Try tofu or eggs.
- Marinate foods in pineapple or lime juice, vinegar, wine, or sweet and sour sauce.
- Eat with plastic utensils in place of metal ones.
- Cinnamon or sugar-free gum or mints may help mask the metallic taste.

Strategies to address hypogeusia:

- Reinforce proper oral care.
- Encourage experimenting with strong flavors and seasonings.
- Encourage liberal intake of "treats" or comfort foods.
- Emphasize a variety in colors and textures in a meal to encourage eating.

(*continued*)

**TABLE 9.7 Strategies to Improve Intake in Cancer Patients (*continued*)**

Strategies to address diarrhea:

- When appropriate use antidiarrheal agents as prescribed by physician first to avoid dietary limitations.
- Encourage energy-dense fluids to match output.
- Encourage potassium-rich foods.
- Try the following modifications one at a time to determine effectiveness:
- Limit bowel stimulants (e.g., caffeine, alcohol, prunes).
- Adjust fat intake as tolerated.
- Restrict lactose.
- Restrict fiber intake.
- Limit gas-producing foods.
- Limit foods or fluids that exacerbate symptoms (e.g., spices).

Strategies to address constipation:

- Encourage intake of naturally laxative foods such as prunes, rhubarb, papaya, and mango.
- Encourage physical activity as able.
- Advise the use of a bulk-forming laxative and reinforce the importance of adequate fluid intake.
- Daily use of stool softeners as necessary

Data from American Institute for Cancer Research (www.aicr.org).

with their ability to ingest, digest, and absorb adequately. Ideally, early intervention is the key to minimize weight loss and to prevent or correct nutritional deficiencies as soon as possible and if at all possible.

### Chronic Obstructive Pulmonary Disease

COPD is a slow, progressive obstruction of the airways of the lung. According to the National Heart, Lung, and Blood Institute (NHLBI, 2013), COPD affects more than 24 million Americans (12 million diagnosed and 12 million undiagnosed) and is the third leading cause of death (see http://www.nhlbi.nih.gov/health/public/lung/copd/index.htm). There are two categories of patients with COPD. Type 1, or emphysema, is seen in older patients who are thin and cachectic, with corpulmonale that develops late in course. **Corpulmonale** is a medical term used to describe enlargement and failure of the right side of the heart as a result of pulmonary hypertension from prolonged high blood pressure in the arteries or veins of the lungs. Type 2, or chronic bronchitis, is seen in patients with normal weight or excessive weight and prominent hypoxemia and in whom corpulmonale develops early in the course of the disease.

**corpulmonale** A heart condition characterized by enlargement of the right side of the heart (right ventricle) and failure caused by pulmonary hypertension.

#### Nutritional Assessment

It is well known that the maintenance of a well-nourished nutritional state plays a key role in maintaining ventilatory muscle strength, improved immune response, and reduced risk of respiratory mortality (Gray-Donald, Gibbson, Shapiro, Macklern, & Martin, 1996; Rogers, Donahoe, & Constantino, 1992; Whittaker, Ryan, Buckley, & Road, 1990). Malnutrition, however, is common among patients with COPD (Laaban et al., 1993; Schols, Mostert, Soester, Greve, & Wouters, 1989). Given the degree of malnutrition in the COPD population, Thorsdottir, Gunnarsdottir, and Eriksen (2001) conducted a study to evaluate and develop a screening method for detecting malnutrition in patients with COPD based on previously published work by Elmore and associates (1994) and refinement of their previously published general screening tool (Thorsdottir, Eriksen, & Eysteinsdottir, 1999). Serum albumin, total lymphocyte count, BMI, and triceps skinfold thickness were sensitive measures in the identification of malnutrition. Triceps skinfold thickness and BMI were shown to be the best single parameters for detecting malnutrition, but neither one individually or together equaled the quality of the screening tool using the four-point criteria. Results of the study confirmed that patients with COPD are frequently malnourished and that the screening tool developed could identify 69% of malnourished patients.

In a study conducted by Soler-Cataluna and coworkers (2005), muscle mass depletion was estimated indirectly by determining the midarm muscle area (of the nondominant arm) and was determined to be a better predictor of mortality than BMI in COPD patients. This has important implications for nutritional screening, given the obesity epidemic in this country, because normal and overweight patients show muscle mass depletion (Schols et al., 1993). Adequacy of dietary intake and vitamins and minerals also needs to be addressed.

#### Evidence Analysis for Evidence-Based Practice

Identifying and correcting malnutrition are the main goals when working with patients with COPD. Energy intake must be increased to meet needs;

however, this must be done in balance. A balanced protein:fat:carbohydrate ratio (15–20%:30–45%:40–55% of calories) is needed to preserve the respiratory quotient. Patients' nutritional needs should be assessed on an individual basis; however, a study conducted by Thorsdottir and Gunnarsdottir (2002) recommended energy intakes to be above 140% of basal energy expenditure and protein consumption to be at least 1.2 g/kg body weight to avoid protein losses, prevent weight loss, and prevent worsening nutritional status.

Deficiency in lung elasticity and respiratory muscle results in hypoproteinemia and in calcium, magnesium, phosphorus, vitamin K, and vitamin C deficiencies. Intakes based on the DRIs should be provided, with magnesium and phosphorus levels monitored for patients receiving aggressive nutritional support. Increased vitamin D and vitamin K may be warranted based on results of **bone mineral density** tests, glucocorticoid medication use, and adequacy of intake (Mahan, 2004). Lung function has been shown to be better when higher antioxidant levels are given (Hu & Cassano, 2000).

**bone mineral density** A measurement of bone mass after development is complete; reported in grams per centimeter squared.

## Diabetes

Diabetes is a serious chronic disease characterized by abnormalities in the metabolism of cholesterol, protein, and fat. The incidence of type 2 diabetes is tracking right along with the obesity epidemic and is fueling the diabetes epidemic (Cowie, MMWR, & CDC Surveillance System, 2003). Glucose intolerance or hyperglycemia is the common denominator, and the body does not produce or respond to insulin. Insulin is a hormone produced by the B cells of the pancreas, and it is the key that "unlocks" the cells so the cells can then utilize glucose as a fuel source. Diabetes affects 25.8 million people, with more than 7million undiagnosed; it is the sixth leading cause of death (CDC, 2011; Heron, 2011).

The three major forms of diabetes are type 1, type 2, and gestational diabetes. Type 2 diabetes accounts for 90% to 95% of all cases. Diabetic ketoacidosis (DKA) may be the first symptom of undiagnosed diabetes, but it also can occur in people diagnosed with type 1 and type 2 diabetes as a result of an illness, infection, stress, trauma, or poor compliance with insulin therapy. Symptoms include hyperglycemia, vomiting, dehydration, shortness of breath, confusion, and if severe enough, it can cause coma and even death. Treatment includes administration of intravenous fluids that include electrolytes to correct dehydration, insulin to promote uptake of glucose by the cells, and treatment for underlying cause(s) (Kitabchi, Umpierrez, Miles, & Fisher, 2009).

### Nutritional Assessment

A body weight assessment should be conducted (i.e., assessment of height, weight, BMI, and waist circumference). Waist circumference more than 40 inches for men and more than 35 inches for women has been shown to increase **insulin resistance** (Centers for Disease Control and Prevention [CDC], 2002). Blood pressure should be checked to determine whether a patient is hypertensive as well (at least 140/90 mm Hg). Laboratory work should be reviewed and evaluated based on the following: hemoglobin A1c, or **glycosylated hemoglobin (HbA1c)**, fasting glucose, urinary glucose or ketones, blood urea nitrogen, creatine, potassium, sodium, alanine and aspartate aminotransferases, lactic acid dehydrogenase, and lipid profile. Clinical signs of malnutrition and poor control need to be recognized and addressed. Adequacy of intake and compliance need to be assessed based on established goals individualized for each patient.

**insulin resistance** An impaired biological response to either exogenous or endogenous insulin; plays a role in the etiology of type 2 diabetes.

**glycosylated hemoglobin (HbA1c)** A blood test that can reflect blood glucose control over a 3-month period. An A1c of 6% reflects an average plasma glucose level of about 120 mg/dL. In general, each 1% increase in A1c is a reflection of an increase in average glucose levels of about 30 mg/dL.

### Evidence Analysis for Evidence-Based Practice

Although specific evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications exist, they should be individualized based on each patient's usual intake and eating habits, metabolic profile, treatment goals, and desired outcomes (American Diabetes Association, 2008). The nutritional principles and recommendations are based on the following goals of medical nutrition therapy for diabetes as outlined in the 2008 American Diabetes Association position statement on the goals of MNT that apply to individuals with diabetes:

- To achieve and maintain blood glucose levels in the normal range or as close to normal as is safely possible
- A lipid and lipoprotein profile that reduces the risk for vascular disease

- Blood pressure levels in the normal range or as close to normal as is safely possible
- To prevent, or at least slow, the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle
- To address individual nutrition needs, taking into account personal and cultural preferences and willingness to change
- To maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence

In terms of specific dietary recommendations, evidence exists regarding the following:

- *Carbohydrates:* The total amount of carbohydrate in the meal or snack should be considered along with the source or type. The glycemic index was developed to measure a consistent amount of carbohydrate-containing foods (50 g) compared to a reference value (white bread or glucose) to measure postprandial response for each of the carbohydrate-containing foods tested. Whole grains, fruits, vegetables, and low-fat milk are healthy low-glycemic food choices. Research has shown that people with diabetes who regularly consume a high-glycemic-index diet could on average reduce A1c by 0.4% from consuming a low-glycemic-index diet (Brand-Miller et al., 2006). Sugar (sucrose) does not increase blood sugar levels any more than the same kilocalorie level of starch-containing food and as such does not need to be restricted in people with diabetes. If sucrose is consumed, it must be substituted for other carbohydrate sources. Nonnutritive sweeteners have been shown to be safe when consumed within the acceptable daily intake levels established by the U.S. Food and Drug Administration.
- *Protein:* There is no evidence to suggest that usual protein intakes (15% to 20%) need to be modified if renal function is not impaired.
- *Dietary fat:* Given the comorbidity of cardiovascular disease, a heart-healthy diet is warranted. Recommendations include less than 10% of total kilocalories from saturated fat, and for individuals with low-density lipoprotein cholesterol more than 100 mg/dL, it is recommended that saturated fat consumption be lowered to less than 7% of total kilocalories. The recommendation for total cholesterol intake is less than 300 mg/day. For individuals with low-density lipoprotein cholesterol more than 100 mg/dL, cholesterol intake should be reduced to less than 200 mg/day.
- *Energy balance:* Weight loss, even modest weight loss, has been shown to improve insulin resistance and overall blood sugar control.
- *Fiber:* Research has shown that consuming a high-fiber diet (~50 g fiber/day) reduces glycemia in type 1 diabetes and glycemia, hyperinsulinemia, and lipemia in type 2 diabetes (Franz et al., 2002). Consuming such a high- fiber diet poses problems in terms of palatability, limited food choices, and gastrointestinal side effects. Increased fiber intake has been shown to be of benefit for people with diabetes; thus, fiber intake goals should be at least those set for the general population at 14 g/1,000 kcal (IOM, 2002).
- *Micronutrients:* There is no clear evidence to support vitamin and mineral supplementation in people with diabetes unless they have known underlying vitamin and mineral deficiencies.
- *Alcohol:* The same guidelines apply to people with diabetes regarding alcohol intake based on the *Dietary Guidelines* as for those who do not have diabetes. In addition to the fact that alcohol intake is limited to one drink for women and two drinks for men, to reduce the risk of hypoglycemia it is suggested that alcohol be consumed with food. (One drink is considered a 12-oz beer, 5-oz glass of wine, or 1.5 oz of 80-proof spirits.)
- *Older adults with diabetes:* Older adults require fewer kilocalories to meet their nutritional needs, and they are more likely to be undernourished. Caution should be used when prescribing weight-loss diets in this population.

**Learning Point** For women with diabetes, nutrition requirements during pregnancy and lactation are similar as those for women who do not have diabetes. The major difference is the focus on appropriate weight gain, maintaining normoglycemia, and the absence of ketones. If weight gain is excessive, modest energy restriction may be appropriate.

### Chronic Kidney Disease

Chronic kidney disease is the inability of the kidney to function normally. The primary functions of the kidney are to maintain homeostatic balance with respect to fluids, electrolytes, and organic solutes and to remove waste from the body through the production of urine. The kidney filters approximately 1,600 L of blood per day and produces 1 to 2 L of urine per day. Symptoms of chronic kidney disease include severe headache, **dyspnea**, failing vision, poor appetite, nausea and vomiting, abdominal pain, and mouth ulcers. Dialysis is postponed for as long as possible.

**dyspnea** Shortness of breath.

#### Nutritional Assessment

Nutritional assessment of the patient with chronic kidney disease is very similar to a standard nutritional assessment with some additional features based on the Kidney Dialysis Outcome Quality Initiative (K/DOQI) guidelines. In addition to evaluating the standard laboratory values available on admission, which would include albumin, the following laboratory values should be evaluated: serum prealbumin, predialysis serum creatinine, urea reduction ratio, serum bicarbonate, and lipid profile. The K/DOQI also recommends the use of the Subjective Global Assessment, a four-item seven-point scale, to monitor nutrition status changes and patients' anthropometric measurements using each patient as his or her own control.

#### Evidence Analysis for Evidence-Based Practice

Current clinical practice guidelines for nutritional care in chronic kidney disease are based on recommendations by the 2002 National Kidney Foundation K/QODI guidelines that can be accessed online (http://www.kidney.org/professionals/kdoqi). The guidelines are evidence-based with the rationale for each guideline explained and referenced based on extensive literature review evaluated for quality and strength (Beto & Bansal, 2004).

More recently, under the 2003 K/DOQI guidelines, chronic kidney disease has been classified into five stages based on **glomerular filtration rate** so as to diagnose and base care in a uniform manner (Beto & Bansal, 2004). Stages 1 to 4 are predialysis, and stage 5 requires replacement therapy (dialysis) to sustain life. Dialysis therapies mimic the function of the kidneys to the extent possible, exchanging high levels of circulating products such as urea, phosphorus, and potassium that the kidney would normally remove using osmotic pressure to cross a barrier membrane to be removed from the body. There are two types of dialysis treatment: hemodialysis and peritoneal dialysis. Hemodialysis is artificial filtering of blood by a machine that requires permanent vein access through a fistula and the dialysis fluid is similar to plasma. Peritoneal dialysis is artificial filtering of the blood by a hyperosmolar solution through the peritoneum.

**glomerular filtration rate** The quantity of glomerular filtrate formed per unit in all nephrons of both kidneys.

In clinical nutrition practice, intervention needs to be matched to the function of the kidney based on biochemical data and corresponding K/DOQI nutritional guidelines. This can be challenging because the function of the kidneys changes day to day and compliance with the diet can be difficult because of all the restrictions that must managed. The goals of nutritional intervention for stages 1 to 4 are to minimize tissue catabolism; maintain nutritional status, weight, appetite, electrolyte balance, and lean body mass; and postpone dialysis as long as possible. The specific nutrient recommendations based on K/DOQI guidelines for energy, protein, fat, and fluid are referenced in **Table 9.8**, and the nutrient guidelines for sodium, potassium, calcium, and phosphorus are referenced in **Table 9.9**.

### HIV/AIDS

AIDS is a disease characterized by the transmission of a retrovirus called the human immunodeficiency virus by the exchange of bodily fluids through sexual contact, infected blood, contaminated needles, or mother-to-child transmission. AIDS was first described by the Centers for Disease Control and Prevention in 1981 and included severe depression of cellular immunity accompanied by unusual **opportunistic infections** such as *Pneumocystis carinii* pneumonia, cytomegalovirus, or Kaposi's sarcoma (Gottlieb, Schanker, Saxon, Weisman, & Pozalski, 1981). AIDS is defined as an HIV infection with a CD4 cell count of 200 or less and the presence of at least one opportunistic infection, dementia, wasting syndrome, or malignant disease.

**opportunistic infection** Infection by an organism that would not ordinarily cause disease, but because of an impaired immune response the organism becomes pathogenic.

**TABLE 9.8** K/DOQI Nutritional Guidelines for Energy, Protein, Fat, and Fluid

| Kidney Function | Energy (kcal/kg/day) | Protein (g/kg/day) | Fat (% total kcal) | Fluid (mL/day) |
|---|---|---|---|---|
| Normal kidney function | 30–37 | 0.8 | 30–35 | Unrestricted |
| Stage 1 to 4 chronic kidney disease | 35 < 60 yr<br>30–35 > 60 yr | 0.6–0.75<br>50% HBV | * | Unrestricted with normal output |
| Stage 5 hemodialysis | 35 < 60 yr<br>30–35 > 60 yr | 1.2<br>50% HBV | * | 1,000 + urine output |
| Stage 5 peritoneal dialysis | 35 < 60 yr<br>30–35 > 60 yr<br>Include kcal from dialysate | 1.2–1.3<br>50% HBV | * | 1,500–2,000 monitored |
| Transplant | 30–35 initial<br>25–30 maintenance | 1.3–1.5 initial<br>1.0 maintenance | * | Unrestricted unless indicated |

* Patients at the highest cardiovascular risk should follow the NCEP Adult Treatment Panel III guidelines to the extent possible. HBV, high biological value.

Data from National Kidney Foundation Kidney Dialysis Outcome Quality Initiative.

**TABLE 9.9** K/DOQI Nutritional Guidelines for Sodium, Potassium, Calcium and Phosphorus

| Kidney Function | Sodium (mg/day) | Potassium (mg/day) | Calcium (mg/day) | Phosphorus (mg/day) |
|---|---|---|---|---|
| Normal kidney function | Unrestricted | Unrestricted | Unrestricted | Unrestricted |
| Stage 1 to 4 chronic kidney disease | 2,000 | Based on lab values | 1,200 | Based on lab values |
| Stage 5 hemodialysis | 2,000 | 2,000–3,000 (8–17 mg/kg/day) | < 2,000 from diet and medications | 800–1,000 |
| Stage 5 peritoneal dialysis | 2,000 | 3,000–4,000 (8–17 mg/kg/day) | < 2,000 from diet and medications | 800–1,000 |
| Transplant | Unrestricted; monitor medication effect | Unrestricted; monitor medication effect | 1,200 | Unrestricted unless indicated |

Data from National Kidney Foundation Kidney Dialysis Outcome Quality Initiative.

### Nutritional Assessment

No nationally accepted standard of care for HIV/AIDS exists, but evidence does exist that medical nutrition therapy plays a critical role in increasing energy, protein, and micronutrient intake among HIV-positive and AIDS patients. The nutritional assessment should be comprehensive and include an evaluation of weight in terms of percentage of usual weight. Anthropometric measurements are useful because many patients have multiple clinic visits and/or hospitalizations, making it possible to have numerous measurements over a period of time. Patients should be evaluated for **HIV wasting syndrome**, which is characterized by an unintentional loss of 10% body weight over 6 months, loss of 5% lean body mass within 6 months, body cell mass less than 35% and BMI less than 27 (men), body cell mass less than 23% and BMI less than 27 (women), and BMI less than 20 (Polsky, Kotler, & Steinhart, 2001). Biochemical assessment of nutrition-related laboratory values may reflect inflammatory response rather than nutritional status; however, metabolic abnormalities result in changes in organ or tissue function, leading to altered use, storage, and excretion of nutrients (Stambullian et al., 2007). Evaluation of dietary intake should include an assessment of usual intake and all factors that affect the ability to consume a well-balanced diet such as access to food, ability to shop and prepare

**HIV wasting syndrome** Catabolic condition; loss of body weight (body fat and muscle stores);very similar to cancer cachexia.

food, body image concerns, cultural practices, complementary and alternative medicine (CAM) therapies, socioeconomic issues, and housing status (ADA, 2010).

#### Evidence Analysis for Evidence-Based Practice

Studies indicate that weight determines outcome and mortality (Grunfeld & Feingold, 1992; Kotler, Tierney, Wang, & Pierson, 1989). Nutrient requirements need to be individualized for each patient and vary depending on health status. Keusch and Thea (1993) recommended that for determining energy requirements clinicians should use Basal Energy Expenditure (BEE) 1.3 for maintenance and BEE1.5 for weight gain. Additional kilocalories and protein need to be provided if fever is present, with energy requirements increasing by 13% and protein requirements increasing by 10% for every degree Celsius of temperature elevation above normal (Grunfeld & Feingold, 1992). Medical nutrition protocols for adults have been developed with the goals of achieving healthy body weight, body composition, and lab values (Fenton, Silverman, & Vazzo, 1998).

## Nutritional Issues of Epidemic Proportion

### Excessive Weight and Obesity

Excessive weight and obesity have reached epidemic proportions in the United States. The prevalence of obesity in the United States has increased at alarming rates in the last three decades but has begun to level off in the current decade (Kuczmarski, Flegal, & Campbell, 1994; Ogden, Carroll, Kit, & Flegal, 2012). Excessive weight is defined as a BMI greater than 25 but less than 30. People who have a BMI of 30 or higher are considered obese. Consider the following obesity data; although not in line with the life stage groups of young, middle, and older adults discussed earlier in the chapter, the data do differentiate between the first and second half of adulthood based on gender reported verbatim from the National Center for Health Statistics (Ogden et al., 2012):

- More than 35% of U.S. men and women were obese in 2009–2010. There was no significant difference in prevalence between men and women at any age. Overall, adults aged 60 and over were more likely to be obese than younger adults. Among men there was no significant difference in obesity prevalence by age. Among women, however, 42.3% of those aged 60 and over were obese compared with 31.9% of women aged 20–39.
- See **Figure 9.2**.
- Between 1999–2000 and 2009–2010, the prevalence of obesity increased among men but not among women. In 1999–2000, 27.5% of men were obese, and by 2009–2010 the prevalence had increased to 35.5%. Among women, 33.4% were obese in 1999–2000 with no significant change in 2009–2010 (35.8%). In 1999–2000, the prevalence of obesity was higher in women than in men. Between 1999–2000 and 2009–2010, the difference in the prevalence of obesity between men and women decreased so in 2009–2010, the prevalence of obesity in men was virtually

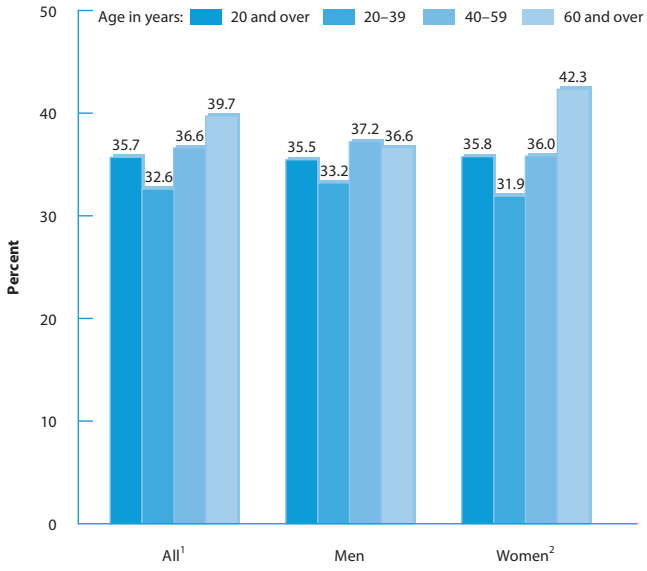



**Figure 9.2** Prevalence of obesity among adults aged 20 years and older, by sex and age: United States, 2009–2010.

[1] Significant increasing linear trend by age ($p < 0.01$). [2] Significant increasing linear trend by age ($p < 0.001$). Note that these estimates were age-adjusted by the first method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60+.

Courtesy of CDC/NCHS. Reproduced from Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). *Prevalence of obesity in the United States, 2009–2010*. NCHS data brief, no 82. Hyattsville, MD: National Center for Health Statistics.

equal to that in women (see **Figure 9.3**). There was no significant change in the prevalence of obesity from 2007–2008 to 2009–2010 overall or among men or women.

Research in obesity has been extensive in the last two decades and has helped build our understanding of the genetic, psychological, metabolic, and environmental influences on body weight, which has served to increase our awareness of the complexities of weight management (American Dietetic Association, 2009). The most-often-asked question is what is the cause? We live in an environment that is conducive to promoting obesity because of our sedentary lifestyles and the abundance of energy-dense foods (Allison, Fontaine, Manson, Steven, & Vanitallie, 1999; Wolf & Colditz, 1998). Data on weight-loss interventions do not produce long-term results and have a success rate of about 5% (National Institutes of Health & National Heart, Lung, and Blood Institute, 1998). What then is the role of the dietitian? The dietitian can play a critical role as a facilitator of change through the counseling process to help the patient formulate reasonable goals that can be met and sustained with a healthy eating approach. In terms of the nation as a whole, public policy must be changed and a battle must be waged on all fronts (i.e., the media, the food industry) if we are to win the war on obesity.

### Osteoporosis

Osteoporosis, often called the "silent epidemic," is a disease that takes a lifetime to develop and consequently a lifetime to prevent. It is characterized by a decrease in bone mass and deterioration of bone tissue with no outward signs or symptoms until late in the disease when bones become much more fragile and are apt to break. Osteoporosis affects 10 million Americans annually, 80% of whom are women; women are four times more likely than men to develop osteoporosis (Kaunitz, 2000). However, men can develop osteoporosis; in fact, one-third of men by the age of 75 years develop osteoporosis (Nutrition Screening Initiative, 2002). Osteoporosis leads to 1.5 million fractures every year in the United States, most commonly of the vertebrae. Visible signs of osteoporosis are altered posture caused by deformity of the spine, such as increased thoracis kyphosis (humpback) described as a "widow's hump," or postural slumping because of acute pain and loss of height as a result of vertebral collapse fractures. Risk factors associated with osteoporosis include (1) insufficient calcium intake, (2) vitamin D deficiency, (3) sedentary lifestyle, (4) smoking, (5) excessive alcohol consumption, (6) family history of fractures, and (7) a small, slender body, fair skin, and a Caucasian or Asian background—but all ethnic groups can

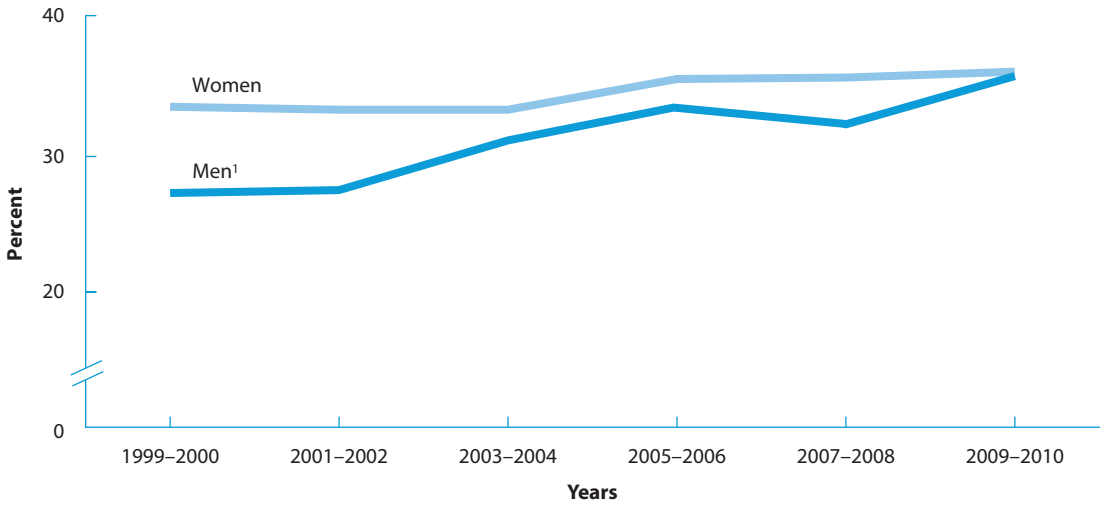



**Figure 9.3** Trends in the prevalence of obesity among adults aged 20 years and older, by sex: United States, 1999–2010.

[1] Significant increasing linear trend 1999–2000 to 2009–2010 ($p < 0.0001$). Note that these estimates were age-adjusted by the first method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60+.

Courtesy of CDC/NCHS. Reproduced from Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). *Prevalence of obesity in the United States, 2009–2010*. NCHS data brief, no 82. Hyattsville, MD: National Center for Health Statistics.

be affected (American Academy of Orthopaedic Surgeons [AAOS], 2009).

Controllable measures an individual can take to avoid osteoporosis are to ensure adequate consumption of calories and to take in enough calcium and vitamin D to promote optimum bone development in the first three decades of life. Many young adults, especially women, do not consume an adequate amount of calcium and may benefit from calcium and vitamin D supplementation. Calcium and vitamin D supplementation may be beneficial for people who have a family history of osteoporosis or who are exhibiting early signs of osteoporosis. Vitamin D deficiency has been reported in elderly free-living men and women (40–100%) and postmenopausal women (50%) (AAOS, 2009). Weight-bearing exercise has been shown to be especially beneficial in helping to reduce the loss of bone mass that happens as a natural part of the aging process. More and more evidence supports the role of antiresorptive therapy, which includes hormonal therapy (estrogen replacement therapy during menopause), bisphosphate therapy (Fosamax [alendronate sodium] or Actonel [risedronate sodium]), selective estrogen receptor modulators (Evista [raloxifene]), and calcitonin (Miacalcin), in reducing the incidence of osteoporosis (National Institutes of Health Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Treatment, 2001; Nutrition Action Healthletter, 2002). The American Academy of Orthopaedic Surgeons (AAOS) recommends that osteoporosis become a national health priority to minimize healthcare costs, morbidity, and mortality from the increasing number of osteoporotic fractures given our aging population (Lane & Nydick, 1999).

## Summary

Although genetic predisposition determines longevity to a degree, each person can choose to live a longer, healthier life by eating healthier and exercising. Nutrigenomics is a new and upcoming field that encompasses the study of nutrition and genetics. Thanks to the Human Genome Project, nutritional science is on the edge of a new frontier (Collins & McKusick, 2001). It may be possible someday to receive a nutrition prescription based on your genetic profile with nutrition recommendations that reflect the latest scientific evidence utilizing evidence-based analysis and practice guidelines. Nutrition experts have much work ahead if we are going to affect the obesity and osteoporosis epidemics in this country. At the same time, we must pay close attention to what is fast becoming the next epidemic—type 2 diabetes. Although each person must take personal responsibility for the choices he or she makes, including food choices and how much to exercise, it is up to us as professionals to interpret the latest nutrition research and translate this information for the public and advocate for public policy that promotes an environment conducive to a healthier life.

### Case Study 1

**Type 2 Diabetes Mellitus**

Jennifer L. Bueche, PhD, RD, CDN

Mary Smith is a 72-year-old female admitted to the ICU complaining of thirst, frequent urination, and feeling unusually tired. She states that the symptoms have worsened over the last few days. She thought she might have a bladder infection. She doesn't remember the last time that she ate but complains that her appetite is very poor, food tastes funny, and she feels really nauseous. She thinks she has lost nearly 10 lb in the last week or two. Lab work upon admission revealed a glucose level of 661 with an A1cof 11.2%. Mrs. Smith was diagnosed with diabetic ketoacidosis (DKA), given IV fluids with electrolytes, and placed on an insulin drip. A no-concentrated-sweets no-added-salt (NCS NAS) diet was ordered.

Patient Information:
DOB: 8/10/1940 Age: 72 Sex: Female
Occupation: Retired school teacher
Ethnic Background: Caucasian
Religious affiliation: Roman Catholic
Marital status: Married, lives with 78-year-old husband in single-family home
Primary care physician: Dr. Maria Wright

**Patient History:** Mrs. Smith is a relatively healthy 72-year-old and is the primary caregiver to her 78-year-old husband, who is in poor health. They still live in the home they purchased when they first got married but have considered on numerous occasions moving to a "retirement community." Mrs. Smith's medical history includes the following:

PMH: Essential HTN – Dx 2 years ago; occasional bladder infections, arthritis
Meds: Capoten 50 mg bid, ibuprofen prn
Smoker: No
Family Hx: Coronary artery disease, diabetes type 2
Physical Exam:
General Appearance: Pale, tired-looking elderly Caucasian female
Vitals: Temp 99.1°F, BP 100/68 mm Hg, HR 88 bpm

(*continued*)

Height/Weight: 5 foot4 inches, 184lb (83.6kg), BMI 31.5, class 1 obesity

Heart: Increased heart rate with normal rhythm

HEENT:

Head: normocephalic

Eyes: wears glasses for myopia

Ears: tympanic membranes normal

Nose: dry mucous membranes without lesions

Throat: dry mucous membranes without exudates or lesions

Genitalia: Normal without lesions

Neurologic: Confused, normal gait, normal reflexes

Extremities: Normal muscle tone for age, arthritis evident in hands and feet

Skin: Warm and dry

Chest/lungs: Respirations elevated, clear lung sounds, fruity odor on breath

Peripheral vascular: Peripheral pulses palpable

Abdomen: Nontender without masses or organomegaly

**Nutrition history:**

Usual dietary intake:

Breakfast: white toast w/jelly, orange juice, tea w/sugar

Lunch: sandwich (variety) and soup (from can), 2–3 cookies, and tea w/sugar

Dinner: casserole (meat/starch), tossed green salad with Italian dressing, dessert (1 slice cake or 1 scoop icecream), and tea w/sugar

Mrs. Smith has no food allergies, purchases and prepares all food herself for her and her husband, and does not take any dietary supplements. She finds casseroles easiest to prepare for dinner, so she can double the recipe and freeze for nights that she doesn't have time to cook because of caring for her husband, who is in poor health. She does not add salt to food.

Treatment plan:

- Normalize blood glucose levels.
- Initiate diabetes education based on new T2DM diagnosis protocol (self-management of diet and meal planning, blood sugar levels, signs/symptoms and treatment of hypo- vs. hyperglycemia, appropriate exercise).

### Questions

1. What is DKA and what are the signs and symptoms?
2. What nutrition-relevant information would you gather in conducting an initial nutritional assessment for Mrs. Smith?
3. Based on the nutrition assessment data gathered, which nutrition diagnosis or diagnoses might be considered for Mrs. Smith?
4. What is the "best fit" problem etiology statement (PES)?
5. What is the role of the registered dietician in the treatment of type 2 diabetes mellitus?

## Case Study 2

### Management of End-Stage Renal Disease

Jeannine Lawrence, PhD, RD, LD

Mr. R is a 53-year-old African American male who lives with his wife of 15 years and two teen-aged children. He has been receiving hemodialysis (HD) for 2 years because of end-stage renal disease (ESRD) secondary to previously uncontrolled hypertension. He has recently undergone peritoneal catheter implantation and has been referred for nutrition counseling prior to beginning peritoneal dialysis (PD).

**Current medical diagnoses:** Hypertension, dyslipidemia

**Anthropometrics:** Height 5 foot 11 inches; weight 206.8lb; waist circumference 41 inches

**Current medications:** Norvasc (amlodipine; a dihydropyridine calcium channel blocker) and Lescol (fluvastatin; a HMG-CoA reductase inhibitor)

**Lab values:** Blood pressure 112/75 mmHg; total cholesterol 190 mg/dL; LDL-C 125 mg/dL; HDL-C 30 mg/dL; triglycerides 175 mg/dL; albumin 3.9 g/dL; UOP 250 mL/day

### Questions

1. A successful transition from HD to PD can have a dramatic effect on a patient's daily activities and quality of life. Describe some of these positive effects your patient can expect.
2. What is the leading cause of mortality in patients with ESRD? How will this affect your dietary counseling?
3. Using the current K/DOQI Nutrition Guidelines, calculate energy, protein, and fluid requirements for this patient once PD is established. How will these requirements differ from his previously recommended intakes when receiving HD?
4. Evaluate the recommendations for Na+, K+, Ca++, and Phos for patients receiving HD and PD. Which recommendations will change once your patient transitions to PD? Why?

**Nutrition Follow-Up:** At his visit with you 6 months later, Mr. R reports that his appetite has decreased drastically since beginning PD. Despite the additional glucose calories he absorbs from his daily dialysate infusions, he has experienced marked unintentional weight loss. Current weight: 190.2lb; albumin 3.3 g/dL.

### Questions

1. Describe why PD can negatively affect appetite in some patients. What recommendations would you suggest to Mr. R to offset these effects and help to improve his dietary intake?

## Case Study 3

### Body Weight and Lipids in HIV Infection

Ben Atkinson, MS, RD, CD

Mr J is a Caucasian 54-year-old man and a nonsmoker. He was diagnosed with HIV after routine testing at a bathhouse, with risk factors of unprotected sex with partners of unknown HIV status and IV drug use. He is recently sober from years of methamphetamine use, of which he states they "trashed his mind and body." He states that his HIV diagnosis motivated him to change his lifestyle.

On his first visit he weighs 62kg (136 lb), height is 168cm (66 inches), and he has a BMI of 22. His abdominal circumference is 81cm (31 7/8 inches), midarm circumference is 27.6cm (10 7/8 inches), and maximal calf circumference is (13 3/8 inches). He would like to gain 6kg (13.2 lb) to weigh 68kg (150 lb) but states that he doesn't want to "look fat."

His appetite is increasing, after being sober from methamphetamines. He states that when he was using these drugs, he wouldn't eat during entire days, and then later would binge on high-calorie sugary foods. His 24-hour dietary recall reveals the following:

- Breakfast: 2 eggs fried in butter, 1 strip fried bacon, 1 cream cheese pastry, 12 oz of 2% milk, orange juice, and coffee
- Snack: None
- Lunch: 2 whole sandwiches made of white bread, ham, lettuce, mustard, mayo, and cheese. One 1-oz bag of regular potato chips, 1 regular size Snickers bar, and 12 oz of 2% milk
- Snack: None
- Dinner: Two pieces of fried chicken with the skin on, 2 cups of green salad, 3 Tbs of regular Ranch dressing, and 12oz of 2% milk
- After-dinner snack: Two whole English muffins with 1 tsp of butter each, and 6 oz of plain tea

He will also start highly active antiretroviral therapy (HAART, or HIV medication) today, consisting of Truvada (emtricitabine and tenofovirdisoproxilfumarate), atazanavir, and ritonavir. He plans to take these pills with his after-dinner snack. The package inserts for these medications state that "Coadministration of a single 300-mg dose of atazanavir and a 100-mg dose of ritonavir with a light meal (336 kcal, 5.1 g fat, 9.3 g protein) resulted in a 33% increase in the area under the curve (AUC) and a 40% increase in both the maximum concentration (Cmax) and the 24-hour concentration of atazanavir relative to the fasting state" (from http://packageinserts.bms.com/pi/pi_reyataz.pdf).

Two years later, Mr J returns with a referral to see the dietitian for a lipid-lowering diet. His blood pressure isn't elevated, and he has no family history of premature cardiovascular disease. His most recent lipid labs are (all in mg/dL):

Total cholesterol: 236
Triglycerides: 512
LDL cholesterol: Unable to calculate
HDL cholesterol: 25
Non-HDL cholesterol: 211

#### Questions

1. Would his desire to gain weight to 68kg keep him in the healthy BMI range or put him in the overweight range?
2. Did you expect that the patient's body weight would increase?
3. Does the food that he eats when he takes his HAART improve their function?
4. What are likely contributors to his increasing lipids, and what should his lipid values be?

## Issues to Debate

1. What can the dietetics profession do to help people make better lifestyle choices?
2. How can some of these ideas be made a practical reality?
3. What is the responsibility of corporate America in keeping people healthy?

## References


Allison, D. B., Fontaine, K. R., Manson, J. E., Steven, J., & Vanitallie, T. B. (1999). Annual deaths attributable to obesity in the United States. *Journal of the American Medical Association, 282*, 1530–1538.

American Academy of Orthopaedic Surgeons. (2009). Position statement: Osteoporosis/bone health in adults as a national health priority. Retrieved from www.aaos.org/about/papers/position/1113.asp

American Cancer Society. (2012). *Cancer facts and figures 2012*. Atlanta, GA: Author.

American Diabetes Association. (2002). Position statement: Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Journal of the American Dietetic Association, 102*, 109–120.

American Diabetes Association. (2008). Nutrition recommendations and interventions for diabetes: A position statement of the American Diabetes Association. *Diabetes Care, 31*(Supp1), S61–S78.

American Dietetic Association. (2000). ADA reports: Position of the American Dietetic Association: Nutrition, aging and the continuum of care. *Journal of the American Dietetic Association, 100*, 580.

American Dietetic Association. (2009). Position of the American Dietetic Association: Weight management. *Journal of the American Dietetic Association, 109*, 330–346.

American Dietetic Association. (2010). Position of the academy of nutrition and dietetics: food and nutrition for older

adults: Promoting health and wellness. *Journal of the Academy of Nutrition and Dietetics, 112*(8), 1255–1277.

American Dietetic Association & Canadian Dietetic Association. (1994). Nutrition intervention in the care of persons with human immunodeficiency virus infection. *Journal of the American Dietetic Association, 94,* 129–132.

Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., … Karanja, N. (1997). A clinical trial of the effects of dietary pattern on blood pressure. *New England Journal of Medicine, 336,* 1117–1124.

Berry, J. K., & Braunschwig, C. A. (1998). Nutritional assessment of the critically ill patient. *Critical Care Nursing Quarterly, 21,* 33.

Beto, J. A., & Bansal, U. K. (2004). Medical nutrition therapy in chronic kidney failure; integrating clinical practice guidelines. *Journal of the American Dietetic Association, 104,* 404–409.

Brand-Miller, J., Wolever, T., Foster-Powell, K., & Colagiuri, S. (2006). *The new glucose revolution*. New York, NY: Marlowe & Company.

Brandi, L. S., & Calafa, M. (1997). Indirect calorimetry in critically ill patients: Clinical applications and practical advice. *Nutrition, 13,* 349–358.

Brown, J. E., Isaacs, J. S., Krinke, U. B., Murtaugh, M. A., Sharbaugh, C., Stang, J., & Woldridge, N. H. (2005). *Nutrition through the life cycle* (2nd ed.). Belmont, CA: Wadsworth.

Centers for Disease Control and Prevention. (2002). Overweight and obesity. Retrieved from www.cdc.gov/obesity/adult/index.html

Centers for Disease Control and Prevention. (2011). *National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Charney, P., O'Sullivan Maillet, J., Touger-Decker, R., Splett, P., Meyers, E., & Haque, S. (2006). Reliability of nutrition diagnostic labels when used by registered dietitians at three levels of practice. *Journal of the American Dietetic Association*, *106*, A12.

Chin, A., Paw, M. J., DeJong, N., Schouten, E. G., Hiddink, G. J., & Kok, F. J. (2001). Physical exercise and/or enriched foods for functional improvement in frail, independent living elderly: A randomized controlled trial. *Archives of Physical and Medical Rehabilitation, 82*, 811–817.

Collins, F. S., & McKusick, V. A. (2001). Implications of the Human Genome Project for medical science. *Journal of the American Medical Association, 285*, 540.

Cowie, C. C., (2003). Prevalence of diabetes and impaired fasting glucose in adults, United States, 1999–2000. *Mortality and Morbidity Weekly Report, 52*(35), 833–837.

Elmore, M. F., Wagner, D. R., Knoll, D. M., Eizember, L., Oswall, M. A., Glowinski, E. A., … Rapp, P. A. (1994). Developing an effective adult nutrition screening tool for a community hospital. *Journal of the American Dietetic Association, 94*, 1113–1118, 1121.

Fenton, M., Silverman, E., & Vazzo, L. (1998). HIV/AIDS adult medical nutrition therapy protocol. In: J. Gilbreath, A. Inman-Felton, E. Q. Johnson, G. Robinson, & K. Smith (Eds.), *Medical nutrition therapy across the continuum of care*. Chicago, IL: American Dietetic Association.

Fields-Gardner, C., Fergusson, P., American Dietetic Association, & Dietitians of Canada. (2004). Position of the American Dietetic Association and Dietitians of Canada: Nutrition intervention in care of persons with HIV infection. *Journal of the American Dietetic Association, 104*, 1425–1441.

Flegal, K. M., Carroll, M. D., Ogden, C. L., & Johnson, C. L. (2002). Prevalence and trends in obesity among US adults, 1999–2000.*Journal of the American Medical Association, 288*, 1723–1727.

Fonesa, V., & Wall, J. (1995). Diet and diabetes in the elderly. *Clinical Geriatric Medicine, 11*, 613–624.

Food and Nutrition Board, Committee on Diet and Health, & National Research Council. (1989). *Diet and health: Implications for reducing chronic disease risk*. Washington, DC: National Academies Press.

Food and Nutrition Board, National Research Council, & National Academy of Sciences. (1989). *Recommended Dietary Allowances*. Washington, DC: National Academies Press.

Frankenfield, D., Roth-Yousey, L., & Compher, C. (2005). Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: A systematic review. *Journal of the American Dietetic Association, 105*, 775–790.

Franz, M. J., Bantle, J. P., Beebe, C. A., Brunzell, J. D., Chiasson, J. L., Garg, A., … Wheeler, M. (2002). Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care, 25,* 148–198.

Gallagher-Allred, C. R., Voss, A. C., Finn, S. C., & McCamish, M. A. (1996). Malnutrition and clinical outcomes: The case for medical nutrition therapy. *Journal of the American Dietetic Association, 96*, 361–367.

Gary, R., & Fleury, J. (2002). Nutritional status: Key to preventing decline in hospitalized older adults. *Topics in Geriatric Rehabilitation, 17*, 40–71.

Goldman, A., Hewitt, M., Collins, G. S., Childs, M., Hain, R., & United Kingdom Children's Cancer Study Group/Paediatric Oncology Nurses' Forum Palliative Care Working Group. (2006). Symptoms in children/young people with progressive malignant disease: United Kingdom Children's Cancer Study Group/Paediatric Oncology Nurses Forum survey. *Pediatrics, 117*, 1179.

Gottlieb, M. S., Schanker, H. M., Saxon, A., Weisman, J. D., & Pozalski, I. (1981). *Pneumocystis* pneumonia—Los Angeles. *Morbidity and Mortality Weekly Review, 30*, 250–252.

Gottschlich, M. M. (Ed.). (2007). *The A.S.P.E.N. nutrition support core curriculum: A case-based approach—the adult patient*. Silver Spring, MD: American Society for Parenteral and Enteral Nutrition.

Gray-Donald, K., Gibbson, L., Shapiro, S. H., Macklern, P. T., & Martin, J. G. (1996). Nutritional status and mortality in chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine, 153*, 961–966.

Grunfeld, C., & Feingold, K. R. (1992). Metabolic disturbances and wasting in the acquired immunodeficiency syndrome. *New England Journal of Medicine, 327*, 329–337.

Hanley, D. A., & Davison, K. S. (2005). Vitamin D insufficiency in North America. *Journal of Nutrition, 135*, 332–338.

Harvard Health Letter. (2002). Aging: Living to 100. What's the secret? *Harvard Health Letter, 27*, 1.

Hedley, A. A., Ogden, C. L., Carroll, M. D., Curtin, L. R., & Flegal, K. M. (2004). Overweight and obesity among US children, adolescents, and adults, 1999–2002.*Journal of the American Medical Association, 291*, 2847–2850.

Heron, M. (2011). Deaths: Leading causes for 2009. *National Vital Statistics Reports*,*61*(7).

Hu, G., & Cassano, P. (2000). Antioxidant nutrients and pulmonary function: The Third National Health and Nutrition Examination Survey. *American Journal of Epidemiology, 151*, 975.

Institute of Medicine. (2002). Dietary Reference Intakes: Energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academies Press.

Institute of Medicine Committee to Review Dietary Reference Intakes for Vitamin D and Calcium. Ross, A. C., Taylor, C. L., Yaktine, A. L., et al. (Eds.). (2011). *Dietary Reference Intakes for calcium and vitamin D*. Washington, DC: National Academies Press.

Ireton-Jones, C. J. (1997). Why use predictive equations for energy expenditure assessment? *Journal of the American Dietetic Association, 9*, A44.

Jenkins, M., Meyers, E., Charney, P., & Escott Stump, S. (2006). American Dietetic Association's standardized nutrition language: Project logic model and current status. *Studies in Health Technology and Informatics*, *122*, 710–714.

Kant, A. K. (2000). Consumption of energy-dense nutrient-poor foods by adult Americans: Nutritional and health implications. The third National Health and Nutrition Examination Survey, 1984–1994. *American Journal of Clinical Nutrition, 72*, 929.

Kaunitz, A. M. (2000). Osteoporosis: The silent epidemic. *Jacksonville Medicine, 51*(5).

Kelly, K. (2003). The "freshman 15." *U.S. News and World Report, 135*, 54.

Keusch, G. T., & Thea, D. M. (1993). Malnutrition in AIDS. *Medical Clinics of North America, 77*, 795.

Kitabchi, A. E., Umpierrez, G. E., Miles, J. M., & Fisher, J. N. (2009). Hyperglycemic crises in adult patients with diabetes. *Diabetes Care*, *32*(7), 1335–1343.

Klipstein-Grobusch, K., Reilly, J., Potter, J., Edwards, C., & Roberts, M. (1995). Energy intake and expenditure in elderly patients admitted to hospital with acute illness. *British Journal of Nutrition, 73*, 323–324.

Kochanek, K. D., Xu, J. Q., Murphy, S. L., Miniño, A. M., & Kung, H. C. (2011). Deaths: Final data for 2009. *National Vital Statistics Reports*, *60*(3).

Kotler, D. P., Tierney, A. R., Wang, J., & Pierson, R. N. (1989). Magnitude of body cell mass depletion and the timing of death from wasting in AIDS. *American Journal of Clinical Nutrition, 50*, 444–447.

Kuczmarski, R. J., Flegal, K. M., & Campbell, S. M. (1994). Overweight prevalence and trends for children and adolescents: The National Health and Nutrition Examination Surveys, 1963 to 1991. *Journal of the American Medical Association, 149*, 1085–1092.

Laaban, J. P., Kouchakji, B., Dore, M. F., Orvoen-Frija, E., David, P., & Rochemaure, J. (1993). Nutritional status of patients with chronic obstructive pulmonary disease and acute respiratory failure. *Chest, 103*, 1362–1368.

Lacey, K. P. E. (2003). Nutrition Care Process and Model: ADA adopts road map to quality care and outcome management. *Journal of the American Dietetic Association, 103*, 1061–1072.

Lane, J.M., & Nydick, M. (1999). Osteoporosis: Current modes of prevention and treatment. *Journal of the American Academy of Orthopaedic Surgeons*, *7*(1), 1931.

Levitsky, D. A., & Youn, T. (2004). The more food young adults are served, the more they overeat. *Journal of Nutrition, 134*, 2436–2550.

Mahan, L. E. (2004). *Krause's food, nutrition, and diet therapy* (11th ed.). Philadelphia, PA: Saunders.

Martin, J. A., Hamilton, B. E., Ventura, S. J., et al.(2012). Births: Final data for 2010. *National Vital Statistics Reports*, *61*(1).

Myers, E. F., Pritchett, E., & Johnson, E. Q. (2002). Evidence-based practice guides vs protocols: What's the difference? *Journal of the American Dietetic Association, 101*, 1085–1090.

National Cancer Institute. (2012). Data sources for cancer trends progress reports. Retrieved from www.progressreport.cancer.gov/data-sources-matrix.pdf

National Center for Health Statistics. (2011). *Health, United States, 2011. With special feature on socioeconomic status and health*. Hyattsville, MD: Author. Retrieved from www.cdc.gov/nchs/data/hus/hus11.pdf

National Cholesterol Education Program. (1988). Report of the National Cholesterol Education Program Expert Panel on Detection, Education, and Treatment of High Blood Cholesterol in Adults. *Archives of Internal Medicine, 148*, 36–69.

National Cholesterol Education Program. (2001). Executive summary of the third report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Journal of the American Medical Association, 285*, 2486.

National Heart, Lung, and Blood Institute. (2013). What is COPD? Retrieved from www.nhlbi.nih.gov/health/health-topics/topics/copd

National Heart, Lung, and Blood Institute & National Institutes of Health. (2002). *Third report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Cholesterol in Adults (Adult Treatment Panel III)*. Washington, DC: National Institutes of Health.

National Institutes of Health & National Heart, Lung, and Blood Institute. (1998). Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults—the evidence report. *Obesity Research, 6*, 121.

National Institutes of Health Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Treatment. (2001). Osteoporosis prevention, diagnosis, and treatment. *Journal of the American Medical Association, 285*, 785.

Nielsen, S. J., Kit, B. K., Fakhouri, T., & Ogden, C. L. (2012).*Calories consumed from alcoholic beverages by U.S. adults, 2007–2010*. NCHS data brief, no 110. Hyattsville, MD: National Center for Health Statistics.

Nutrition Action Healthletter. (2002). Bare bones: How to keep yours strong. *Nutrition Action Healthletter,* 29.

Nutrition Screening Initiative. (2002). *A physician's guide to nutrition in chronic disease management for older*

*adults*. Retrieved from www.geriatria.unimore.it/PDF/Nutrizione%20nelle%20patologie%20croniche.pdf

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). *Prevalence of obesity in the United States, 2009–2010*. NCHS data brief, no 82. Hyattsville, MD: National Center for Health Statistics.

Olshansky, S. J., Ludwig, D. S., Passaro, D. J., Allison, D. B., Hershow, R. C., Butler, R., … Ludwig, D. S. (2005). A potential decline in life expectancy in the United States in the 21st century. *New England Journal of Medicine, 352*, 1138–1146.

Polsky, B., Kotler, D., & Steinhart, C. (2001). HIV-associated wasting in the HAART era: Guidelines for assessment, diagnosis and treatment. *AIDS Patient Care and STDs, 15*, 411.

Public Health Service. (1988). *The Surgeon General's report on nutrition and health: Summary and recommendations*. Pub. No. 88-50211. Washington, DC: Department of Health and Human Service, Public Health Service.

Rogers, R. M., Donahoe, M., & Constantino, J. (1992). Physiologic effects of oral supplemental feeding in malnourished patients with chronic obstructive pulmonary disease. *American Review of Respiratory Disease, 146*, 1511–1517.

Russell, R. (1997). New views on the RDAs for older adults. *Journal of the American Dietetic Association, 97*, 515–518.

Schoeller, D. A. (1999). Recent advances from application of doubly labeled water to measurement of human energy expenditure. *Journal of Nutrition, 129*, 1765–1768.

Schols, A. M. W. J., Mostert, R., Soester, P., Greve, L. H., & Wouters, E. M. F. (1989). Inventory of nutritional status in patients with COPD. *Chest, 96*, 247–249.

Schols, A. M. W. J., Soeters, P. B., Dingemans, A. M. C., Mostert, R., Frantzen, P. J., & Wouters, E. F. (1993). Prevalence and characteristics of nutritional depletion in patients with COPD eligible for pulmonary rehabilitation. *American Review of Respiratory Disease, 147*, 1151–1156.

Skipworth, R. J., Stewart, G. D., Dejong, C. H., Preston, T., & Fearon, K. C. (2007). Pathophysiology of cancer cachexia: Much more than host-tumour interaction? *Clinical Nutrition, 266*, 667.

Smith, R. (2003). Expanding medical nutrition therapy: An argument for evidence-based practices. *Journal of the American Dietetic Association, 103*, 313–314.

Soler-Cataluna, J. J., Sanchez-Sanchez, L., Martinez-Garcia, M. A., Sanchez, P. R., Salcedo, E., & Navarro, M. (2005). Mid-arm muscle area is a better predictor of mortality than body mass index in COPD. *Chest, 128*, 2108–2115.

Stambullian, M., Felieu, S., & Slobodianik, N. H. (2007). Nutritional status in patients with HIV infection and AIDS. *British Journal of Nutrition*, *98*(Suppl. 1), 140–143.

Thorsdottir, I., Eriksen, B., & Eysteinsdottir, S. (1999). Nutritional status at submission for dietetic services and screening for malnutrition at admission to hospital. *Clinical Nutrition, 18*, 15–21.

Thorsdottir, I., & Gunnarsdottir, I. (2002). Energy intake must be increased among recently hospitalized patients with chronic obstructive pulmonary disease to improve nutritional status. (Research and Professional Briefs). *Journal of the American Dietetic Association, 102*, 247.

Thorsdottir, I., Gunnarsdottir, I., & Eriksen, B. (2001). Screening method evaluated by nutritional status measurements can be used to detect malnourishment in chronic obstructive pulmonary disease. *Journal of the American Dietetic Association, 101*, 648.

Trials of Hypertension Prevention Collaborative Research Group. (1997). Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. *Archives of Internal Medicine*, *157*, 657.

U.S. Department of Health and Human Services. (2011). Healthy People 2020. Retrieved from www.healthypeople.gov/2020/default.aspx

Wardlaw, G. M., Hampl, J. S., & DiSilvestro, R. A. (2004). *Perspectives in nutrition* (6th ed.). New York, NY: McGraw-Hill.

Whittaker, J. S., Ryan, C. F., Buckley, P. A., & Road, J. D. (1990). The effects of refeeding on peripheral and respiratory muscle function in malnourished chronic obstructive pulmonary disease patients. *American Review of Respiratory Disease, 142*, 283–288.

Wolf, A. M., & Colditz, G. A. (1998). Current estimates of the economic cost of obesity in the United States. *Obesity Research, 6*, 97–106.

Worthington-Roberts, B. S. (1996). *Nutrition throughout the life cycle* (3rd ed.). St. Louis, MO: Mosby.

Wright, J. D., & Wang, C.-Y. (2010). Trends in intake of energy and macronutrients in adults from 1999–2000 through2007–2008. NCHS data brief, no 49. Hyattsville, MD: National Center for Health Statistics.

Writing Group of the Nutrition Care Process/Standardized Language Committee. (2008a). Nutrition Care Process and Model part 1: The 2008 update. *Journal of the American Dietetic Association*, *108*, 1113–1117.

Writing Group of the Nutrition Care Process/Standardized Language Committee. (2008b). Nutrition Care Process part II: Using the international dietetics and nutrition terminology to document the Nutrition Care Process. *Journal of the American Dietetic Association*, *108*, 1287–1293.

Worthington-Roberts, B. S. (1996). *Nutrition throughout the life cycle* (3rd ed.). St. Louis: Mosby.

CHAPTER 10

# Special Topics in Adults and Chronic Diseases: Nutrition and Public Health

*Judith Sharlin, PhD, RD, and Amy Sheeley, PhD, RD, LDN*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Identify the primary causes of death and disability in adults in the United States.
2. Describe primary, secondary, and tertiary levels of health prevention and health promotion and their relationship to nutrition program planning.
3. Identify risk factors for chronic diseases and their implication for nutrition.
4. Describe the dietary risk factors associated with the leading chronic diseases.
5. Discuss the common features of the dietary guidelines issued by the major U.S. health organizations.
6. Compare different dietary interventions a community or public health nutritionist/dietitian offers to the community, family, or an individual at risk.
7. Recognize the mission and role of public health nutrition in preventing disease and promoting adult health.
8. Name some of the public health nutrition programs that exist today for maintaining adult health.

## Preventing Disease and Promoting Health

Growing scientific evidence reveals that nutrition or dietary intake in adults contributes significantly to preventing illness and premature death in the United States (Danaei et al., 2009). Life expectancy has increased dramatically over the past 100 years, and today the average life expectancy is at a record high of 78.5 years (National Center for Health Statistics [NCHS], 2012). Health and nutrition programs for adults aim at prevention and improving quality of life. Chronic diseases, however, account for 7 out of every 10 deaths in the United States (NCHS, 2012). It is estimated that nearly 1 in 2 adults live with at least one chronic condition (National Center for Chronic Disease Prevention and Health Promotion [NCCDPHP], 2009). The increasing cost of crisis medical care and the growing economic burden provide a cost-effective incentive for individuals and our nation to prevent chronic disease. Chronic diseases cost the United States approximately 75% of the $2.6 trillion spent on health care annually (Centers for Medicare and Medicaid Services [CMS], 2012). Despite this, only 3–8% of the total annual healthcare expenditures in our nation were spent on prevention (Miller, Roehrig, Hughes-Cromwick, & Lake, 2008). Nutrition interventions and policies target the factors related to adult health and aim to prevent chronic diseases that are the leading causes of death and disability.

## Chronic Diseases: The Leading Causes of Death and Disability

To successfully create programs to promote health, longevity, and quality of life, we must examine the leading causes of death and disability. Data from the National Vital Statistics report show that chronic diseases are the leading causes of death and disability (see **Table 10.1**) (NCHS, 2012). Cardiovascular diseases and cancer account for approximately half of all deaths in the United States (NCHS, 2012).

**Learning Point** The majority of chronic disease mortality can be attributed to lifestyle factors such as diet, which can be modified (NCCDPHP, 2009).

A report on the health status of U.S. adults emphasized that smoking continues to be a major problem for the population as well as an increasing number of people who are physically inactive and are overweight or obese (NCHS, 2011b). In a study comparing 19 high-income, industrialized countries, the United States ranked last for preventable death rates, with 96 preventable deaths per 100,000 (Nolte & McKee, 2011). Smoking and high blood pressure were found to be the leading causes of death, while physical activity and overweight and obesity were each responsible for every 1 in 10 deaths (Danaei et al., 2009). Although mortality rates from heart disease, stroke, and cancer have declined, poor diet and infrequent physical

**TABLE 10.1** Deaths and Percentage of Total Deaths for the 10 Leading Causes of Death: United States, 2008–2009

| Cause of Death and Year | Rank | 2009 Deaths | 2009 Percentage of Total Deaths | 2008 Deaths | 2008 Percentage of Total Deaths |
|---|---|---|---|---|---|
| All causes | N/A | 2,437,163 | 100.0 | 2,471,984 | 100.0 |
| Diseases of the heart | 1 | 599,413 | 24.6 | 616,828 | 25.0 |
| Malignant neoplasms | 2 | 567,628 | 23.3 | 565,460 | 22.9 |
| Chronic lower respiratory diseases | 3 | 137,353 | 5.6 | 141,090 | 5.7 |
| Cerebrovascular diseases | 4 | 128,842 | 5.3 | 134,148 | 5.4 |
| Accidents (unintentional injuries) | 5 | 118,021 | 4.8 | 121,902 | 4.9 |
| Alzheimer's disease | 6 | 79,003 | 3.2 | 82,435 | 3.3 |
| Diabetes mellitus | 7 | 68,705 | 2.8 | 70,553 | 2.9 |
| Influenza and pneumonia | 8 | 53,692 | 2.2 | 56,283 | 2.3 |
| Nephritis, nephrotic syndrome, and nephrosis | 9 | 48,235 | 2.0 | 48,237 | 2.0 |
| Intentional self-harm (suicide) | 10 | 36,909 | 1.5 | 36,035 | 1.5 |

Rank is based on number of deaths. N/A, category not applicable.

Reproduced from Heron, M. (2012). Deaths: Leading causes for 2009. *National Vital Statistics Report*, *61*(7), 9.

activity have contributed to more than 70% of men and more than 60% of women being overweight or obese (Flegal, Carroll, Ogden, & Curtin, 2010; Hoyert, 2012). Dietary factors are now associated with 4 of the 10 leading causes of death: coronary heart disease (CHD), some types of cancer, stroke, and type 2 diabetes (NCHS, 2011a). The *Dietary Guidelines for Americans 2010* states that "eating and physical activity patterns that are focused on consuming fewer calories, making informed food choices, and being physically active can help people attain and maintain a healthy weight, reduce their risk of chronic disease, and promote overall health" (USDA & U.S. Department of Health and Human Services, 2010).

### Risk Factors and Chronic Disease

Chronic diseases, although prevalent and costly, are among the most preventable. The World Health Organization (WHO) defines risk factors as "any attribute, characteristic or exposure of an individual that increases the likelihood of developing a disease or injury." The 2002 World Health Report states that individuals, populations, and government all share the responsibility to reduce risks to live a long and health life (World Health Organization [WHO], 2002). The modifiable behaviors of smoking, insufficient physical activity, poor diet, and excessive alcohol use are thought to be responsible for the majority of chronic diseases (NCCDPHP, 2009). There are five types of risk factors or determinants of health (U.S. Department of Health and Human Services [U.S. DHHS], 2010a):

1. Biological factors, such as an individual's genetic makeup, family history, age, or gender
2. Physical environment, such as the availability of safe walking paths
3. Social environment, such as income and education level
4. Access to quality health care
5. Individual behavior and lifestyle factors, such as smoking, exercise, and good eating habits

*Healthy People 2020* (U.S. DHHS, 2010b) provides the most recent guidelines and recommendations for specific health objectives and goals for the nation (U.S. DHHS, 2010b). Biological factors such as age or gender are not modifiable. However, the leading causes of death are associated with dietary factors, which are modifiable. The determinants of health illustrate how individual biology and behaviors influence health through the individual's social and physical environments (U.S. DHHS, 2010b). Finally, health can be improved through policies and interventions with access to quality

health care. Because the leading causes of illness and death in the United States, such as cardiovascular disease, some types of cancer, and type 2 diabetes, are associated with dietary factors, we must address the concern that chronic diseases have resulted from dietary excesses and imbalance (U.S. DHHS, 2010b). Furthermore, there is evidence that more intensive dietary counseling can lead to reduced intakes of dietary fat and cholesterol and increased fiber, fruit, and vegetable consumption, resulting in reduced disease risk (U.S. Preventative Services Task Force [USPSTF], 2011).

## Prevention Strategies

Health promotion and disease prevention provide complementary interventions to change health risk factors. Prevention efforts in public heath, community, and worksite settings are divided into three levels: primary prevention (health promotion), secondary prevention (risk appraisal and reduction), and tertiary prevention (treatment and rehabilitation) (American Dietetic Association, 2006; Edelstein, 2011; Shamansky & Clausen, 1980).

### Primary Prevention: Health Promotion

**primary prevention strategies** Encourage health-enhancing behaviors by giving individuals, families, and communities ways to reduce risk factors associated with disease and injury.

**Primary prevention strategies**, or health promotion, encourage health-enhancing behaviors by giving individuals, families, and communities ways to reduce risk factors associated with disease and injury (American Dietetic Association, 2006). Risk factors include environmental, economic, social, and biological aspects. Good examples of primary prevention strategies include worksite walking groups, environmental changes to provide nutritious choices in a school cafeteria vending machine, and nutrition labeling of restaurant menus (American Dietetic Association, 2006). Primary prevention strategies seek to expand the positive potential of health (Reddy & Katan, 2004).

**holistic approach** Individual lifestyle factors, social and political issues, and access to quality health care.

A **holistic approach**, embracing individual lifestyle factors, the environment, and economic, social, and political factors, can help communities and private sector partners achieve health promotion and disease prevention goals. Providing information, available on food labels and through health messages to the public, is one effective method. These messages encourage consumers to apply the *Dietary Guidelines*, the USDA MyPlate, and the Dietary Reference Intakes (Institute of Medicine [IOM], 2010; USDA, 2011; USDA & U.S. DHHS, 2010). Voluntary community and health organizations, the federal government, worksite programs, and schools can reach consumers daily to promote nutrition interventions.

The media remain effective means of communicating nutrition issues to the public (American Dietetic Association, 2007). Programs to promote physical activity and fitness, good nutrition, and smoking cessation must be broadly accessible. Worksite nutrition-centered health promotion and disease prevention programs provide opportunities to harness social support and influence (Goetzel & Ozminkowski, 2008).

Motivations should be provided for food processors and vendors, restaurant chefs, and school and worksite cafeteria managers to prepare and serve foods lower in fat, calories, and sodium. Finally, legislation and regulations can be ratified to endorse more complete food and nutrition labeling. Food labels that provide short, unequivocal, and easy to understand messages prove beneficial to consumers (Hawley et al., 2012).

### Secondary Prevention: Risk Appraisal and Risk Reduction

**secondary prevention** Risk appraisal and screening to emphasize early detection and diagnosis of disease.

**Secondary prevention** includes risk appraisal and screening to emphasize early detection and diagnosis of disease (American Dietetic Association, 2006; Elo & Caltrop, 2002; Shamansky & Clausen, 1980). Secondary prevention begins at the point where the pathology of a disease may occur. It encompasses diagnostic services that include screening, surveillance, and clinical examinations (Elo & Caltrop, 2002). Screening strategies include follow-up education, counseling, and health referral. Models for secondary prevention involve screening for early detection of cardiovascular problems, such as elevated blood pressure, dyslipidemia, and high glucose levels (O'Keefe, Carter, & Lavie, 2009). For people with an elevated blood cholesterol level, this means introducing therapeutic lifestyle changes, such as reducing total and saturated fat in the diet, increasing physical activity, and reducing or maintaining a healthy weight. If this is not effective, or if low-density lipoprotein levels are abnormally high, drug therapy can be recommended by the physician.

Strategies in secondary prevention are aimed at self-care for people with chronic diseases.

An example of a secondary prevention program involving self-care is diabetes self-management education (DSME) programs that are evidence-based and have been shown to improve patient outcomes (Funnell et al., 2009; Shamansky & Clausen, 1980).

### Tertiary Prevention: Treatment and Rehabilitation

**tertiary prevention**
Treatment and rehabilitation; reduction in the amount of disability caused by a disease to achieve the highest level of function.

**Tertiary prevention** involves treatment and rehabilitation and is defined as the reduction in the amount of disability caused by a disease to achieve the highest level of function (Pender, Murdaugh, & Parsons, 2010). Examples of tertiary diseases include diabetes, kidney disease, and angina. The goal of treatment and rehabilitation is the prevention of further disability and any secondary conditions that might result from the initial health problem.

Examples of tertiary prevention programs include medical nutrition therapy (MNT) for people suffering from kidney disease, nutrition education about vitamin and mineral supplementation and feeding strategies to prevent further complications of wasting from HIV/AIDS, and cardiac rehabilitation through diet, exercise, and stress management. The ultimate goal of tertiary prevention is, through rehabilitation, to restore the individual to an "optimal" level of functioning, given the constraints of the disease (American Dietetic Association, 2006).

### Implications of the Prevention Levels

The prevention levels are useful concepts to help set objectives for public health programs concerned with adult health in communities, worksites, and other settings. Recent findings underscore the need to emphasize population-based prevention programs (American Heart Association [AHA], 2008). As discussed earlier, research points to a holistic approach that aims at health and prevention when using the level concept for public health programs. This approach is embraced in *Healthy People 2020* in terms of the nutrition and health objectives (U.S. DHHS, 2010b). For each goal, the holistic approach involving individual lifestyle factors, environmental factors, social and political issues, and access to quality health care is addressed.

Public health endeavors focus on primary or secondary prevention. It is important to choose the appropriate prevention level when planning a public health program. This is illustrated with two different approaches for handling CHD risk factors: one intended to reduce the prevalence of high blood pressure and one to reduce obesity levels.

Almost all Americans have had their blood pressure measured sometime in their lives. Ninety percent of Americans have had their blood pressure measured in the past 2 years and could state whether it was normal or abnormally high (U.S. DHHS, Centers for Disease Control and Prevention [CDC], & NCHS, 2008), and more than 90% of Americans are aware of the relationship between hypertension and stroke and hypertension and heart disease (U.S. DHHS & National Heart, Lung, and Blood Institute [NHLBI], 2012). Almost 80% of people with hypertension know they have high blood pressure, yet less than half (45%) have their blood pressure under control (less than 140/90 mm Hg) (AHA, 2012a). Kannel, Dawber, and McGee (1980) showed that systolic (higher number) blood pressure is a more important predictor of heart disease than is diastolic blood pressure, especially in older adults. In addition, ethnic disparities exist for prevalence rates of high blood pressure, and African Americans in the United States have a greater prevalence of high blood pressure than do whites (AHA, 2012a). These data suggest that to reduce mortality from hypertension, secondary prevention strategies should be directed toward persons with hypertension (especially those with elevated systolic pressures), African Americans, and older adults. This would be more effective than primary prevention strategies aimed at increasing public awareness.

In contrast, many Americans are just beginning to be aware of the growing obesity epidemic and the significance of elevated body mass index (BMI) (30 and above) for the risk of death and illness (AHA, 2008). Americans understand the viability of lowering this risk factor associated with many chronic diseases. Extensive data exist to substantiate that those making lifestyle changes such as engaging in regular physical activity and healthy eating could, at the very least, halt the continued growth of the obesity epidemic (AHA, 2008). Given the dramatic increases in overweight and obese U.S. adults, population-based efforts could be more efficient at reducing the obesity epidemic than are efforts to identify those individuals at risk (Flegal et al., 2010).

One primary prevention effort, called America on the Move (USDA, National Institute of Food and Agriculture, 2012), is a national initiative dedicated to helping individuals and communities become more physically active and eat more healthfully. The program creates and supports

an "integrated grassroots network" at the state level to build communities that support individual behavior changes. In addition, America on the Move involves public and private partnerships at the national, state, and local levels. It publicizes small behavioral changes such as cutting 100 calories a day and taking 2,000 steps daily.

Health promotion and chronic disease prevention programs should focus on coalition building between community-based nutrition and health professionals, government, local businesses, health agencies, and insurers (American Dietetic Association, 2006). In this rapidly changing healthcare environment, continued training and research in public health program development is crucial to provide evidence of the efficacy of health prevention and promotion.

All levels of prevention should be addressed when devising strategies for dietary behavior changes for chronic disease prevention. However, for some diseases, the nutrition strategy may prove similar at each prevention level. Weight reduction, for example, may prevent the onset of hypertension or may be part of the treatment for type 2 diabetes. In the case of other diseases, such as cancer, nutrition strategies might vary with the prevention level.

## Dietary Guidelines for Disease Prevention

Various health organizations and government agencies have issued dietary guidelines and recommendations based on current scientific evidence. In 1989, the National Research Council published its report, *Diet and Health: Implications for Reducing Chronic Disease Risk*, providing evidence for the relationship between all major chronic conditions and diet (National Research Council Committee on Diet and Health, 1989). More recently, *Healthy People 2020* set forth a comprehensive health promotion and disease prevention program for the nation. *Healthy People 2020*'s comprehensive health agenda has two overarching goals: (1) to increase the quality and years of a healthy life and (2) to eliminate health disparities (U.S. DHHS, 2010b). These goals embrace the dietary recommendations set forth by several government and health organizations.

In 2010, the U.S. *Dietary Guidelines* were assessed in terms of surveillance and research needs, especially related to risk for chronic diseases, such as heart disease (Flock & Kris-Etherton, 2011). The dietary guidelines of the American Heart Association (AHA) give greater emphasis to the diet as a whole and, specifically, to certain protective foods for chronic disease risk prevention. Scientifically based dietary guidelines from the American Cancer Society (ACS) reinforce these guidelines and further emphasize eating foods from plant sources (American Cancer Society [ACS], 2012a). In addition, the Alternate Healthy Eating Index was developed to target food choices and nutrient intake associated with chronic disease risk (Guenther, Reedy, Krebs-Smith, & Reeve, 2008).

> Visit http://www.nap.edu for more information about the National Research Council's report, *Diet and Health: Implications for Reducing Chronic Disease Risk*.

The dietary guidelines from these major health organizations agree in their basic message. The American Cancer Society stated, "The ACS Guidelines are consistent with guidelines from the American Heart Association and the American Diabetes Association for the prevention of coronary heart disease and diabetes, as well as for general health promotion, as defined by the 2010 *Dietary Guidelines for Americans*" (ACS, 2012a).

The American Cancer Society, the American Diabetes Association, and the American Heart Association typically collaborate to work on health promotion and disease prevention (Eyre, Kahn, & Robertson, 2004). In their collaborative efforts these organizations are making unified health statements concerning the prevention of heart disease, cancer, diabetes, and related risk factors. Furthermore, these organizations agree on the following dietary patterns, which may help reduce the risk of chronic diseases (Eyre et al., 2004):

- Consume a diet that emphasizes whole grains and legumes, vegetables, and fruits.
- Decrease saturated fat and dietary cholesterol; limit red meat and full-fat dairy products.
- Limit intake of foods and beverages high in added sugars.
- Limit overall intake of calories and engage in regular physical activity to maintain a healthy body weight.

Despite the evidence of the importance of diet to health, vegetable and fruit consumption among adults continues to be below recommended amounts: Less than 15% of U.S. adults consume five servings a day (CDC, 2009). The majority of

the U.S. population did not meet the minimum recommendations of any of the MyPyramid nutrient-rich food groups, except grains and meat and beans (Krebs-Smith, Guenthe, Subar, Kirkpatrick, & Dodd, 2010).

The 2010 *Dietary Guidelines for Americans* was released in January 2011 by the U.S. DHHS and the USDA. The dietary guidelines have consistently encouraged the consumption of complex carbohydrates and fiber by eating more fruits, vegetables, and whole grains. The 2010 recommendations addressed increasing consumption of plant foods (vegetables, cooked dry beans and peas, fruits, whole grains and nuts and seeds). The updated guidelines included two new chapters, "The Total Diet," which considers healthy dietary patterns, and "Translating and Integrating the Evidence," which addresses environmental and social changes. Other changes to the 2010 guidelines discuss eating behaviors such as snacking and fast food, and 8 oz of seafood consumption twice a week. In 2011, the MyPlate symbol replaced the former MyPyramid, to act as a practical tool to assist Americans in following the *Dietary Guidelines* (USDA, 2011). See **Figure 10.1**.

In summary, diverse health organizations stress the similarities in the dietary recommendations for reducing disease risk. These recommendations are to eat less saturated fat and cholesterol (limit red meat and full-fat dairy products); increase consumption of fruits, vegetables, whole grains, and legumes; limit added sugar and sodium; drink alcohol in moderation; and increase physical activity. By using clear terms, public health nutrition and dietetic professionals can make the nutrition message easier for the public to understand, value, and implement.

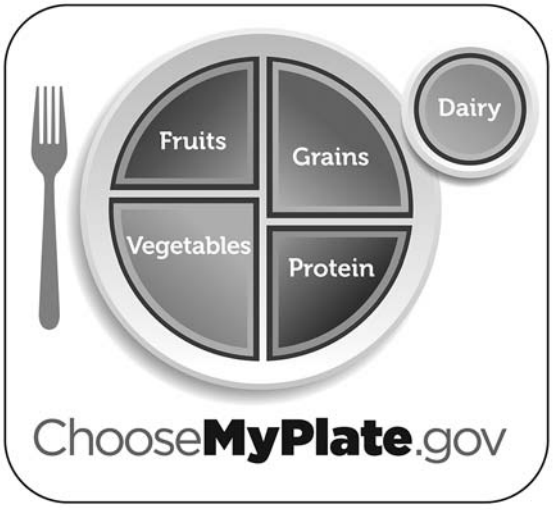



**FIGURE 10.1** MyPlate.gov.

Courtesy of USDA Center for Nutrition Policy and Promotion.

## Diet and Health: Nutrition Strategies and Risk Factors

Nutrition strategies are essential in the prevention and management of several chronic diseases and their risk factors.

**Learning Point**

When developing a public health program, risk factor assessment needs to be addressed. The following criteria can be assessed:

- The risk factor must have a strong association with the development of a chronic disease (e.g., obesity and heart disease).
- The risk factor must affect a significant number of people.
- The risk factor must be modifiable, so it can be reduced or changed.
- The risk factor must have a modification that, when changed or reduced, results in decreased mortality.

Because many risk factors can be modified by healthy lifestyle changes, early recognition of the risk factors for chronic disease prevention is critical.

### Obesity

Obesity has reached epidemic proportions in the United States. In 2009–2010, nearly 70% of adults were overweight or obese (NCHS, 2011b). Rates of excessive weight and obesity are steadily growing in our country. Nearly 55% of the U.S. adult population was overweight or obese in 1988–1994, compared with 46% in 1976–1980 (U.S. DHHS, 2010d). The National Institutes of Health (NIH) Expert Panel uses BMI for defining excessive weight and obesity (National Heart, Lung, and Blood Institute [NHLBI], 1998). The cut-off point for *overweight* status is a BMI of 25. *Obesity*, defined as having a BMI of 30 or greater, has doubled among adults since 1980. Many diseases are associated with excessive weight and obesity, including CHD, stroke, high blood pressure, diabetes, arthritis-related disabilities, sleep apnea, gallbladder disease, and some cancers (U.S. DHHS, 2010d).

The public health burden of excessive weight and obesity is overwhelming in terms of premature deaths and disability, lost productivity, and social stigmatization (U.S. DHHS, 2010d). In 2008, the

total cost of obesity was estimated at $147 billion (Finkelstein, Trogdon, Cohen, & Dietz, 2009). Obesity rates are higher among certain population groups, such as Hispanic Americans, African Americans, Native Americans, and Pacific Islander American women (U.S. DHHS, 2010d). Research has reported, however, that overweight status has increased in *all* parts of the U.S. population (Flegal et al., 2010). The recent trend of overweight in the United States has become so severe that if it is not reversed in the next few years, an estimated 86% of adults will be overweight or obese by 2030, with about 50% obese (see **Figure 10.2**) (Wang, Beydoun, Liang, Caballero, & Kumanyika, 2008).

Many factors contribute to excessive weight and obesity. For each individual, metabolic and genetic factors, as well as behaviors affecting dietary intake and physical activity, contribute to being overweight. Cultural, environmental, and socioeconomic influences also play a role. Most overweight and obese individuals eat more calories from food than they expend through physical activity. As body weight increases, so does the prevalence of health risks in an individual. For this reason, encouraging obese individuals to adopt new eating and physical activity habits is of vital importance.

### Weight Management

The goals and outcomes of weight management programs should be guided by an assessment of an individual's weight (BMI) and health. The NHLBI guidelines recommend intervention for people who are overweight and have two or more risk factors associated with their weight (NHLBI, 1998). Furthermore, according to the position paper of the Academy of Nutrition and Dietetics

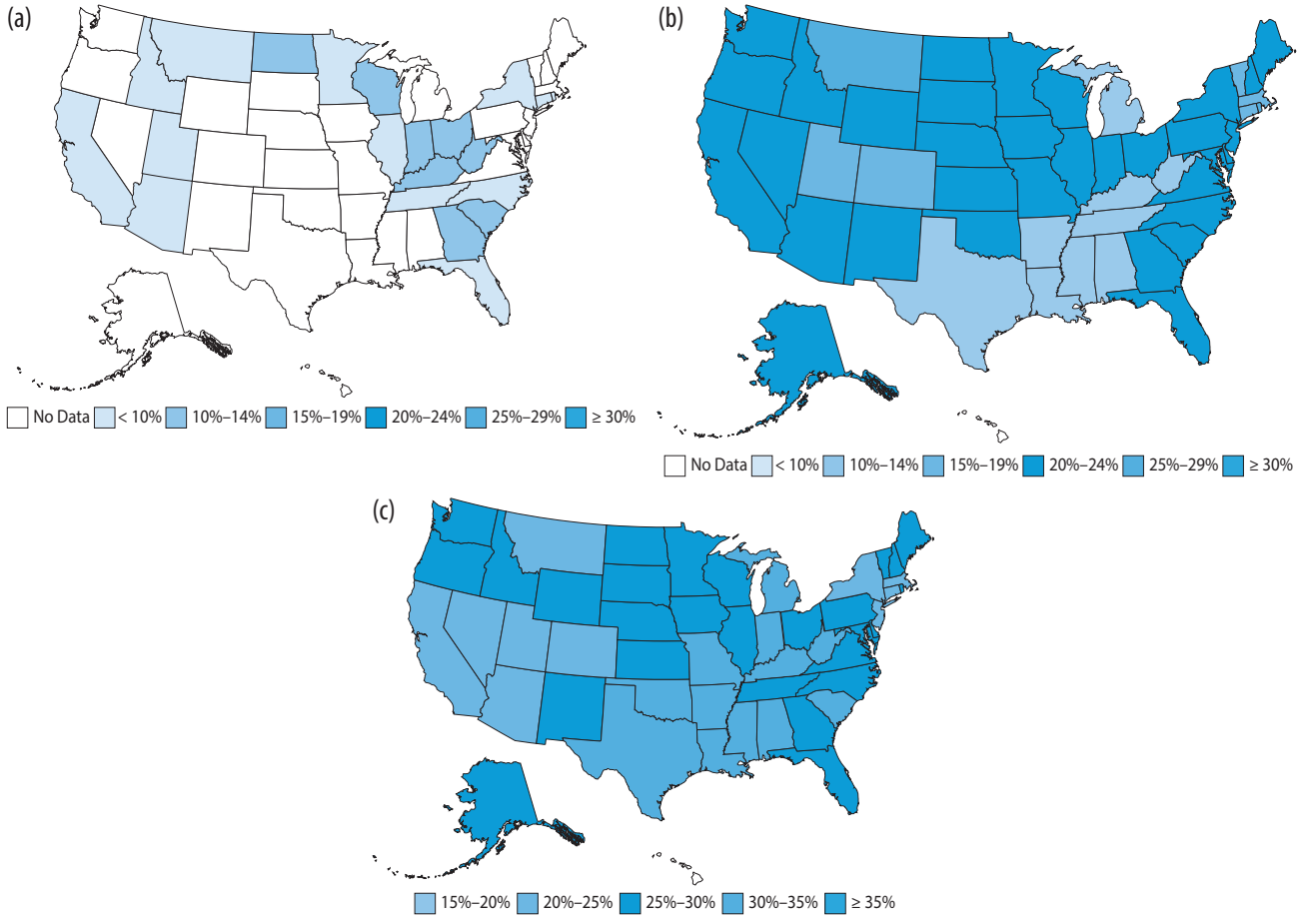



**FIGURE 10.2** Percentage of obesity (BMI > 30) in U.S. adults. (a) 1985. (b). 2004. (c) 2011.

Reproduced from Centers for Disease Control and Prevention. Overweight and obesity. Adult obesity statistics: Obesity prevalence in 2011 varies across states and regions. Retrieved from www.cdc.gov/obesity/data/trends.html

(American Dietetic Association, 2009) for weight management, assessment should incorporate the following areas:

- *Anthropometrics:* The assessment of height, weight, BMI, and waist circumference (as waist measurement increases, so do health risks)
- *Medical causes:* Identifying potential causes, age at onset, obesity-associated complications, and severity of obesity
- *Psychological causes:* Eating disorders, possible psychological causes, and barriers to treatment
- *Nutritional causes:* Weight history, diet history, current eating patterns, nutritional intake, environmental factors, meals eaten away from home, exercise history, and motivation to change

For an individual, the goal of a weight management program should focus on the prevention of weight gain as well as weight loss. This recommendation encourages individuals to adopt healthier lifestyles, such as increasing physical activity and choosing less calorically dense foods. With this approach, weight loss will lead to reductions of health risks (Mozaffarian, Hao, Rimm, Willett, & Hu, 2011). A weight loss of as little as 10% can improve health risks associated with being overweight and obese (NIH & NHLBI, 1998).

> Visit http://www.nhlbi.nih.gov for more information about the NHLBI's recommendations.

For effective weight management programs, a multidisciplinary team should be involved, including a physician, a dietitian, an exercise physiologist, and a behavioral therapist. Healthcare professionals should be especially dedicated and sensitive to the needs of overweight and obese individuals. It is appropriate to discuss realistic goals of weight loss and maintenance so shared responsibility for weight management can develop between the provider and individual. Goals might include the following (American Dietetic Association, 2009):

- Prevention or cessation of weight gain in an individual who is continuing to see an increase in his or her weight
- Progress in physical and emotional health
- Small maintainable weight losses achieved through sensible eating and exercise
- Improvements in eating, exercise, and any behaviors apart from weight loss

Establishing both short- and long-term treatment goals and documenting measures before implementing the weight management plan and after the individual has started on the plan are important goals of care. Positive behavior changes, other than absolute weight, should be rewarded because these can be very motivating.

Physical activity is highly recommended as an essential part of a weight management program. It is well established that to maintain weight loss, healthful dietary habits must be coupled with increased physical activity (U.S. DHHS, 2010d). Physical activity contributes to weight loss not only by changing energy balance but also by positively changing body composition by increasing lean body mass. Exercise decreases the risk of chronic disease and improves mood and quality of life. Many experts believe that physical inactivity is responsible for the increasing prevalence of excessive weight and obesity in the United States (U.S. DHHS & NHLBI, 2001). Combining weight loss with regular physical activity reduces one's risk for chronic diseases such as heart disease and diabetes by reducing both blood cholesterol and blood glucose levels.

The biggest challenge in weight management is to maintain a healthy weight once it is achieved. It is essential to include physical activity in weight loss programs because regular physical activity is one of the best predictors of weight maintenance (Donnelly et al., 2009). Unfortunately, long-term weight loss maintenance is very difficult for many individuals to achieve (Elfhag & Rössner, 2005). The NIH recommends that both dietary and physical activity changes need to be continued indefinitely for weight loss to be maintained (NIH & NHLBI, 1998). A comprehensive lifestyle program that focuses on nutrition, exercise, cognitive behavioral changes, and medical monitoring has been shown to be most effective for long-term success (Kruger, Blanck, & Gillespie, 2008).

Because of the increased prevalence in overweight and obesity in the United States, weight management becomes crucial for primary prevention of many chronic diseases. It is the foundation of secondary and tertiary prevention of hypertension, high blood cholesterol, diabetes, arthritis, and some cancers. It is important for healthcare providers to lobby for public health policies that endorse the treatment and management of weight. Also, clients must be informed of the known

healthy and positive outcomes achieved through weight management programs.

### Cardiovascular Disease

Cardiovascular disease is the nation's leading cause of death, accounting for close to 33% of all deaths in the United States. In 2008, more than 810,000 Americans died of cardiovascular disease (AHA, 2012a). Three modifiable health behaviors—poor nutrition, lack of physical activity, and smoking—contribute greatly to the burden of heart disease.

Cardiovascular diseases include diseases of the heart and blood vessels: CHD, stroke, and peripheral vascular diseases. CHD is the most common form of cardiovascular disease and usually involves atherosclerosis and hypertension. Atherosclerosis is characterized by the buildup of plaques along the inner walls of the arteries, causing inadequate blood flow and leading to serious cardiovascular problems.

The consequences of cardiovascular disease are usually heart disease and stroke; it is estimated that approximately every 25 seconds, someone in the United States will have a coronary event, and approximately every minute, someone will die of one (AHA, 2012a). About 83 million Americans live with the effects of heart disease and stroke. The annual cost of cardiovascular disease and stroke is estimated at almost $298 billion (AHA, 2012a). From 1998 to 2008, the death rate from cardiovascular disease fell by approximately 30% (AHA, 2012a). The decrease in the mortality rates from cardiovascular disease is attributed to primary prevention (i.e., a decrease in dietary intake of saturated fat), secondary prevention (i.e., early detection and treatment of hypertension), and improved medical and surgical treatments (AHA, 2012a). In general, mortality and prevalence rates of cardiovascular disease could be improved by reducing the major risk factors: high blood pressure, high blood cholesterol, tobacco use, physical inactivity, and poor nutrition. Controlling one or more of these risk factors could have a major public health impact in our country.

#### Hypertension

High blood pressure, or hypertension, remains a "silent killer" in the United States, affecting about 68 million, or 1 in 3, Americans (CDC, 2011e). High blood pressure is a major independent risk factor for cardiovascular disease. Hypertension increases the risk of heart attack, heart failure, stroke, and kidney disease (U.S. DHHS, NIH, NHLBI, &National High Blood Pressure Education Program [NHBPEP], 2004). This is significant because heart disease and stroke are, respectively, the first and fourth leading causes of death in the United States (NCHS, 2012).

In the latest classification of blood pressure for adults, normal blood pressure is considered to be less than 120/80 mm Hg, and prehypertension is designated as 120 to 139 systolic or 80 to 89 mm Hg diastolic pressure (U.S. DHHS, NIH, NHLBI, & NHBPEP, 2004). High blood pressure for adults is defined as a systolic pressure of 140 mm Hg or higher or a diastolic pressure of 90 mm Hg or higher. Studies have supported the finding that systolic blood pressure is a more important predictor of CHD in older adults than is diastolic blood pressure (NHLBI, 1997).

The number of people who were able to control their high blood pressure from lifestyle changes and the use of antihypertensive drugs rose from about 16% in 1971–1974 to about 81% in 2005–2008 (NHLBI, 2012). The age-adjusted death rate attributed to hypertension rose by about 20% over the past decade (data from 1998 to 2008), with the actual number of deaths increasing by approximately 50% (AHA, 2012a). Twenty-one percent of people with hypertension are on medication but are inadequately controlled; only 49% are on medication and well controlled (NHLBI, 2012). Significant disparities exist among persons diagnosed with hypertension; for example, African Americans have high blood pressure at an earlier age and in general have higher blood pressures (AHA, 2012a). Approximately 21% of Americans are unaware they have hypertension (NHLBI, 2012).

The latest guidelines for the treatment of hypertension are found in the *Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*; an update of this report is currently under development (U.S. DHHS, NIH, NHLBI, & NHBPEP, 2004). This report advocates major lifestyle modifications shown to lower blood pressure, enhance antihypertensive drug efficacy, and decrease cardiovascular risk. General lifestyle modifications include reducing weight for overweight or obese individuals (the goal is for a BMI of 18.5 to 24.9) (Harsha & Bray, 2008; U.S. DHHS, NIH, NHLBI, & NHBPEP, 2004), adopting of the

Dietary Approaches to Stop Hypertension (DASH) eating plan (Sacks et al., 2001), reducing sodium intake (AHA, 2012b), increasing physical activity (Whelton, Chin, Xin, & He, 2002), and moderating alcohol consumption (Sesso, Cook, Buring, Manson, & Gaziano, 2008). The DASH eating plan is a diet rich in calcium and potassium, consisting of fresh fruits, vegetables, and low-fat dairy products (Sacks et al., 2001; Vollmer et al., 2001). The DASH eating plan advocates a low sodium intake; intakes of 1,600 mg have been found to be as effective as single-drug therapy in lowering blood pressure (Sacks et al., 2001). Other specific lifestyle modifications include reducing sodium to 2,400 mg a day, engaging in regular aerobic physical activity at least 30 minutes a day (most days of the week), and limiting alcohol consumption (two drinks a day for men and one drink a day for women) (U.S. DHHS, NIH, NHLBI, & NHBPEP, 2004). The report's guidelines recommend these lifestyle modifications for all individuals. For those people who have not achieved the goal blood pressure (< 140/90 mm Hg), antihypertensive drugs, such as thiazide-type diuretics, may also be recommended (Psatsy et al., 1997).

**Learning Point** A usual food intake in the United States, complete with convenience and fast foods, may provide 10,000 to 20,000 mg of sodium per day. Visit http://www.nhlbi.nih.gov/guidelines/hypertension/ for more information about the *Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.*

### Cholesterol

High blood cholesterol is one of the major independent risk factors for heart disease and stroke (NIH & NHLBI, 2002). Modifying this risk factor is effective in reducing cardiovascular disease mortality. Animal, epidemiologic, and metabolic research shows that having elevated blood cholesterol levels is associated with cardiovascular disease. Between 1999 and 2010, the percentage of adults who have total blood cholesterol levels of 200 mg/dL or greater has decreased from 18% to 13%, and almost 34% have a low-density lipoprotein (LDL) cholesterol of 130 mg/dL or higher (Carroll, Kit, & Lacher, 2012; CDC, 2011d).

Research from the 1980s showed that lowering high blood cholesterol significantly reduces the risk for heart attacks and reduces overall mortality rates. As a result, the National Cholesterol Education Program was launched in 1985 (NIH & NHLBI, 2002). Since its inception, the percentage of people who have had their cholesterol checked more than doubled, from 35% in 1983 to 75% in 2008 (U.S. DHHS, 2010c). Current guidelines recommend that all adults, aged 20 years or older, have their blood cholesterol levels checked every 5 years as a preventive measure (NIH & NHLBI, 2002). Nevertheless, more than 80% of Americans who have high blood cholesterol do not have it under control (AHA, 2012a).

In terms of dietary trends and blood cholesterol levels, as dietary consumption of total fat, saturated fat, and cholesterol declined in the 1980s and 1990s, average blood cholesterol levels in adults declined from 213 mg/dL in 1978 to 198 mg/dL in 2005–2008 (U.S. DHHS, 2010c). Research shows that as little as a 10% decrease in total cholesterol levels can reduce the incidence of CHD by almost 30% (NIH & NHLBI, 2002).

In 2001, the National Cholesterol Education Program released updated clinical guidelines in its report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, referred to as the Adult Treatment Panel (ATP) III (NIH & NHLBI, 2002). This report updates the recommendations made in ATP II and I for people with high blood cholesterol levels and reinforces findings from studies that confirm elevated LDL cholesterol to be a major cause of CHD. The guidelines in ATP III focus on LDL cholesterol–lowering strategies and on primary prevention in persons with multiple risk factors (NIH & NHLBI, 2002). In 2004, an update was added to the National Cholesterol Education Program guidelines on cholesterol management that advised physicians to consider new, more intensive medical treatments, such as statin drugs, for people at high and moderately high risk for a heart attack (Grundy et al., 2004).

**Learning Point** Statin drugs lower blood cholesterol by inhibiting 5-hydroxy-3-methylglutaryl coenzyme A reductase, a liver enzyme that is responsible for producing cholesterol.

The ATP III report recommends a complete lipoprotein profile that includes total cholesterol, LDL cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides as the preferred test rather than screening for total cholesterol and HDLs alone. For LDL-lowering therapy, a person's risk status needs to be assessed based on multiple risk factors, including cigarette smoking, hypertension,

low HDL, family history of premature CHD, and age. Diabetes is considered as a CHD equivalent in assessing a person's risk. In addition, the current guidelines use the Framingham scoring projections of 10-year absolute CHD risk to identify people who need more intensive therapy and those with multiple metabolic risk factors. National Cholesterol Education Program defines high-risk patients as those who have CHD, or diabetes, or multiple (two or more) risk factors, such as hypertension or smoking, which gives them a greater than 20% chance of having a heart attack within 10 years (NIH & NHLBI, 2002). Very high risk patients are those who have cardiovascular disease together with either multiple risk factors (especially diabetes), or badly controlled risk factors (e.g., smoking), or metabolic syndrome (a constellation of risk factors associated with obesity). For moderately high risk and high-risk persons, the ATP III report and update recommend drug therapy in addition to therapeutic lifestyle changes, which include intensive use of nutrition, weight control, and physical activity (Grundy et al., 2004; NIH & NHLBI, 2002).

The specific parts of therapeutic lifestyle changes include the following: reducing intakes of saturated fat to less than 7% of total calories and decreasing cholesterol to less than 200 mg/day. Total calories from fat can be in the 25% to 35% range, as long as saturated and trans-fatty acids are kept low. Individuals are encouraged to use plant stanols and sterols in their diet (2 g/day). Small amounts of plant sterols, or phytosterols, occur naturally in pine trees and such foods as soybeans, nuts, grains, and oils. Increasing soluble fiber to 10 to 25 g/day is also recommended with weight reduction and increased physical activity. Because excessive weight and obesity are considered major underlying risk factors for CHD, weight reduction enhances LDL-lowering interventions.

In comparing the recommendations set forth in the ATP III report to the more recent updates, more intensive treatment options are delineated for very high risk individuals. For high-risk patients, the goal in both reports remains an LDL level of less than 100 mg/dL; for very highrisk patients, a therapeutic option is to treat with statins to lower levels to less than 70 mg/dL. The update lowers the threshold for drug therapy to an LDL of 100 mg/dL or higher and recommends drug therapy for those high-risk people whose LDL is 100 to 129 mg/dL.

**Learning Point** The decision to go on drug therapy for high LDL levels depends on a multitude of risk factors and is decided by the physician and the patient.

In ATP III, for moderately high risk persons the LDL treatment goal is less than 130 mg/dL, and drug therapy is recommended if LDL levels are 130 mg/dL or higher. In the update, there is a therapeutic option to set the treatment goal at LDL less than 100 mg/dL and to use statin drug therapy if LDL is 100 to 129 mg/dL to reach the goal. In the update, when LDL drug therapy is used it is advised that enough medication be used to achieve at least a 30% to 40% reduction in LDL levels. In both reports, anyone with LDL above the goal is a candidate for therapeutic lifestyle changes. In the update, any person at high or moderately high risk who has lifestyle-related risk factors should follow therapeutic lifestyle changes, regardless of LDL level (Grundy et al., 2004; NIH & NHLBI, 2002).

As individuals learn about their risk factors and begin therapeutic lifestyle changes, more registered dietitians and nutritionists will be asked to help people make necessary dietary changes. ATP III guidelines recommend that physicians refer individuals to dietitians for MNT (NIH & NHLBI, 2002). At all stages in the model of therapeutic lifestyle changes, the referral to a dietitian is recommended. This challenges public health providers to provide diet and exercise recommendations that will make a difference to those with elevated blood cholesterol levels.

**Learning Point** Nutritionists also need to consider prevailing eating and ethnic habits when counseling people to lower blood cholesterol levels. In terms of adhering to the ATP III protocol guidelines (and the update), patients and healthcare providers are key players in realizing the benefits of cholesterol lowering and in attaining the highest possible levels of CHD risk reduction.

Both screening for risk factors and compliance in adopting the lipid-lowering guidelines are essential. In reality, fewer than 50% of those persons eligible for meeting the criteria actually receive treatment (NIH & NHLBI, 2002).

### Physical Activity

Research demonstrates that virtually all individuals benefit from regular physical activity. Yet, only approximately 20% of American adults meet recommendations for aerobic physical activity and

muscle strengthening (NCHS, 2011b). Twenty-five percent of all adults are not active at all (CDC, 2010b). Inactivity increases with age and is more common among women than men. Also, those with lower incomes and less education exercise less than those with higher incomes or education (NCHS, 2011b). Thus, physical inactivity is a prevalent risk in the United States. People who are sedentary are almost twice as likely to develop CHD as people who engage in regular physical activity (U.S. DHHS, 2010e). The risk imposed by physical inactivity is almost as high as other well-known CHD risk factors, such as high blood cholesterol, high blood pressure, or smoking (U.S. DHHS, 2008, 2010e). Research shows that moderate physical activity, such as walking 30 minutes a day five times a week, is more likely to be adopted and maintained than vigorous activity (Haskell et al., 2007).

There are many health benefits to be gained from regular physical activity. It enhances cardiovascular function, reduces very low density lipoprotein levels, raises HDL cholesterol, and can lower LDL cholesterol levels (NIH & NHLBI, 2002). Physical activity lowers blood pressure and reduces insulin resistance. Overall, physical activity improves muscle function, cardiovascular function, and physical performance and aids in weight management (Haskell et al., 2007). Nutritionists should become aware of the different types of physical activity, especially moderate levels of activity that can lower an individual's risk for cardiovascular disease.

### Smoking

Cigarette smoking is responsible for more than 443,000 premature deaths each year and is the single largest preventable cause of death and disease among U.S. adults (CDC, 2008). Public health professionals, concerned about the risk factors for cardiovascular disease, need to discuss smoking with clients because it is a leading risk factor. Of the estimated total 12 million deaths from smoking since the 1960s, almost half (5.5 million) are deaths from cardiovascular diseases (U.S. DHHS, 2004). In 2008, an estimated 935,000 Americans had a new or recurrent heart attack (AHA, 2012a). Cigarette smoking was associated with sudden cardiac death in adult men and women (U.S. DHHS, 2004). Smoking-related CHD may also contribute to congestive heart failure, causing 4.6 million to suffer from this disease (U.S. DHHS, 2004). Smoking also is a major cause of stroke, which is the fourth leading cause of death in the United States (NCHS, 2011a). However, the risk of stroke decreases when an individual stops smoking.

Smoking cessation is also effective in preventing heart disease. In fact, people who quit smoking have a significantly lower risk of heart disease and certain cancers, even after a short time of abstinence (U.S. DHHS, 2004). Studies have shown that secondhand smoke exposure causes heart disease among adults, with close to 50,000 deaths each year from secondhand smoke exposure (CDC, 2008; Office on Smoking and Health, 2006).

Smoking is a modifiable risk factor, and its modification is effective in preventing cardiovascular disease mortality. Smoking cessation is particularly important in people with other cardiovascular risk factors, such as high blood pressure and elevated blood cholesterol, because these risk factors work synergistically. Cigarette smoking is also a risk factor for other leading causes of death and disability, including several kinds of cancer and chronic lung diseases (U.S. DHHS, 2004). Smoking has also been linked to other conditions such as reproductive complications, cataracts, hip fractures, and peptic ulcer disease (U.S. DHHS, 2004).

### Trends

Progress has been made in decreasing mortality and risk factors for cardiovascular diseases. Between 1999 and 2009, the age-adjusted death rate for CHD declined by 32.5%, whereas deaths caused by stroke declined by 19.4% (AHA, 2012a; CDC, 2011a). However, the *Healthy People 2010* objectives on CHD and stroke were not reached. Hypertension rates climbed from 29% to 32% (NCHS, 2011a). The prevalence of high blood cholesterol, however, declined from 18% to 13% between 1999–2000 and 2009–2010. There was also a slight decline in smoking between 2000 and 2010. Four modifiable risk factors, poor nutrition, physical inactivity, tobacco use, and excessive alcohol consumption, however, remain responsible for much of the illness and early deaths from chronic disease (CDC, National Center for Chronic Disease Prevention and Health Promotion, & U.S. DHHS, 2009).

Results from epidemiologic studies suggest that diets rich in fruits, vegetables, whole grains, and low-fat dairy foods are associated with a

lower risk of mortality from many chronic diseases, including heart disease (Mente, de Koning, Shannon, & Anand, 2009). This nutritional pattern is supported by all the major health organizations. Nevertheless, a large gap remains between recommended dietary patterns and what U.S. adults actually eat. Only about one-sixth of U.S. adults eat the recommended five or more servings of fruits and vegetables each day (CDC, 2009). Recent research on frequency of fruit and vegetable consumption shows little change from 1994 to 2005 (Blanck, Gillespie, Kimmons, Seymour, & Serdula, 2008). Caloric intake from total fat declined from 36% to 34% from 1976 to 1994, but there was no significant change in 2008 (Wright & Wang, 2010). According to data from the Framingham Heart Study, decreases in dietary fat, especially saturated fat, coincided with increases in HDL cholesterol levels (Ingelsson et al., 2009).

From 1968 to 2008, age-adjusted death rates from cardiovascular diseases declined 68% (NHLBI, 2012). Although this is a positive trend, other health indicators have not improved significantly. Increasing prevalence rates in obesity and the continued high levels of blood pressure and stroke pose a continued public health challenge (NHLBI, 2012).

### Cancer

*Cancer* is an umbrella term used to describe a large group of diseases characterized by uncontrolled growth and spread of abnormal cells (ACS, 2012b). Cancer is the second leading cause of death in the United States, causing 1in every 4deaths. In 2011,an estimated 572,000 Americans died from this disease (ACS, 2011a). According to the ACS (2012b), approximately one-third of cancer deaths are preventable and are attributed to dietary factors, physical inactivity, being overweight, or obesity. For U.S. adults who do not smoke, dietary choices and physical activity are the most modifiable determinants of cancer risk. All cancers caused by cigarettes and the heavy use of alcohol could be prevented. The National Cancer Institute estimates that approximately 12 million Americans who had been diagnosed with cancer at some point in their lives were alive in 2008 (ACS, 2012b). This estimate includes people living with cancer and those who were cancer free. The 5-year relative survival rate for all cancers is 67%, which represents people who are living 5 years after diagnosis of cancer (ACS, 2012b).

Age-standardized death rates from all cancers decreased by 12% between 2000 and 2008 (NCHS, 2012). Despite a decrease in the death rate from cancer, the total number of people who develop cancer each year is expected to double by 2050 because of our aging population (ACS, 2012b). The overall decrease in death rates from cancer is caused by a decline in smoking and more effective detection and screening. The recent decrease in deaths from breast cancer in white women, for example, is the result of a greater use of breast screening in regular medical care. Cancer death rates vary by gender, race, and ethnicity. For example, the death rate is 25% higher for African Americans than for whites for all cancers combined (National Cancer Institute [NCI], 2012).

Although inherited genes play a significant role in cancer risk, they explain only part of all cancer incidences (ACS, 2012b). Most of the variation in cancer incidence cannot be explained by inherited factors. The predominant causes of cancer are external factors such as cigarette smoking, diet or nutrition, weight, and physical inactivity (ACS, 2011b; Khan, Afaq, & Mukhtar, 2010). These factors act to modify the risk of cancer at all stages. It is estimated that more than 50% of all cancers are preventable (Khan et al., 2010). Doll and Peto (1981) estimated that approximately 10% to 70% of deaths from cancer were attributable to diet. However, more recently it has been estimated that diet, physical inactivity, and weight contribute to approximately 33% of cancer deaths (ACS, 2011b). The science of nutrition and cancer is evolving but still is not as developed as that of diet and cardiovascular disease. More large-scale studies are needed to further elucidate the relationship between various nutrients and cancer (Ross, 2010). Future research points to the areas of diet and gene interactions and biomarkers for cancer that will further our understanding in this area (Mathers, 2004).

Current dietary recommendations are based on evidence from the ACS and the American Institute of Cancer Research (AICR)/World Cancer Research Fund (WCRF) (ACS, 2012a; American Institute of Cancer Research & World Cancer Research Fund [AICR & WCRF], 2007). In 2012, the ACS updated its guidelines on nutrition and physical activity after reviewing current scientific evidence (ACS, 2012a). In general, the current dietary guidelines support the 2010 *Dietary Guidelines for Americans* and also encourage the consumption

of plant-based diets, without relying on processed foods. The guidelines also endorse eating a diet that promotes healthy weight control along with physical activity. Specifically, nutrition and food scientists agree on the following recommendations to lower cancer risk (ACS, 2012a):

- Eat a plant-based diet that includes a wide variety of fruits, vegetables, whole grains, beans, and legumes. The recommendation is to choose whole grains over refined sources and 2½ or more cups of vegetables and fruits per day.
- Limit consumption of processed meat and red meat.
- Choose food and beverages in amounts that help achieve and maintain a healthy weight.
- Engage in at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity physical activity each week.
- Drink alcohol in moderation.

Nutrition supplements are not universally recommended for cancer prevention. Instead, the cancer prevention benefits of diet are considered among the best because of the interactions of many vitamins, minerals, and other plant-derived substances found naturally occurring in foods (ACS, 2012a). Although possible benefits of supplemental folate, calcium, and selenium are noted, there is not enough data to support supplementation (ACS, 2012a). By eating whole foods and following the cancer prevention dietary recommendations along with physical activity, the protection of the body's cells may take place during the initiation, promotion, and progression stages of cancer. These food substances may repair damage that has already occurred in cells. Some literature sources state that individuals do not need to be concerned about the pesticide residues on fruits and vegetables because the benefits of eating fruits and vegetables far outweigh any potential risk (ACS, 2012a). More long-term studies may help to state this definitively.

The study of diet and cancer prevention is relatively new. Dietitians and other public healthcare professionals need to stay abreast of these discoveries to give appropriate guidance to individuals at risk. The basis of MNT should include those recommendations set forth by the ACS and the World Cancer Research Fund/AICR (ACS, 2012a; AICR & WCRF, 2007).

### Diabetes

Diabetes is a serious, costly, and increasingly common chronic disease that poses a significant public health challenge. In 2009, diabetes was the seventh leading cause of death in the United States (NCHS, 2011a). Approximately 25.8 million Americans have diabetes, and more than 7 million of these people are unaware they have the disease (CDC, 2011c). Type 2 diabetes, once referred to as adult onset diabetes, may account for approximately 90% to 95% of all diagnosed cases of diabetes (CDC, 2011c). Projections indicate that the prevalence of diabetes could increase from 8% in 2010 to as high as 33% by 2050 (Boyle, Thompson, Gregg, Barker, & Williamson, 2010). The increase in the number of cases has been particularly high within certain ethnic and racial groups in the United States (CDC, 2011c).

Medical complications of type 2 diabetes include heart disease, kidney failure, leg and foot amputations, and blindness. Close to 700,000 people with diabetes have advanced diabetic retinopathy that could lead to severe vision problems (CDC, 2011c). Most deaths caused by diabetes are to the result of diabetes-associated cardiovascular disease. The presence of diabetes in adults is associated with a two- to fourfold increase in CHD compared with nondiabetic adults. Almost 67% of adults with diabetes have hypertension (CDC, 2011c). Diabetes is the cause of 44% of new kidney failure cases. Severe forms of nervous system damage occur in 60% to 70% of diabetic adults. Approximately 60% of all nontraumatic amputations in the United States occur in people with diabetes (CDC, 2011c). Periodontal or gum disease is also more common among diabetics. As a result, diabetes is a costly disease, with the total attributable costs (direct and indirect) estimated at $174 billion annually (CDC, 2011c).

Type 2 diabetes is associated with the following factors:

- *Age*: Diabetes is most common in people older than 45 years (CDC, 2011c).
- *Ethnicity:* Deaths from diabetes are twice as high for African Americans than for whites, Native Americans, and Hispanic Americans; certain Pacific Islander American and Asian American populations also have higher rates.
- *Genetics and family history:* Genetic markers that indicate a greater risk for type 2 diabetes have been identified.

- *Obesity:* The increased prevalence of obesity among adults is positively associated with the increased rates of diabetes (Nguyen, Nguyen, Lane, & Wang, 2010). Data from clinical trials strongly support the potential of moderate weight loss to reduce the risk of type 2 diabetes (Diabetes Prevention Program Research Group, 2009).
- *History of gestational diabetes in women:* Gestational diabetes is a form of glucose intolerance that develops in some women during pregnancy. Obesity is associated with gestational diabetes. Women with a family history of diabetes and Hispanic American and African American women are at an increased risk (CDC, 2011c).
- *Impaired glucose metabolism:* People with pre-diabetes, or those who have an increased risk of developing diabetes, have impaired fasting blood glucose levels and/or a high hemoglobin A1c (measure of average blood sugar control over a 2- to 3-month period). Research studies suggest that weight loss and increased physical activity among people with pre-diabetes may return glucose levels to normal and prevent the onset of diabetes (CDC, 2011c).
- *Physical inactivity:* People who are at higher risk for diabetes and fairly inactive or exercise fewer than three times a week are more likely to develop type 2 diabetes.

In the United States, recent lifestyle changes such as decreased physical activity and increased energy consumption, which contribute to the increased prevalence rates of obesity, are also strong risk factors for diabetes (Diabetes Prevention Program Research Group, 2009). On the other hand, positive lifestyle changes such as diet, weight loss of 5% to 7%, and moderate-intensity physical activity (e.g., walking 30 minutes a day) can delay the onset of diabetes.

**Learning Point** The Diabetes Prevention Program, a major large-scale study of more than 3,000 people at high risk for developing diabetes, confirmed that positive lifestyle changes such as diet, weight loss of 5–7%, and moderate-intensity physical activity (such as walking 30 minutes/day) can delay onset of diabetes. The study also found a 58% reduction in the development of diabetes over a 3-year period. The findings from this study, sponsored by the NIH, showed that exercise, a healthy diet, and weight loss can reduce the risk of developing diabetes by as much as 71% in high-risk individuals (Diabetes Prevention Program Research Group, 2009).

MNT is an essential part of diabetes management for adults. Objectives for MNT include the following (American Diabetes Association, 2008):

- Attaining and maintaining optimal metabolic outcomes, including normalizing blood glucose levels, maintaining a lipid profile that reduces vascular disease risk, and normalizing blood pressure levels
- Preventing, delaying, and treating the onset complications by modifying nutrient intake and lifestyle to prevent and treat obesity, cardiovascular disease, hypertension, and nephropathy
- Optimizing health through sensible food choices and physical activity
- Addressing personal and cultural preferences as well as lifestyle factors, including a person's willingness to change, when determining individual nutritional needs

In terms of specific nutrients and dietary recommendations for type 2 diabetes, studies in healthy subjects and those at risk for type 2 diabetes support the importance of including foods containing complex carbohydrates in the diet, particularly those from whole grains, legumes, fruits, vegetables, and low-fat milk (American Diabetes Association, 2008). Reduced intake of total fat, especially saturated fat, may reduce the risk of diabetes.

In summary, lifestyle changes such as reduced energy intake, increased physical activity, and nutrition education (with the goal of promoting weight loss) represent essential aspects of type 2 diabetes management for adults. *Healthy People 2020* (U.S. DHHS, 2010a) discusses the challenges of diabetes and the preventive interventions aimed at them: primary prevention, screening and early diagnosis, access, and quality of care. This includes secondary and tertiary prevention, such as glucose control and decreasing complications from diabetes (U.S. DHHS, 2010a). Many opportunities exist for dietitians, public health professionals, and educators to contribute to the effective management of diabetes.

### Osteoporosis

As one grows, bones develop and become larger, heavier, and denser. At approximately age 30 years, peak bone mass is achieved in both men and women. After this time period adults begin to lose bone mass, and this continues as they get older.

Osteoporosis, or porous bone disease, develops when bone loss reaches the point of causing fractures under common everyday stresses. Because of increased bone fragility, there is a greater susceptibility to fractures of the hip, spine, and wrist. Both men and women suffer from osteoporosis.

An estimated 9% of adults older than 50 years have osteoporosis, and roughly one-half have low bone mass (Looker, Borrud, Dawson-Hughes, Shepherd, & Wright, 2012). Approximately 77% of women older than 50 years will be affected by osteoporosis or low bone mass compared to 42% of men. These rates correspond to 1 in 2 women and 1 in 4 men, aged 50 years and older, will experience an osteoporosis-related fracture in their lifetime (Office of the Surgeon General, 2004). Osteoporosis causes significant disability with important economic consequences: Caring for bone fractures from osteoporosis costs about $18 billion each year (U.S. DHHS, 2012). Of the 1.5 million fractures occurring each year as a result of osteoporosis, the most common are hip, vertebral, and wrist fractures. Approximately 1 in 5 people are admitted to a nursing home within 1 year of a hip fracture, and approximately 1 in 5 patients older than age 50 years die in the year following their hip fracture (NIH Osteoporosis and Related Bone Diseases National Resource Center, 2011; U.S. DHHS, Public Health Service, & Office of the Surgeon General, 2012).

Osteoporosis often is called the "silent disease" because it can occur without any overt symptoms. The technical standard for measuring bone mineral density is dual-energy X-ray absorptiometry. A low bone mass density is a strong predictor of fracture risk (National Osteoporosis Foundation, 2012). Bone density tests can detect osteoporosis before a fracture occurs and can serve as a predictor for future fracture risks.

The chances of developing osteoporosis are greatest in white women beyond menopause. Women have less bone tissue and lose bone more easily than do men because of the hormonal changes involved in menopause. Those individuals who have a low dietary intake of calcium and vitamin D over a lifetime, who are physically inactive, who are cigarette smokers, who are excessive alcohol drinkers, who are thin and small framed, and who have a family history of osteoporosis are at increased risk (U.S. DHHS, Public Health Service, & Office of the Surgeon General, 2012). In addition, white and Asian American women are at highest risk, whereas African American and Hispanic American women have lower risks (Looker et al., 2012). Anorexia nervosa, the use of certain medications, and low testosterone levels in men are also risk factors for osteoporosis. The five factors that can be modified to prevent osteoporosis are (1) a diet rich in calcium and vitamin D, (2) weight-bearing exercise, (3) a healthy lifestyle that excludes smoking and excessive alcohol intake, (4) routine bone density measurements, and (5) the use of medication, when appropriate (U.S. DHHS, Public Health Service, & Office of the Surgeon General, 2012).

Nutrition is an important modifiable risk factor in terms of both bone health and the prevention and treatment of osteoporosis. An adequate amount of calcium and vitamin D contributes significantly to bone health. Low calcium intakes are associated with low bone mass, rapid bone loss, and high fracture rates (Heany, 2009). Bone is a living, growing tissue, and 99% of the body's calcium is found in bone. Throughout one's lifetime, bone formation and resorption occur. This process, known as bone turnover, is responsive to dietary calcium regardless of age. Dietary calcium works to strengthen bone by suppressing bone resorption and parathyroid hormone (Heany, 2009).

According to the NIH Osteoporosis and Related Bone Diseases National Resource Center (2011), calcium is the most important nutrient for the prevention of osteoporosis. Despite this, actual calcium intakes for most of the U.S. population are considerably lower than the current Dietary Reference Intake (Mangano, Walsh, Insogna, Kenny, & Kerstetter, 2011). Many studies have shown that adult skeletal health is improved by increasing dairy foods or calcium intake in the diet (Heany, 2009). The DASH diet, used to treat hypertension, is a low-fat diet rich in calcium and has been shown to reduce bone turnover and reduce the risk of osteoporosis (Sacks et al., 2001). Calcium requirements can be met with low-fat dairy products; however, typical American diets meet only 52% of recommended dairy intake (USDA & U.S. DHHS, 2010). Other foods contain calcium, such as dark leafy greens, but these foods usually provide less calcium per serving than milk does, and most Americans do not eat these vegetables often. For individuals who do not consume enough calcium in their diets, calcium supplements are recommended.

Vitamin D also plays an important role in calcium absorption and bone health. Vitamin D is a major determinant of intestinal calcium absorption.

When skin is exposed to sunlight, the body synthesizes vitamin D. However, studies show decreased production of vitamin D in older adults and individuals who are housebound, especially in the winter months (NIH Osteoporosis and Related Bone Diseases National Resource Center, 2011). The most recent Dietary Reference Intake increased the vitamin D requirements for children, adults, and older adults (IOM, 2011). Low-fat and nonfat milk, excellent sources of calcium, are fortified with 100 IU of vitamin D per serving.

> For those living in colder climates without much sunshine, vitamin D supplementation is suggested to accompany calcium intake.

In conclusion, osteoporosis is a serious public health disease and is largely preventable. Early in life, both females and males should be advised on how to incorporate sources of calcium into their diets. The use of low-fat and nonfat dairy foods should be recommended, and if persons cannot consume dairy products, other food sources and calcium supplements are necessary. Adequate vitamin D intake needs to be addressed as well. Public health and community nutritionists should encourage individuals to participate regularly in physical activity. Modifiable lifestyle factors should be discussed to promote bone health throughout life.

### HIV/AIDS

In 1981, a new infectious disease, AIDS, was first identified in the United States. A few years later HIV was discovered, and this was identified as the viral agent that causes AIDS. HIV/AIDS has affected almost every ethnic, socioeconomic, and age group in the United States (CDC, 2011b).

AIDS, a deadly disease, is the end stage of HIV infection. The infection progresses to overwhelm the immune system and leaves individuals defenseless against numerous other infections and diseases. HIV is spread through direct contact with contaminated body fluids, sexual intercourse, direct blood contact, or from mother to infant. From 1995 to 1998, death rates from AIDS in the United States declined for the first time and remained stable from 1999 to 2008 (CDC, 2011b). Nevertheless, HIV/AIDS remains a significant cause of illness, disability, and death in the United States. According to the CDC, in 2009 there were approximately 489,000 people living with an AIDS diagnosis and 1.1 million people with HIV infection (CDC, 2010a). Death rates have dropped dramatically in the United States as a result of the introduction of antiretroviral therapies (CDC, 2011b).

Health complications for people with HIV/AIDS are immune dysfunction and its associated complications, which include malnutrition and wasting. HIV targets the immune system, rendering an individual susceptible to infections and disease. The CDC defines the AIDS-related wasting syndrome as a 10% weight loss in a 6-month period accompanied by diarrhea or fever for more than 30 days (CDC, 1993). Malnutrition and its complications can reduce an individual's tolerance to medications and other therapies. Malnutrition occurs in the form of tissue wasting, fat accumulation, increased lipid levels, and risk of other chronic disease. The Academy of Nutrition and Dietetics (American Dietetic Association, 2010) strongly supports nutrition evaluation and MNT as parts of the ongoing health care of individuals infected with HIV. In terms of MNT, this includes early assessment and treatment of nutrient deficiencies, the maintenance and restoration of lean body mass, and continued support for performing daily activities and maintaining quality of life. According to the Academy (American Dietetic Association, 2010), nutrition education and guidance should incorporate the following aspects:

- Healthful eating principles
- Water and food safety issues
- Perinatal and breastfeeding issues
- Nutrition management for symptoms such as anorexia, swallowing problems, diarrhea, and so on
- Food–medicine interactions
- Psychosocial and economic issues
- Alternative feeding methods (supplementation, tube feeding, or parenteral nutrition)
- Additional therapies, including physical activity and disease management
- Guidelines for evaluating nutrition information, diet claims, and individual mineral and vitamin supplementation
- Strategies for treatment of altered fat metabolism

In addition, it is important for an individual infected with HIV to have adequate access to food, health care, and other support systems. The maintenance and restoration of nutrition stores are interrelated with recommended medical therapies; therefore, it is essential that a public health nutritionist be an active participant in the healthcare team to provide optimal MNT.

## Case Study 1

### Cardiovascular Disease: Mr. Cohen's Physical Examination Results

Margaret Udahogora PhD, RD

Mr. Cohen is a 53 year-old executive manager. His job does not allow him much free time, and he daily orders lunch from the nearest fast food restaurant to eat from his desk. Each evening, his wife faithfully encourages him to consume 2 cups of green tea and 2–3 fresh garlic cloves as part of their regimen to decrease the risk of heart disease. Mr. Cohen has no prior history of chronic diseases or hospitalizations. However, his family history is positive for heart problems. His father had a stroke at age 45 years, and his father's brother had a heart attack at age 55 years. Mr. Cohen has recently complained of being short of breath with chest discomfort and is unable to exercise. He is scheduled for a routine physical examination at the recommendation of his wife. After a 8-hour overnight fast, Mr. Cohen's vital signs and laboratory results are as follows: height of 5 feet 8 inches (1.73 m), weight of 214 lb (97.3 kg), BMI of 32.7 kg/m$^2$, blood pressure of 148/88 mmHg, waist circumference of 45 inches, HDL cholesterol of 37 mg/dL, LDL cholesterol of 159 mg/dL, total cholesterol of 270 mg/dL, and triglycerides of 310 mg/dL. Mr. Cohen was started on lipid-lowering drug therapy and was advised to lose weight.

#### Questions

1. What are his cardiac risk factors and how should his lipid profile and vital signs be interpreted based on National Cholesterol Education Program/Adult Panel III (NCEP/ATPIII) guidelines?
2. What additional information would be appropriate to collect from Mr. Cohen during the assessment of his risk factors for coronary heart disease?
3. What are the recommended Therapeutic Lifestyle Changes (TLC) and how can Mr. Cohen translate them into food choices?
4. What is the role of dietary supplements in the prevention and management of cardiovascular diseases?

## Case Study 2

### Frequent Flier at Risk for Chronic Disease

Amy Sheeley, PhD, RD, LDN

Mr. Lewis is a 65-year-old male sent by his physician after his annual physical to see an outpatient dietitian for weight loss. Mr. Lewis reports that he has steadily been gaining weight over the past 10 years. He travels for work; therefore, he reports, he eats a lot of fast food (cheeseburgers, pizza, and burritos) at the airport between flights and does not have the time or energy to exercise. He doesn't add salt to his foods because he knows his blood pressure is borderline high, and he drinks diet soda to control his calories. Mr. Lewis is married and reports that he eats much better when he is at home and not traveling. He doesn't take any medications except for a multivitamin, but he is worried that his doctor may put him on cholesterol and blood pressure medications soon. He is 6 foot 1 inch and weighs 283 lb.

#### Lab values provided by the physician:

Total cholesterol: 230 mg/dL
LDL: 145 mg/dL
Blood pressure: 135/85 mmHg

#### Questions

1. What is Mr. Lewis's BMI? How would you evaluate this information?
2. How much weight would you recommend he lose?
3. How would you explain to Mr. Lewis why weight loss is important? What are some of the risks associated with obesity?
4. What do his laboratory values indicate?
5. What other lab values might you want to request and why?
6. What are some of this patient's medical and nutritional causes of obesity that you should look for in your assessment?
7. What would you say to Mr. Lewis about the dietary changes he has already made?
8. What dietary recommendations would you recommend? What lifestyle factors do you need to take into account?

## Case Study 3

### Osteoporosis

Jessica Brie Leonard, RD

EN is a 71-year-old Hispanic American female who has become concerned about her risk of osteoporosis. Her older sister recently slipped and fell while walking up the front steps of her house and broke her hip. While in the hospital to have the fracture repaired, her sister was diagnosed with osteoporosis. Her sister was in the hospital for 5 days after her surgery, and then required a 2-week stay in a rehab facility with extensive physical therapy, after which she has been unable to regain her prior level of mobility. Watching her sister go through the injury, surgery, and rehab was frightening to EN because her sister is only 2 years older than her.

EN has heard that osteoporosis runs in families and decided to address her concerns about her bone health with her primary care doctor. Her doctor agrees that there may be reason for concern given her sister's injury and upon hearing that EN's grandmother had a hump on her back in her later years. EN's primary care doctor refers her to a registered dietician (RD) to discuss how she can make adjustments to her current eating habits to optimize her bone health.

#### Nutrition Assessment from the Registered Dietician

EN meets with the RD and shares her concerns and the story of her sister's injury. The RD starts by gathering EN's anthropometrics:

Weight: 98 lb
Height: 5 foot 1 inch

(*Continued*)

## Case Study 3 (*Continued*)

BMI: 18.5
Wrist size: 5.3 inches

The RD questions EN about her height and weight history and finds out that her usual body weight is approximately 100 lb and that she used to be 5 foot 2 inches.

EN is at risk for osteoporosis based on the following criteria:

Family history of osteoporosis (sister and grandmother)
Low body weight, < 127 lb
BMI < 19
Wrist size smaller than 5.5 inches

To find out how best to help EN, the RD inquires about her living situation. She finds out that EN lost her husband a little more than a year ago and is now the only person in her household. EN has found cooking for one to be a challenge and often consumes prepared foods or skips meals and eats small snacks instead. While she was growing up she didn't enjoy drinking milk, even though her mother served it at dinner nightly, and was often left at the dinner table to finish alone.

A 24-hour recall with EN reveals the following:

Breakfast: Black coffee; ½ cup oatmeal with brown sugar and raisins; 8 oz orange juice
Lunch: Chicken noodle soup
Dinner: ½ grilled marinated chicken breast; ½ cup steamed broccoli with lemon juice; ½ cup rice pilaf
Snack: Chocolate pudding

The RD recommends EN consume 1,000–1,200 mg of calcium a day and that incorporating more dairy into her daily meal plan is the best way to start reaching this goal. EN shares that she does like some dairy, such as yogurt and cheese, but doesn't remember to eat it every day. The RD encourages EN to continue to eat broccoli and pudding for their calcium content and recommends the following substitutions and additions based on her 24-hour recall.

Make oatmeal with milk or soy milk instead of water
Add sliced almonds to oatmeal
Add sliced Swiss cheese and crackers to lunch

The RD also provides EN with a list of suggested foods to work into her meal plan based on what she has learned about EN's food pattern and preferences.

### Dairy Sources of Calcium

High Calcium (200mg/serving or more)
- 1 oz cheese (cheddar, mozzarella, muenster)
- 1 cup reduced-fat or whole milk
- 1 cup fortified soy milk
- 8 oz yogurt

Moderate Calcium (50–200 mg/serving)
- 1 oz almonds
- 1.5 oz chocolate
- ½ cup cottage cheese
- ½ cup hummus
- ½ cup ice cream

### Nondairy Sources of Calcium

High Calcium (200mg/serving or more)
- ½ cup calcium-fortified cereal

Moderate Calcium (50–200 mg/serving)
- 1 oz almonds
- 1.5 oz chocolate
- ½ cup hummus
- ½ cup spinach (cooked), 1 cup raw

In addition to monitoring her calcium intake, the RD emphasizes the importance of getting adequate amounts of vitamin D (15 mcg or 600 IU for women > 70 years old) so her body can utilize the calcium she is consuming, though they don't need to be consumed at the same sitting.

### Vitamin D–fortified Foods

Milk
Juices
Margarine
Soy milk
Yogurt

### Foods Naturally High in Vitamin D

Fatty fish
Egg yolks
Cod liver oil

### Nutrition Follow-Up with the Registered Dietician

After 2 weeks of working to incorporate calcium and vitamin D into her diet, EN returns to see the RD for a follow-up visit. She reports she's been doing better with adding the yogurt, cheese, and fortified juice into her diet than with adding milk. The RD commends EN for her hard work and lets her know the foods she has added are adding calcium, which was the goal.

As a next step in addressing EN's concerns about her bone health, the RD spends some time educating EN on the importance of eating enough protein (2–3 servings, or 5.5 oz protein-rich food) as well as reducing the amount of sodium in her diet. The RD shares with EN that prepared and processed foods such as canned soups and vegetables can be high in sodium, as can deli meats. Knowing that EN is currently relying on some of these foods to meet her energy needs, the RD recommends EN choose low-sodium soups, meats, and frozen meals.

EN reports that she has had more energy as a result of adding more calories to her diet through dairy foods and is considering taking a yoga class at the nearby senior center that a friend of hers teaches. The RD tells EN she thinks this is a good idea because doing weight-bearing, balance, and flexibility exercises can also help promote her bone health, and the RD recommends that EN confirm this with her doctor before starting exercising. The RD reminds EN to be sure to stay adequately hydrated while exercising because dehydration can lead to dizziness and falls.

#### Questions

1. Name three foods that are commonly fortified with calcium.
2. Name the four areas of the RD's care plan for EN.

3. If vitamin D is needed for adequate calcium utilization, why don't these nutrients need to be consumed at the same time?
4. Why is it important to address sodium consumption with patients with osteoporosis?
5. Why is it important to ask about the patient's height history and not just his or her current height?

## Issues to Debate

1. What would you propose should be incorporated into public health programs that focus on nutrition as an important preventive factor in illness, disability, and death?
2. What advice would you give to the individual in the community with regard to preventing chronic disease risk factors such as obesity, physical inactivity, and smoking to reduce society's financial burden?
3. What proportion of public funds designated for nutrition services should be given for primary, secondary, and tertiary prevention versus acute medical care?
4. Programs such as for-profit weight loss centers are proliferating in this country. What are positive and negative aspects of this trend compared with public health weight-management programs?

## References


American Cancer Society. (2011a). *Cancer facts and figures 2011*. Atlanta, GA: Author.

American Cancer Society. (2011b). *Cancer prevention and early detection, facts and figures, 2011*. Atlanta, GA: Author.

American Cancer Society. (2012a). American Cancer Society guidelines on nutrition and physical activity for cancer prevention: Reducing the risk of cancer with healthy food choices and physical activity. *Cancer Journal for Clinicians*, *62*, 30–67.

American Cancer Society. (2012b). *Cancer facts and figures 2012*. Atlanta, GA: Author.

American Diabetes Association. (2008). Nutrition recommendations and interventions for diabetes: A position statement of the American Diabetes Association. *Diabetes Care*, *31*, S61–S78.

American Dietetic Association. (2006). Position of the American Dietetic Association: The roles of registered dietitians and dietetic technicians, registered in health promotion and disease prevention. *Journal of the American Dietetic Association*, *106*, 1875–1884.

American Dietetic Association. (2007). Position of the American Dietetic Association: Total diet approach to communicating food and nutrition information. *Journal of the American Dietetic Association*, *107*, 1224–1232.

American Dietetic Association. (2009). Position paper: Weight management. *Journal of the American Dietetic Association, 109*, 330–346.

American Dietetic Association. (2010). Position of the American Dietetic Association: Nutrition intervention and human immunodeficiency virus infection. *Journal of the American Dietetic Association*, *110*, 1105–1119.

American Heart Association. (2008). Population-based prevention of obesity: The need for comprehensive promotion of healthful eating, physical activity, and energy balance: A scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention. *Circulation*, *118*, 428–464.

American Heart Association. (2012a). Heart disease and stroke statistics—2012 update: A report from the American Heart Association. *Circulation*, *125*, 2–220.

American Heart Association. (2012b). Sodium, blood pressure, and cardiovascular disease: Further evidence supporting the American Heart Association sodium reduction recommendations. *Circulation*. Retrieved from http://circ.ahajournals.org/content/early/2012/10/31/CIR.0b013e318279acbf.full.pdf+html

American Institute of Cancer Research. (2004). Physical Activity. Retrieved from www.aicr.org/reduce-your-cancer-risk/physical-activity

American Institute of Cancer Research & World Cancer Research Fund. (2007). *Food, nutrition, physical activity, and the prevention of cancer: A global perspective*. Washington, DC: AICR.

Blanck, H. M., Gillespie, C., Kimmons, J. E., Seymour, J. D., & Serdula, M. K. (2008). Trends in fruit and vegetable consumption among U.S. men and women, 1994–2005. *Preventing Chronic Disease*, *5*, A35. Retrieved from www.cdc.gov/pcd/issues/2008/apr/07_0049.htm

Boyle, J.P., Thompson, T.J, Gregg, E.W., Barker, L.E., & Williamson, D.F. (2010). Projection of the year 2050 burden of diabetes in the US adult population: Dynamic modeling of incidence, mortality, and prediabetes prevalence. *Population Health Metrics*, *8*, 29. Retrieved from www.pophealthmetrics.com/content/8/1/29

Carroll, M. D., Kit, B. K., & Lacher, D. A. (2012, April). Total and high-density lipoprotein cholesterol in adults. National Health and Nutrition Examination Survey, 2009–2010. *NCHS Data Brief*, (92), 1–8.

Centers for Disease Control and Prevention. (1993). 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *Morbidity and Mortality Weekly Report, 41*(51), 961–962.

Centers for Disease Control and Prevention. (2008). Annual smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 2000–2004. *Morbidity and Mortality Weekly Report, 57*, 1226–1228.

Centers for Disease Control and Prevention. (2009). *State indicator report on fruits and vegetables, 2009*. Retrieved from www.cdc.gov/nutrition/downloads/State-IndicatorReport2009.pdf

Centers for Disease Control and Prevention. (2010a). Diagnoses of HIV infection and AIDS in the United States and dependent areas, 2010.*HIV Surveillance Report*, *22*. Retrieved from www.cdc.gov/hiv/surveillance/resources/reports/2010report/pdf/2010_HIV_Surveillance_Report_vol_22.pdf

Centers for Disease Control and Prevention. (2010b). *State indicator report on physical activity, 2010*. Retrieved from www.cdc.gov/physicalactivity/downloads/PA_State_Indicator_Report_2010.pdf

Centers for Disease Control and Prevention. (2011a). Age-adjusted death rates for heart disease and cancer: United States, 1999–2009. *Morbidity and Mortality Weekly Report*, *60*, 713.

Centers for Disease Control and Prevention. (2001b). HIV surveillance—United States, 1981—2008. *Morbidity and Mortality Weekly Report*, *60*, 689–693

Centers for Disease Control and Prevention. (2011c). *National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta, GA: U.S. DHHS and CDC.

Centers for Disease Control and Prevention. (2011d). Vital signs: Prevalence, treatment, and control of high levels of low-density lipoprotein cholesterol. United States, 1999–2002 and 2005–2008. *Morbidity and Mortality Weekly Reports*, *60*, 109–114.

Centers for Disease Control and Prevention. (2011e). Vital signs: Prevalence, treatment, and control of hypertension—United States, 1999–2002 and 2005–2008. *Morbidity and Mortality Weekly Report*, *60*, 103–108.

Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, & U.S. Department of Health and Human Services. (2009). *The power of prevention: Chronic disease … the public health challenge of the 21st century*. Retrieved from www.cdc.gov/chronicdisease/pdf/2009-power-of-prevention.pdf

Centers for Medicare and Medicaid Services. (2012). National health expenditure data. Retrieved from www.cms.gov/Research-Statistics-Data-and-Systems/Statistics-Trends-and-Reports/NationalHealthExpendData/index.html?redirect=/nationalhealthexpenddata

Chobanian, A. V., & Hill, M. (2000). National Heart, Lung, and Blood Institute workshop on sodium and blood pressure: A critical review of current scientific evidence. *Hypertension, 35*, 858–863.

Danaei, G., Ding, E. L., Mozaffarian, D., Taylor, B., Rehm, J., … Ezzati, M. (2009). The preventable causes of death in the United States: Comparative risk assessment of dietary, lifestyle, and metabolic risk factors. *PLOS Medicine*, *6*(4), e1000058.doi:10.1371/journal.pmed.1000058

Diabetes Prevention Program Research Group. (2009). 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet*, *374*, 1677–1686.

Doll, R., & Peto, R. (1981). The causes of cancer: Qualitative estimates of avoidable risks of cancer in the U.S. today. *Journal of the National Cancer Institute, 66*, 1191–1308.

Donnelly, J. E., Blair, S.N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). American College of Sports Medicine position stand: Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise*, *41*, 459–471.

Edelstein, S. (2011). *Nutrition in public health: A handbook for developing programs and services* (3rd ed.). Burlington, MA: Jones & Bartlett Learning.

Elfhag, K., & Rössner, S. (2005). Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obesity Reviews*, *6*, 67–85.

Elo, S. L., & Caltrop, J. B. (2002). Health promotive action and preventive action model (HPA model) for the classification of healthcare services in public health nursing. *Scandinavian Journal of Public Health, 30*, 200–208.

Eyre, H., Kahn, R., & Robertson, R. M. (2004). Preventing cancer, cardiovascular disease, and diabetes. *Diabetes Care, 27*, 1812–1824.

Finkelstein, E. A., Trogdon, J. G., Cohen, J. W., & Dietz, W. (2009). Annual medical spending attributable to obesity: Payer- and service-specific estimates. *Health Affairs*, *28*, 822–831.

Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence and trends in obesity among U.S. adults, 1999–2008. *Journal of the American Medical Association*, *303*, 235–241.

Flock, M. R., & Kris-Etherton, P. M. (2011). *Dietary Guidelines for Americans 2010*: Implications for cardiovascular disease. *Current Atherosclerosis Reports*, *13*, 499–507.

Funnell, M. M., Brown, T. M., Childs, B. P., Haas, L. B., Hosey, G. M., … Weiss, M. A. (2009). National standards for diabetes self management. *Diabetes Care, 32 (Supp 1)*, S87–S94.

Goetzel, R. Z., & Ozminkowski, R. J. (2008). The health and cost benefits of work site health-promotion programs. *Annual Review of Public Health*, *29*, 303–323.

Grundy, S. M., Cleeman, J. I., Merz, C. N., Brewer, Jr., H. B., Clark, L. T., Hunninghake, D.B., … American Heart Association. (2004). For the coordinating committee of the National Cholesterol Education Program. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation, 110*, 227–239.

Guenther, P. M., Reedy, R., Krebs-Smith, S. M., & Reeve, B. B. (2008). Evaluation of the Healthy Eating Index-2005. *Journal of the American Dietetic Association*, *108*, 1854–1864.

Harsha, D. W., & Bray, G.A. (2008). Weight loss and blood pressure control. *Hypertension*, *51*, 1420–1425.

Haskell, W. L., Lee, I. M., Pate, R. R., Powell, K. E., Blair, S. N., … Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise*, *39*, 1423–1434.

Hawley, K. L., Roberto, C. A., Bragg, M. A., Liu, P. J., Schwartz, M. B., & Brownell, K. D. (2012).The science on front-of-package food labels. *Public Health Nutrition, 22*, 1–10.

Heany, R. P. (2009). Dairy and bone health. *Journal of the American College of Nutrition, 28*, 82S–90S.

Heron, M. (2012). Deaths: Leading causes for 2009.*National Vital Statistics Report*,*61*(7), 9.

Hoyert, D. L. (2012). *75 years of mortality in the United States, 1935–2010*. NCHS data brief, no 88. Hyattsville, MD: National Center for Health Statistics.

Ingelsson, E., Massaro, J. M., Sutherland, P., Jacques, P. F., Levy, D., D'Agostino, R. B., … Robins, S. J. (2009). Contemporary trends in dyslipidemia in the Framingham Heart Study. *Archives of Internal Medicine, 169*, 279–286.

Institute of Medicine. (2010). *Food and Nutrition Board Dietary Reference Intakes: Recommended intakes for individuals*. Washington, DC: National Academies Press.

Institute of Medicine. (2011). *Dietary Reference Intakes for calcium and vitamin D*. Washington, DC: National Academies Press.

Kannel, W. B., Dawber, T. R., & McGee, D. L. (1980). Perspectives of systolic hypertension, the Framingham Study. *Circulation, 61*, 1179–1182.

Khan, N., Afaq, F., & Mukhtar, J. (2010). Lifestyle as risk factor for cancer: Evidence from human studies. *Cancer Letters*, *28*, 133–143.

Krebs-Smith, S. M., Guenther, P. M., Subar, A. F., Kirkpatrick, S. I., & Dodd, K. W. (2010). Americans do not meet federal dietary recommendations. *Journal of Nutrition*, *140*, 1832–1838.

Kruger, J., Blanck, H. M., & Gillespie, C. (2008). Dietary practices, dining out behavior, and physical activity correlates of weight loss maintenance. *Preventing Chronic Disease*, *5*(1). Retrieved from www.cdc.gov/pcd/issues/2008/jan/06_0158.htm

Looker, A. C., Borrud, L. G., Dawson-Hughes, B., Shepherd, J. A., & Wright, N. C. (2012, April). Osteoporosis or low bone mass at the femur neck or lumbar spine in older adults: United States, 2005–2008. *NCHS Data Brief*, (93), 1–8.

Mangano, K. M., Walsh, S. J., Insogna, K.L., Kenny, A. M., & Kerstetter, J. E. (2011). Calcium intake in the United States from dietary and supplemental sources across adult age groups: New estimates from the National Health and Nutrition Examination Survey 2003–2006. *Journal of the American Dietetic Association*, *111*, 687–695.

Mathers, J. C. (2004). The biological revolution—towards a mechanistic understanding of the impact of diet on cancer risk. *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 551*, 43–49.

Mente, A., de Koning, L., Shannon, H. S., & Anand, S. S. (2009). A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. *Archives of Internal Medicine, 169*, 659–669.

Miller, G., Roehrig, C., Hughes-Cromwick, P., & Lake, C. (2008). Quantifying national spending on wellness and prevention. *Advances in Health Economics and Health Services Research*, *19*, 1–24.

Mozaffarian, D., Hao, T., Rimm, E. B., Willett, W. C., & Hu, F. B. (2011). Changes in diet and lifestyle and long-term weight gain in women and men. *New England Journal of Medicine, 364*, 2392–2404.

National Cancer Institute. (2012). Cancer death disparities. Retrieved from www.cancer.gov/cancertopics/factsheet/disparities/cancer-health-disparities

National Center for Chronic Disease Prevention and Health Promotion. (2009). *Chronic diseases: The power to prevent, the call to control: At a glance*. Atlanta, GA: CDC.

National Center for Health Statistics. (2011a). Deaths: Final data for 2009. *National Vital Statistics Reports*, *60*(3), 1–17.

National Center for Health Statistics. (2011b). *Health, United States, 2011. With special feature on socioeconomic status and health*. Hyattsville, MD: Author. Retrieved from www.cdc.gov/nchs/data/hus/hus11.pdf

National Center for Health Statistics. (2012). Preliminary data for 2010. *National Vital Statistics Reports*, *60*(4), 1–24.

National Heart, Lung, and Blood Institute. (1997). Clinical advisory statement: Importance of systolic blood pressure in older Americans. Retrieved from www.nhlbi.nih.gov/health/prof/heart/hbp/hbpstmt/hbpstmt.htm

National Heart, Lung, and Blood Institute. (1998). *Morbidity and mortality: 1998 chartbook of cardiovascular, lung, and blood diseases*. Bethesda, MD: Public Health Service, NIH, and NHLBI.

National Heart, Lung, and Blood Institute. (2012). *Morbidity and mortality 2012 chartbook of cardiovascular, lung, and blood diseases*. Bethesda, MD: Public Health Service, NIH, and NHLBI.

National Institutes of Health. (2001). Consensus development panel on osteoporosis. *Journal of the American Medical Association, 285*, 785.

National Institutes of Health & National Heart, Lung, and Blood Institute. (1998). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obesity Research, 6*, 1105.

National Institutes of Health & National Heart, Lung, and Blood Institute. (2002). Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*, *106*, 3143–3421.

National Osteoporosis Foundation. (2012). Making a diagnosis. Retrieved from www.nof.org/articles/8

National Research Council Committee on Diet and Health. (1989). *Diet and health: Implications for reducing chronic disease risk*. Washington, DC: National Academies Press.

Nguyen, N. T., Nguyen, X. T., Lane, J., & Wang, P. (2010). Relationship between obesity and diabetes in a US adult population: Findings from the National Health and Nutrition Examination Survey, 1999–2006.*Obesity Surgery*, *21*, 351–355.

NIH Osteoporosis and Related Bone Diseases National Resource Center. (2011). Osteoporosis handout on health. Retrieved from www.niams.nih.gov/Health_Info/Bone/Osteoporosis/osteoporosis_hoh.asp#2

Nolte, E., & McKee, M. (2011). Variations in amenable mortality—trends in 16 high-income nations. *Health Policy*, *103*, 47–52.

Office on Smoking and Health. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the surgeon general*. Atlanta, GA: CDC.

Office of the Surgeon General. (2004). *Bone health and osteoporosis: A report of the surgeon general*. Rockville, MD: Author.

O'Keefe, J. H., Carter, M. D., & Lavie, C. L. (2009). Primary and secondary prevention of cardiovascular diseases: A practical evidence-based approach. *Mayo Clinic Proceedings*, *84*, 741–757.

Pender, N. J., Murdaugh, C. L., & Parsons, M. A. (2010). *Health promotion in nursing practice* (6th ed.). Upper Saddle River, NJ: PrenticeHall.

Psatsy, B. M., Smith, N. L., Siscovick, D. S., Koepsell, T. D., Weiss, N. S., Heckbert, S. R., … Furberg, C. D. (1997). Health outcomes associated with antihypertensive therapies used as first-line agents: A systematic review and meta-analysis. *Journal of the American Medical Association*, *277*, 739–745.

Reddy, K.S., & Katan, M. B. (2004). Diet, nutrition and the prevention of hypertension and cardiovascular diseases. *Public Health Nutrition*, *7*, 167–186.

Ross, S.A. (2010). Evidence for the relationship between diet and cancer. *Experimental Oncology*, *32*, 137–142.

Sacks, F. M., Syerkey, L. P., Vollmer, W. M., Appel, L. J., Bray, G. A., Harsha, D., … DASH–Sodium Collaborative Research Group. (2001). Effects on blood pressure of reduced dietary sodium and Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *New England Journal of Medicine, 344*, 3–10.

Sesso, H. D., Cook, N. R., Buring, J. E., Manson, J. E., & Gaziano, J. M. (2008). Alcohol consumption and the risk of hypertension in women and men. *Hypertension*, *51*, 1080–1087.

Shamansky, S. L., & Clausen, C. (1980). Levels of prevention: Examination of the concept. *Nursing Outlook, 28*, 104–108.

U.S. Department of Agriculture. (2011). ChooseMyPlate. Retrieved from www.choosemyplate.gov

U.S. Department of Agriculture, National Institute of Food and Agriculture. (2012). America on the Move. Retrieved from www.nifa.usda.gov/nea/food/part/health_part_aom.html

U.S. Department of Agriculture & U.S. Department of Health and Human Services. (2010). *Dietary Guidelines for Americans, 2010* (7th ed.). Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (2004). *The health consequences of smoking: A report of the surgeon general*. Atlanta, GA: CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health and Human Services. (2008). *2008 Physical Activity Guidelines for Americans*. Hyattsville, MD: Author. Retrieved from www.health.gov/paguidelines

U.S. Department of Health and Human Services. (2010a). Diabetes. In: *Healthy People 2020*. Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (2010b). *Healthy People 2020*. Washington, DC: Government Printing Office.

U.S. Department of Health and Human Services. (2010c). Heart disease and stroke. In: *Healthy People 2020*. Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (2010d). Nutrition and overweight. In: *Healthy People 2020*. Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (2010e). Physical activity. In: *Healthy People 2020*. Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, & National Center for Health Statistics. (2008). National Health Interview Survey (NHIS). Retrieved from www.healthindicators.gov/Indicators/Bloodpressurescreening_883/Profile/Data

U.S. Department of Health and Human Services & National Heart, Lung, and Blood Institute. (2001). *National Cholesterol Education Program, third report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)*.NIH Pub. No. 01–367. Bethesda, MD: Author.

U.S. Department of Health and Human Services & National Heart, Lung, and Blood Institute. (2012). National High Blood Pressure Education Program. Retrieved from www.nhlbi.nih.gov/about/nhbpep/index.htm

U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute, & National High Blood Pressure Education Program. (2004). *The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*. NIH Pub. No. 03-5233. Bethesda, MD: NHLBI.

U.S. Department of Health and Human Services, Public Health Service, & Office of the Surgeon General. (2012). *The surgeon general's report on bone health and osteoporosis: what it means to you*. Washington, DC: U.S. DHHS.

U.S. Preventive Services Task Force. (2011). *The guide to clinical preventive services 2010–2011*. Washington, DC: U.S. DHHS.

Vollmer, W. M., Sacks, F. M., Ard, J., Appel, L. J., Bray, G. A., Simons-Morton, D. G., … DASH–Sodium Trial Collaborative Research Group. (2001). Effects of diet and sodium intake on blood pressure. Subgroup analysis of the DASH—sodium trial. *Annals of Internal Medicine, 135*, 1019–1028.

Wang, Y., Beydoun, M. A., Liang, L., Caballero, B., & Kumanyika, S.K. (2008). Will all Americans become overweight or obese? Estimating the progression and cost of the US obesity epidemic. *Obesity, 16*, 2323–2330.

Whelton, S. P., Chin, A., Xin, X., & He, J. (2002). Effect of aerobic exercise on blood pressure: A meta-analysis of randomized, controlled trials. *Annals of Internal Medicine, 136*, 493–503.

World Health Organization. (2002). *The world health report 2002: Reducing risks, promoting healthy life*. Geneva, Switzerland: Author. Retrieved from www.who.int/whr/2002/en/whr02_en.pdf

Wright, J. D., & Wang, C. Y. (2010, November). *Trends in intake of energy and macronutrients in adults from 1999–2000 through 2007–2008*. *NCHS Data Brief*, (49), 1–8.

CHAPTER 11

# Special Topics in Adult Nutrition: Physical Activity and Weight Management

*Kristen Kochenour, BS, and Stella Lucia Volpe, PhD, RD, LDN, FACSM*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Describe the differences between overweight and obese and their implications for risk of disease.
2. Explain the components of energy balance and how they can affect weight management.
3. Describe the role of physical activity in maintaining a healthy body weight in adults based on scientific evidence.

Obesity has become a worldwide epidemic. Every country, despite its economic status, has shown some increase in body weight in its population. In the United States it has been estimated that more than 60% of adults and more than 30% of children are overweight and/or obese, and these numbers continue to rise. Many factors contribute to this rise in overweight status, and they include, but are not limited to, genetics, decrease in the thermic effect of food, environmental factors, and the effects of some medications. Nonetheless, though our genes have not changed over thousands of years, people are consuming more energy than before and becoming more sedentary. This imbalance in total energy expenditure can explain the greatest reason for this obesity epidemic.

The purpose of this chapter is to present research in the areas of physical activity, exercise, and weight management. Before the discussion of physical activity and weight management, basic definitions of overweight and obesity are described as well as a basic description of the components of total energy expenditure.

## Definitions of Obesity and Overweight

Overweight and obesity are both used as ranges of body weight that are above what is regarded as healthy for a given height (Centers for Disease Control and Prevention [CDC], 2006). Obesity and overweight also define ranges of body weight that increase the risk of chronic disease (CDC, 2006). There are a number of definitions of overweight and obesity. The universal definitions of overweight and **obesity** have been established using body mass index (BMI; kilograms of body weight/height [$m^2$]). For adults a BMI between 25 and 29.9 is considered overweight, whereas a BMI of 30 or greater is defined as obese (CDC, 2006) (see **Table 11.1**). BMI is a good tool to assess the rates of overweight status and obesity in a population, but it is not a good tool to assess overweight status and obesity in individuals. Thus, a person should be cautious when using BMI in a clinical setting. For most of the population, which is sedentary, BMI correlates well with percentage of body fat, but it is not a measure of percentage of body fat. Therefore, individuals who may be "overweight" but not "over fat," such as athletes, are not considered to be overweight or obese because they have more muscle mass (on average) (CDC, 2006).

**obesity** The universal definitions of overweight and obesity have been established using body mass index (BMI; kilograms of body weight/height [$m^2$]). For adults, a BMI between 25 and 29.9 is considered overweight, whereas a BMI of 30 or greater is defined as obese. Note that this is not the best definition for individuals.

Because of this discrepancy between BMI and percentage of body fat, other definitions of obesity and overweight are based on a percentage above ideal body weight or percentage of body fat. In general, it has been stated that a person is considered overweight if his or her body weight is 20% above ideal body weight. The ideal body weight of a person is typically assessed using the Hamwi equation. The Hamwi equation for ideal body weight in women is calculated as follows:

**TABLE 11.1 Body Mass Index Categories**

| Category | Body Mass Index ($kg/m^2$) |
|---|---|
| Underweight | < 18.5 |
| Normal weight | 18.5–24.9 |
| Overweight | 25–29.9 |
| Obese | > 30 |

Reproduced from National Institutes of Health, National Heart, Lung, and Blood Institute (2013). Available at www.nhlbi.nih.gov/guidelines/obesity/BMI/bmicalc.htm

100 lb for 5 feet, plus 5 lb for every inch over 5 feet, or minus 5 lb for every inch under 5 feet

The Hamwi equation for men is calculated as follows:

106 lb for 5 feet, plus 6 lb for every inch above 5 feet, and minus 6 lb for every inch under 5 feet

For men and women, subtract or add 10%, respectively, if a person is considered small or large framed.

Percentage of body fat measured by underwater (hydrostatic) weighing or by dual-energy X-ray absorptiometry is an ideal method of estimating a person's body fat and, hence, whether they are overweight because of fatness or lean body mass. However, in most situations body fat assessment, even via skinfold measures or bioelectrical impedance measures, is impractical. Thus, for the average population, BMI predicts overweight status and obesity fairly well. It does not predict overweight status and obesity well if someone is muscular and/or has dense bones. When a person who is obviously fit is said to have a high BMI, his or her exercise habits and body composition must be considered.

### GROUP Project

What do you think are the major causes of obesity? Make a list of each cause (making sure that decreased energy expenditure, the topic of this chapter, is one of them), and find at least one recent (within the year) research article that pertains to each item on your list. If your list is short, find two to three research articles for each item. From there, make a theoretical framework where you describe, based on the research, how these components are related and how they affect one another. Use thicker arrows for those components that have a greater impact on other components and thinner arrows for those that have less of an impact. If there is no relationship between one component or another, use no arrows.

## Total Energy Expenditure

**energy expenditure** Kilocalories expended during the day. Total energy expenditure comprises basal metabolic rate, dietary-induced thermogenesis, and the thermic effect of activity. A fourth component is nonexercise activity thermogenesis (NEAT).

Total **energy expenditure** comprises three main components: basal energy expenditure (or basal metabolic rate [BMR]), the thermic effect of food, and the thermic effect of activity. A fourth component, which has had a bit more attention paid to it in recent years, is nonexercise activity thermogenesis. The measurement of total energy expenditure in humans is necessary to assess the metabolic needs of a person in relation to the aforementioned components of total energy expenditure (Levine, 2005).

### Basal Metabolic Rate

Basal energy expenditure and BMR are measured in kilocalories (kcal) per day, or kilocalories per kilogram of body weight per day, or kilocalories per kilogram of fat-free mass per day. Basal energy expenditure or BMR is defined as the energy to maintain circulation, respiration, and so forth at rest. Sometimes basal energy expenditure is referred to as resting energy expenditure or resting metabolic rate (RMR). Although resting energy expenditure and RMR are interchangeable, BMR and RMR are actually not exactly the same because RMR is typically about 5% greater than BMR. This is because with BMR, the individual being assessed via direct or indirect calorimetry does not have to travel to a site and therefore does not have an increased heart rate before the measurement. With RMR, however, the individual typically travels to a site to have his or her RMR assessed. More recent research (Levine, 2005), however, has shown that when given 10 to 15 minutes rest, RMR and BMR are virtually the same. Furthermore, Ventham and Reilly (1999) reported that the measurement of RMR is highly reproducible in children, 6 to 11 years of age, even when children were provided only 5 to 10 minutes of rest before indirect calorimetry measurements. Regardless, both RMR and BMR are measured when an individual has fasted for 8 to 12 hours.

RMR or BMR can be evaluated via indirect calorimetry, direct calorimetry, or doubly labeled water. Although direct calorimetry and doubly labeled water provide more accurate evaluations of energy expenditure, indirect calorimetry is the most often used because of cost, but it too provides a high amount of accuracy (Levine, 2005). Furthermore, doubly labeled water allows the investigator to assess only total energy expenditure, not just RMR or BMR. When resources are limited and/or high accuracy is not required, noncalorimetric methods and total collection systems may be used; however, the limitations of these systems must be noted (Levine, 2005). A more holistic and accurate approach to measure total energy expenditure is to combine any of the described methods with daily log entries of activity (Levine, 2005).

**Learning Point** A rough method to assess BMR is to multiply a person's body weight by 10. Thus, if someone weighs 150 pounds, then 150 × 10 = 1,500 kcal. Although this is a rough estimate of a person's BMR, it can provide an idea of his or her BMR.

### Thermic Effect of Food

The thermic effect of food is another component of the total energy expenditure equation. The thermic effect of food, also known as dietary-induced thermogenesis, is the amount of energy required for absorption and digestion of food. With indirect calorimetry, the thermic effect of food is measured for a 4-hour period postprandially, with about 5 to 10 minutes given to the individual as a break at the end of each hour. The thermic effect of food comprises approximately 10% of total energy expenditure. It has been reported that obese individuals may have a slightly lower thermic effect of food than 10%.

### Thermic Effect of Exercise

The thermic effect of exercise is perhaps the most variable component in the total energy expenditure equation. It can be as low as 10% in very sedentary individuals to as high as 100% in Olympic athletes or professional athletes who work out from 6 to 10 hours per day. **Table 11.2** shows activity factors that are used to estimate thermic effect of activity.

### Nonexercise Activity Thermogenesis

**Nonexercise activity thermogenesis (NEAT)** is defined as "the energy expenditure of all physical activities other than volitional sporting-like exercise. NEAT includes all the activities that render us vibrant, unique, and independent beings such as working, playing, and dancing" (Levine, Vander Weg, Hill, & Klesges, 2006). This has been shown to account for up to 2,000 kcal/day, depending on the amount of NEAT a person accumulates throughout each day (Levine et al., 2006). It has been reported that lean individuals stand and move more than obese individuals, indicating that lean individuals have greater amount of NEAT than do obese individuals (Levine et al., 2005). Even after lean and obese individuals gained weight, NEAT was still significantly greater in lean individuals, indicating central and humoral mediators that drive sedentary behaviors in obese individuals (Levine et al., 2005).

**nonexercise activity thermogenesis (NEAT)** The energy expenditure of all physical activities other than volitional sporting-like exercise. NEAT includes activities such as working, playing, and dancing.

**TABLE 11.2** Activity Factors Used to Estimate Thermic Effect of Activity

| Activity | Multiple of Basal Energy Expenditure |
|---|---|
| Resting | = Basal energy expenditure |
| Very light (sitting, standing) | 0.1 |
| Light (leisure, housework) | 0.25 |
| Moderate (3.5–4.0 mph) | 0.5 |
| Heavy (fast walk +) | 0.7 |

Obese individuals appear to have a natural predisposition to be seated for 2.5 hours a day more than their sedentary lean counterparts. If obese individuals could simply incorporate more NEAT into their daily routine and become more "NEAT-o-type" (Levine et al., 2006), they could expend about 350 more kcal per day, which could lead to a 15-kg weight loss over a year's period (Levine et al., 2005). Levine and colleagues (2006) stated that obesity reflects the surfacing of a "chair-enticing environment," where those who have a natural predisposition to sit have done so and, hence, have become obese. As a society, we need to assist individuals by promoting standing more often and becoming more active throughout the day. This can be done by reconfiguring work, school, and home environments to result in active lifestyles (Levine et al., 2006).

Lower NEAT in obese individuals could be a result of a decreased production of neuromedin U (NMU), which has been associated with the control of energy intake and expenditure (Novak, Zhang, & Levine, 2006). NMU appears to affect the hypothalamic nuclei of the brain. Novak and associates (2006) applied varying doses of NMU directly into the paraventricular and arcuate hypothalamic nuclei of Sprague-Dawley rats. They reported increased physical activity and NEAT when NMU was applied to either nucleus; increased doses resulted in increased activity (Novak et al., 2006). Interestingly, they also reported that NMU decreased energy intake and body weight. Though the study of NMU is in its early stages, there could be promise of this peptide in combating obesity.

Despite the cause of a lower NEAT in obese individuals, a great deal of attention in the clinical and fitness industry has *not* been placed on NEAT. Nonetheless, focusing on NEAT would be a more realistic and simple approach to help obese individuals incorporate more activity into their daily lives, lose weight, and then begin to incorporate physical activity and exercise into their daily routines, after some initial weight has been lost, giving them more confidence to actually exercise.

## Physical Activity and Obesity Prevention

Physical activity and exercise interventions, as well as a combination of physical activity/exercise and diet interventions, have been well researched in the area of obesity prevention. In 2010, the *Dietary Guidelines for Americans* listed physical activity and weight management as two of its nine key recommendations (U.S. Department of Health and Human Services [U.S. DHHS] and U.S. Department of Agriculture [USDA], 2010). The main focus of this chapter is to present recent research in physical activity and a combination of physical activity and diet interventions and discuss the main outcomes of these trials. Though the most recent research is highlighted, some key older studies are also presented to lay the groundwork of where research in this area has been and where it will be moving toward.

### Definitions of Physical Activity and Exercise

Before discussing research in the areas of physical activity and exercise and nutrition, it is important to define the differences between physical activity and exercise. **Physical activity** is defined as "bodily movement that is produced by the contraction of skeletal muscle and that substantially increases energy expenditure" (U.S. DHHS, 1996). Common categories of physical activity include occupational, household, leisure time, and transportation (U.S. DHHS, 1996). Occupational, household, and transportation physical activities are also known as "utilitarian" physical activities. In 2001, the Behavioral Risk Factor Surveillance System reported that in the United States from 28% to 55% of individuals performed leisure time physical activity (CDC, 2003). **Exercise** is defined as planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness (U.S. DHHS, 1996).

**physical activity** Body movement produced by the contraction of skeletal muscle and that substantially increases energy expenditure. Common categories of physical activity include occupational, household, leisure time, and transportation.

**exercise** Planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness.

**Learning Point** The five components of physical fitness are cardiorespiratory endurance, muscular endurance, muscular strength, flexibility, and body composition (Ward, 1999). It has been reported that only approximately 20% of individuals in the United States exercise on a regular basis (three or more times per week, for at least 20 minutes per session).

### Weight Loss Goals

Despite so few people participating in physical activity or exercise on a regular basis, individuals significantly ($p = 0.05$) overestimate their energy expenditure and underestimate their energy intake by approximately 500 kcal and 1,000 kcal, respectively (Lichtman et al., 1992). In addition, it has been reported that obese individuals have unrealistic weight loss goals. Before participating in a weight loss trial, individuals stated that a 38% reduction from their initial body weight (e.g., from an average of 218 pounds to an average of 135 pounds) would be their "dream weight" and that if they lost only 17% of their initial body weight (e.g., from 218 pounds to 180 pounds) they would be "disappointed" (Foster, Wadden, Vogt, & Brewer, 1997). In reality, individuals in most weight loss studies lose an average of about 15% of their initial body weight, which would be below their "disappointed" body weight loss.

Wadden and colleagues (2003) evaluated whether notifying obese participants that they would only lose a modest amount of body weight would then guide them to agree to more reasonable weight loss goals. At the baseline interview, the researchers interviewed 53 obese women, who stated that, on average, they expected to lose about 28% of their initial body weight after the first year of treatment with a weight loss medication. Before initiation of the trial, it was explained to the women, both in writing and verbally, that they should expect to lose about 5% to 15% of their initial body weight (Wadden et al., 2003). Regardless of this prior information, the women did not alter their weight loss expectations throughout the trial. These unrealistic expectations may be one of the many reasons for the high rate of recidivism after weight loss trials.

### How Much Exercise or Physical Activity Is Enough?

In 2008, the physical activity guidelines were established by the U.S. Department of Health and Human Services, in conjunction with the American College of Sports Medicine and the American Heart Association. The guidelines recommend that adults younger than age 65 years perform moderately intense cardiovascular (aerobic) exercise for 30 minutes per day, 5 days per week, or perform vigorously intense cardiovascular activity 20 minutes per day, 3 days per week. They also recommend strength training is included twice per week. The same recommendations are provided

for individuals older than 65 years of age or those 50 to 64 years of age who have a chronic disease. In addition, these individuals are encouraged to perform balance exercise if they are at risk for falling and should have a physical activity plan in place. For those with chronic conditions, that physical activity plan should be made with a certified exercise specialist. For more details on the *2008 Physical Activity Guidelines*, see http://www.health.gov/paguidelines. It is important to note that the physical activity guidelines were established for health promotion and disease prevention and may not be enough minutes per week for weight loss.

In 2009, however, the American College of Sports Medicine updated its position stand on effective weight loss methods (Donnelly, Blair, Jakicic, Manore, & Rankin, 2009). The researchers reported that more evidence supports greater physical activity to prevent weight regain after weight loss. The American College of Sports Medicine now recommends that moderate-intensity physical activity, between 150 and 250 minutes per week, is effective to prevent weight gain; however, moderately intense physical activity, between 150 and 250 minutes per week, will result in only modest weight loss. More than 250 minutes per week has been shown to result in significant weight loss. Exercising at moderate intensity for 150 to 250 minutes per week has been shown to result in weight loss only when energy intake is severely restricted (Donnelly et al., 2009).

The Behavioral Risk Factor Surveillance System assessed participation in the recommended aerobic and muscle-strengthening activities based on the *2008 Physical Activity Guidelines*. In 2011, 51% of adults met the aerobic guidelines and 29.3% met the muscle-strengthening guidelines only (CDC, 2011).

In addition to the *2008 Physical Activity Guidelines*, the National Academy of Sciences of the Institute of Medicine established Dietary Reference Intakes for physical activity and exercise. In brief, the Dietary Reference Intake recommendation states that individuals should exercise at least 30 minutes per day at a high intensity or at least 60 minutes per day at a low intensity (Institute of Medicine, Food and Nutrition Boards, 2005). These parallel the more recently established *Physical Activity Guidelines*; however, they do not mention resistance training or balance activities.

### Physical Activity Interventions and Weight Loss

The research in the area of physical activity and weight loss has covered many types of study designs. In an effort to assess whether multiple bouts of physical activity with differing duration and intensities result in similar weight loss, Jakicic, Marcus, Gallagher, Napolitano, and Lang (2003) conducted a 12-month study in 184 overweight sedentary women, near 37 years of age, with a BMI of 32. There were four groups based on exercise intensity: (1) vigorous intensity/high duration, (2) moderate intensity/high duration, (3) moderate intensity/moderate duration, and (4) vigorous intensity/moderate duration of walking. The women were given motorized treadmills and kept a weekly exercise log. They were also told to maintain an energy intake of between 1,200 and 1,500 kcal/day, with about 20% of total energy intake derived from fat. Jakicic and colleagues (2003) reported significant ($p$ = 0.001), and similar, weight loss in all four groups of women, with the greatest weight loss in the group exercising 200 minutes or more per week. Significant increases in cardiorespiratory fitness were found in all groups. This research establishes a dose–response relationship between exercise and weight loss depending on intensity and duration of activity. Though a well-controlled study, this research lacks the ability to determine the same effect on other types of exercise because the prescribed exercise was brisk walking.

Jakicic, Winters, Lang, and Wing (1999) conducted a longer-term study that somewhat paralleled the aforementioned research. They compared the effects of intermittent exercise with traditional long-bout exercise on weight loss, but they added another component: They examined the effects of intermittent exercise with the effects of using home exercise equipment. The participants were 148 sedentary overweight women (BMI = 32.8 ± 4.0 kg/m$^2$, 36.7 ± 5.6 years of age). The study was 18 months in duration, and the subjects were randomly assigned to one of three groups: long-bout exercise, multiple short-bout exercise, or multiple short-bout exercise with home exercise equipment (SBEQ) using a treadmill. The primary aims of the researchers were to assess the differences among these three groups in body weight, body composition, cardiorespiratory fitness, and exercise adherence.

They reported that all three groups improved their cardiorespiratory fitness, with no difference

among groups. Weight loss was significantly greater ($p = 0.05$) in participants in the SBEQ group compared with participants in the short-bout group, with no differences in weight loss seen between the long-bout group and either SBEQ or the short-bout group (weight loss: –7.4 ± 7.8 kg [SBEQ], –3.7 ± 6.6 kg [short-bout group], –5.8 ± 7.1 kg [long-bout group]).

Two interesting findings resulted from this study. First, the SBEQ group was able to sustain a significantly greater intensity of exercise than were subjects in the short-bout and long-bout groups ($p = 0.05$). In addition, Jakicic and colleagues (1999) found a clear and significant ($p = 0.05$) dose–response relationship between the amount of exercise and the amount of weight loss. Thus, participants who exercised more than 200 minutes per week for the 18-month intervention had a significantly greater weight loss than did individuals who exercised from 150 to 200 minutes per week and compared with individuals who exercised less than 150 minutes per week (–13.1 ± 8.0 kg [200 minutes/week], –8.5 ± 5.8 kg [150 to 200 minutes/week], –3.5 ±6.5 kg [150 minutes/week]). They concluded that access to home exercise equipment may assist in exercise adherence, resulting in weight loss in the long term. Furthermore, the dose–response relationship they reported in this and the aforementioned study, which would seem intuitive, clearly indicates that the greater energy expenditure, the more body weight the person will lose.

### Physical Activity and Weight Loss in the Primary Care Setting

The primary care setting is becoming a more popular place to conduct research on weight loss because the patients, for the most part, have developed a relationship with their primary care practitioners. Booth, Nowson, Huang, Lombard, and Singleton (2006) evaluated the effect of primary care physicians writing prescriptions that recommended lifestyle changes to their patients. They called this the "active nutrition script," which included five nutrition messages and personalized exercise advice for the prevention of weight gain. This was a pilot study whereby family physicians were asked to write 10 active nutrition scripts over a 4-week period to 10 adult patients who had a BMI between 23 and 30. The physicians recorded the patients' body weight, height, waist circumference, gender, date of birth, type and frequency of exercise prescribed, and nutrition messages on each script. Booth and associates (2006) reported that 19 family physicians (63% of them were women) provided approximately nine active nutrition scripts over a 4-week period. A total of 145 patients (57% were women) received the active nutrition scripts (54 ± 13.2 years of age, mean BMI = 31.7 ± 6.3 kg/m$^2$). Seventy-eight percent of the time the physicians wrote the active nutrition scripts for weight loss. Of the physicians interviewed by the researchers (17 of 19 physicians were interviewed), they all stated that the active nutrition scripts messages were clear and easy to deliver. Though the physicians found this script easy to deliver, they mainly prescribed it for weight loss instead of prevention of weight gain. This research needs to be taken to the next step, where body weight and BMI are actually measured over time in the patients who receive the active nutrition scripts. Although this was a pilot study, the primary care setting may be one of the best places for prevention of weight gain and for weight loss.

Jay and colleagues (2013) conducted a study with 23 primary care residents at the New York University School of Medicine. The intervention group of residents received 5 hours of training on how to counsel appropriately patients who are obese. The training was based on the 5As model: Assess, Advise, Agree, Assist, and Arrange. For the intervention portion, 158 obese patients with a BMI greater than 30 were recruited after a medical visit. Chart reviews were conducted to determine weight change. Furthermore, patient exit interview questionnaires were distributed to assess patient characteristics.

Jay and colleagues (2013) reported mean weight change at 12 months between the intervention and control groups. Patients of residents in the intervention group had a mean weight loss of 1.53 kg, whereas patients of residents in the control group reported a weight gain of 0.30 kg. Six patients in the intervention group lost more than 5% of their body weight. Although the amount of weight loss was small, this study shows that training physicians on how to counsel obese patients can encourage weight loss in patients. Further studies need to be conducted with larger sample sizes to determine the statistical significance of these interventions.

McQuigg and coworkers (2005) conducted a study in the United Kingdom in 80 primary care settings whose principal aim was to improve the management of obese adults in a primary care setting.

The participants had a BMI of 30 or a BMI of 28 with at least one obesity-related comorbidity. This counterweight program consisted of four phases: audit and project development, practice training and support, nurse-led patient intervention, and evaluation. For the intervention portion, evidence-based pathways and incorporated approaches were used to empower the health care providers and patients in weight management. This program also had weight management advisers who were registered dietitians specializing in obesity.

McQuigg and coworkers (2005) reported a large number of intervention practices had been trained (almost 94%), with a recruitment of 1,549 patients. At 1 year after implementation, 33% of the patients achieved a weight loss of 5% or more, which is clinically significant because only a small amount of weight loss has been shown to reduce the risk of chronic disease. It has been reported that for every kilogram of weight lost, there is a 16% reduction in the risk of type 2 diabetes mellitus (Hamman et al., 2006).

Although the aforementioned studies were successful, Morrato, Hill, Wyatt, Ghushchyan, and Sullivan (2006) asked the question, "Are health care professionals advising patients with diabetes or at risk for developing diabetes to exercise more?" Though the focus of this chapter is on physical activity and weight loss, one of the primary comorbidities that develops as a result of obesity is type 2 diabetes mellitus. Morrato and colleagues (2006) accessed the Medical Expenditure Panel Survey, whereby more than 26,000 adults in the United States responded in 2002. They found that 73% of the adults with type 2 diabetes mellitus were told by a healthcare professional to exercise more, compared with 31% without diabetes mellitus. In addition, the number of people receiving exercise guidance increased as the amount of diabetes risk factors increased. These researchers reported that BMI and cardiovascular disease risk factors were the strongest predictors for practitioners to give exercise recommendations. They did report, however, that as the diabetes risk factors decreased, exercise advice to patients decreased, resulting in "missed opportunities for disease prevention" (Morrato et al., 2006). Despite the exercise advice, the patients did not always change behavior, which is still the most difficult part of the equation to change (Morrato et al., 2006).

### Cultural Diversity

It is important to respect and try to understand the different cultural practices of different ethnicities; however, it is equally important not to stereotype. For example, if one patient is Asian American, the clinician should not assume that he or she consumes traditional Chinese meals. Asking a patient his or her likes, dislikes, and usual dietary practices is always important. Conversely, if one of your patients is Caucasian, you should not assume that he or she consumes typical "American" meals. Again, talk to the patient and find out likes, dislikes, usual practices, and ethnicity. Healthcare practitioners and researchers cannot make assumptions; each person's individuality needs to be respected. However, there are cultural diversity issues in weight loss, obesity, and other chronic diseases; research has clearly shown differences. Nonetheless, always get to know each patient, client, or study participant as an individual.

There are racial and ethnic differences in perception of body weight. Ness and associates (2012) examined racial differences in the relationship between perception of a healthy body weight and BMI. There were 689 women (145 African American and 544 Caucasian) enrolled in the study. Despite the fact that the African American women had a significantly higher BMI than the Caucasian women, they were less likely to report that they were overweight (BMI = 33.1 versus 29.2, respectively [p = 0.0001]). Ness and coworkers (2012) stated, "Although black women, in general, face a greater threat of morbidity from weight-related chronic diseases, they are more likely to be accepting of their weight at higher BMIs relative to whites. Weight loss interventions and counseling about healthy body size may influence healthy behavior and reduce the risk of chronic diseases."

## How Can People Be Successful at Maintaining Weight Loss?

The maintenance of weight loss is probably the most difficult phase of any weight loss program. After a person feels successful losing weight, he or she often returns to eating the amount of foods consumed before weight loss and decreases physical activity. Though there may be many psychological, and even perhaps physiologic, reasons behind this, there are practical ways people can incorporate physical activity into their day, which can help to increase energy expenditure and maintain weight loss and/or prevent weight gain.

Hill, Wyatt, Reed, and Peters (2006) stated that small changes can lead to big effects on the prevention of weight gain. They stated that if energy balance can be affected by a mere 100 kcal/day, this could lead to a large reduction in the obesity epidemic. People could achieve this small energy deficit by simply walking 15 minutes per day (and multiple bouts would be effective) and/or eat just a few less bites of food at each meal. These small changes are not daunting and may result in greater adherence to weight loss and maintenance.

Though the obesity epidemic is considered a gene–environment interaction (i.e., the human genotype is predisposed to ecologic influences that affect energy intake and expenditure), it is mostly a

problem of energy balance (Hill, 2006). The most successful interventions are those that affect energy balance (increase expenditure and decrease intake) but that also take into consideration behavioral and environmental factors that play a big role in why individuals do not exercise or eat higher amounts of total energy (Hill, 2006). Hill (2006) stated the following:

> Our best strategy for reversing the obesity epidemic is to focus on preventing positive energy balance in the population through small changes in diet and physical activity that take advantage of our biological systems for regulating energy balance. Simultaneously we must address the environment to make it easier to make better food and physical activity choices. This is a very long-term strategy for first stopping and then reversing the escalating obesity rates, but one than can, over time, return obesity rates to pre-1980s levels.

Other tools that individuals can use to help with weight loss and weight maintenance are the *Dietary Guidelines* and the MyPlate communications developed by the U.S. Department of Agriculture to promote choosing healthier foods. MyPlate is designed to remind Americans to eat healthily. The recommendations include reducing sugar and salt intake, making half the plate fruits and vegetables, as well as making at least half the grains whole, and eating fewer foods with solid fat (see **Figure 11.1**). Visit http://www.choosemyplate.gov for more information.

The MyPlate guidelines support the Healthy People 2020 initiative created by the U.S. Department of Health and Human Services. This program was developed to set goals for the American population to achieve by 2020.The goals aim to prevent disease, promote quality of life, and eliminate disparities including obesity (U.S. DHHS, 2010).

Just as the primary care setting can be an ideal location to influence obesity, interventions that include the entire family could also lead to successful maintenance of weight loss. Rodearmel and associates (2006) focused on increasing steps walked per day and cereal consumption (for breakfast and snacks) as a weight reduction intervention for families. In their study they included 105 families who had at least one 8- to 12-year-old child who was at risk for becoming overweight or obese. This was

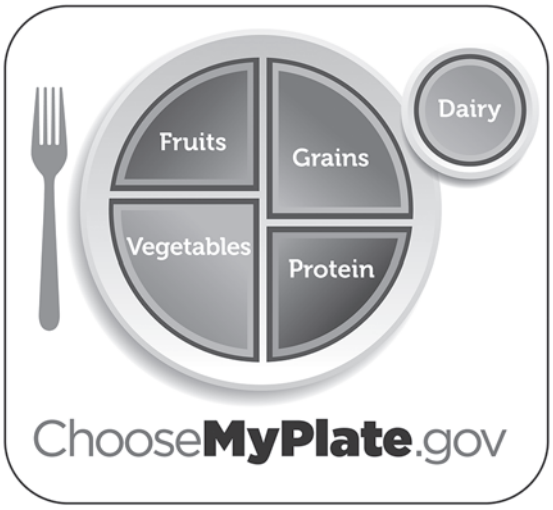



**Figure 11.1** MyPlate.gov.

Courtesy of USDA. Center for Nutrition Policy and Promotion.

a 13-week intervention, whereby 82 families were randomly assigned to the family-based intervention (increase steps to 2,000 per day and consume two servings of ready-to-eat cereal per day) and 23 families were randomly assigned to the control group. The intervention groups showed significant reductions in BMI and BMI-for-age in the adults and children, respectively, especially in mother–daughter pairs. Focusing on the family is a logical and practical way for individuals to maintain weight loss and/or prevent weight gain because all individuals are making healthful changes together and provide a support system for one another.

Perhaps the most well known registry of successful weight loss maintenance is the National Weight Control Registry. This is a registry of individuals who had an initial weight loss average of 33.1 kg and who were able to maintain a 13.6-kg weight loss for 5.8 years before enrolling in the registry (Phelan, Wyatt, Hill, & Wing, 2006). Although many studies have been published on participants in the National Weight Control Registry, Phelan and coworkers (2006) evaluated whether dietary intake habits of registrants changed over the years, especially most recently, with the surge of low-carbohydrate diets. They included 2,708 participants who were part of the National Weight Control Registry since 1995. They reported that from 1995 to 2003 energy from fat intake increased from 23.8% to 29.4%, with an increase in saturated fat from 12.3 to 15.4 g/day. Subsequently, energy from carbohydrate declined from 56% to 49% ($p$ = 0.0001), and those who consumed less than

**TABLE 11.3** Practical Ways to Incorporate More Physical Activity into the Day

- Take the stairs instead of the elevator.
- Park farther away from the destination.
- Sit on a balance ball while working at a desk.
- Exercise during commercials while watching television (if made into a family event it will be even more fun and successful!).
- Have a Thera-Band available by desks, couches, and so on so strength training exercises can be performed.
- Dance.
- Stretch during the day.
- Stand up during meetings.
- Do chair exercises (e.g., leg lifts, work on core strength).
- Move as much as possible throughout the day—be "inefficient."

90 g/day of carbohydrate increased from 5.9% to 17.1% ($p = 0.0001$). Except for 1995, where physical activity was calculated at 3,316 kcal/week, the remaining years were stable at about 2,620 kcal/week. Phelan and associates (2006) stated that weight regain over 1 year was primarily associated with greater energy intake, more fast food consumption, increased fat intake, and lower amounts of physical activity ($p = 0.03$). **Table 11.3** lists practical ways people can incorporate more physical activity into their lives.

**Learning Point** Though a small percentage of individuals in this registry reported consuming a low-carbohydrate diet, the main factors that allowed these individuals to maintain weight loss over time remain the same: consumption of a low-energy diet, moderate fat intake, limited consumption of fast food, and high amounts of physical activity or exercise (Phelan et al., 2006).

## Summary

Obesity is a complex issue that requires a multifaceted approach to result in success. Although it is clearly a gene–environment interaction, the onus is still placed on greater energy expenditure combined with decreased energy intake. Increasing NEAT, multiple versus long bouts of exercise, and physical activity promotion in the primary care setting and family are all promising ways that the obesity epidemic can be reversed.

## Case Study 1

### Eating on the Run

Don is a 34-year-old African American who is an attorney at a large practice in Chicago. He is fairly new to this firm and wants to make partner. His job is very stressful. He works about 100 hours per week and gets little time to eat well or exercise, but he would really like to start to exercise even a few days per week.

Body weight: 225 lb
Height: 5 feet, 8 inches
Exercises once or twice a week "when I get time"

He says, in general, that he consumes his breakfast "on the run," usually in the car on his way to work. His wife is also an attorney at a large firm, and they are both often very busy. When he does eat breakfast, it is usually something quick, like a donut, or he may stop at a fast food restaurant drive-through to get an egg and cheese croissant sandwich. He drinks about five to six cups of coffee per day.

Lunch is either something from the vending machine or, the opposite, a large meal at an expensive restaurant with one of his clients. Thus, his lunch energy intake varies drastically. For dinner he and his wife often eat take-out Chinese food, pizza, submarine sandwiches, or hamburgers and french fries. They do try to eat more "normally" on weekends, but both are often in the office at least one day on the weekend.

#### Questions

1. Calculate Don's BMI.
2. Calculate Don's total energy expenditure.
3. Based on this crude "dietary recall," provide a rough estimate of his energy intake.
4. What ideas would you give Don to help him improve his eating? Be conscious of the fact that he has little time to prepare meals.
5. How might Don be able to incorporate any physical activity in his life? Be creative!

## Case Study 2

### Pre-Diabetes

Samantha has made a decision to try and do something about her weight and eating habits. A week ago she had a yearly checkup and learned that her blood pressure was edging upward, even though she takes medication, and her blood sugar was in the "pre-diabetes" range. Samantha is 5 foot 1 inch and weighs 170lb. The nurse checked her waist circumference and found that it was 36 inches and that her waist to hip ratio was 0.85. The physician increased the blood pressure medication and explained that she would be able to improve her blood pressure, physical mobility, and blood sugar levels if she could lose some weight. Her doctor

also provided a prescription for weight loss medication, but Samantha has not filled it yet.

Your initial interview reveals that Samantha is an active 55-year-old who works part-time at the community library. She states that she would like to "do something about this weight" so she can feel better, get off medications, and enjoy playing with her grandchildren. Her history includes frequent complaints of pain in her knees, ankles, and feet when walking and completing routine activities with a need for daily pain relief medication. Samantha is severely compromised in her ability to move about and cannot do any vigorous aerobic exercises. After approximately 20 minutes of walking her feet ache to the point that she cannot continue. Her blood pressure began to increase 3 years ago, for which she takes medication and tries to limit her intake of salty foods. Her favorite hobby is reading cookbooks and trying new recipes. She is conflicted about her love of cooking and cookbooks in light of the fact that she would like to lose weight. As evidence of her motivation, she cleaned out the kitchen cupboards and planned ways to use up high-calorie, high-fat foods by inviting friends over for meals. She has no known food allergies, enjoys all types of foods, and typically eats 2,100–2,200 calories per day.

**Questions**

1. Assess Samantha's weight status by determining her BMI, degree of overweight, and the significance of her waist circumference and waist to hip ratio. What health risks are associated with her anthropometric measurements?
2. Determine the ideal weight range and a realistic weight range for her. How much weight does she need to lose to improve her health status?
3. Explain the relationships between obesity and the following factors: pain in knees, ankles, and feet; increased blood pressure; blood sugar in the pre-diabetes range.
4. Determine a recommended calorie level and rate of weight loss, and justify the levels you selected.
5. What tools are available to assist her with tracking calories, portions, and nutritional content?
6. Using a food exchange system, plan one day's portions of food for the calorie level you selected. Distribute the food across three meals and one snack.
7. Discuss common types of prescription weight loss medications. What are the advantages and disadvantages of using these medications? Does Samantha fit the guidelines used by physicians when prescribing them?
8. Exercise is a necessary component of a weight loss plan. What recommendations can you give to help her increase her tolerance of exercise, and what exercise is appropriate?
9. Is there a way for Samantha to continue her hobby of reading cookbooks and preparing new recipes as she begins a weight loss plan?
10. What other suggestions can you give to help Samantha get out of the kitchen so she isn't surrounded by food?

## Issues to Debate

1. What are your views on bariatric surgery? Conduct a literature search to find at least one research article that shows a positive effect of bariatric surgery and one that does not. Find a recent (within a year) review article that describes each different type of bariatric surgery and when it needs to be performed. Debate both sides of this issue, providing background from your literature search. Include the pros and cons for prescribing physical activity to individuals who are morbidly obese and, hence, are candidates for bariatric surgery.

## References


Booth, A. O., Nowson, C. A., Huang, N., Lombard, C., & Singleton, K. L. (2006). Evaluation of a brief pilot nutrition and exercise intervention for the prevention of weight gain in general practice patients. *Public Health Nutrition, 9*, 1005–1061.

Centers for Disease Control and Prevention. (2003). Prevalence of physical activity, including lifestyle activities among adults—United States, 2000–2001. *Morbidity and Mortality Weekly Reports, 52*, 764–769.

Centers for Disease Control and Prevention. (2006). Obesity. Defining overweight and obesity. Retrieved from www.cdc.gov/obesity/adult/defining.html

Centers for Disease Control and Prevention. (2011). How much physical activity do you need? Retrieved from www.cdc.gov/physicalactivity/everyone

Centers for Disease Control and Prevention. (2013). Adult participation in aerobic and muscle-strengthening physical activities—United States, 2011.*Morbidity and Mortality Weekly Reports, 62*, 326–330.

Donnelly, J. E., Blair, S., Jakicic, J. M, Manore, M. M., & Rankin, J. W. (2009). American College of Sports Medicine position stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise, 41*, 459–471.

Foster, G. D., Wadden, T. A., Vogt, R. A., & Brewer, G. (1997). What is a reasonable weight loss? Patients' expectations and evaluations of obesity treatment outcomes. *Journal of Consulting and Clinical Psychology, 65*, 79–85.

Hamman, R. F., Wing, R. R., Edelstein, S. L., Lachin, J. M., Bray, G. A., Delahanty, L., … Wylie-Rosett, J. (2006). Effect of weight loss with lifestyle intervention on risk of diabetes. *Diabetes Care, 29*, 2102–2107.

Hill, J. O. (2006). Understanding and addressing the epidemic of obesity: An energy balance perspective. *Endocrine Reviews, 27*, 750–761.

Hill, J. O., Wyatt, H. R., Reed, G. W., & Peters, J. C. (2006). Obesity and the environment: Where do we go from here? *Science, 299*, 853–855.

Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients)*. Washington, DC: National Academies Press.

Jakicic, J. M., Clark, K., Coleman, E., Donnelly, J. E., Foreyt, J., Melanson, E., … American College of Sports Medicine. (2001). American College of Sports Medicine position stand: Appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise, 33*, 2145–2156.

Jakicic, J. M., Marcus, B.H., Gallagher, K.L., Napolitano, M., & Lang, W. (2003). Effect of exercise duration and intensity on weight loss in overweight, sedentary women. *Journal of the American Medical Association, 290*, 1323–1330.

Jakicic, J. M., Winters, C., Lang, W., & Wing, R. R. (1999). Effect of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: A randomized trial. *Journal of the American Medical Association, 282*, 1554–1560.

Jay, M.R., Gillespie, C. C., Schlair S. L, Savarimuthu, S.M., Sherman, S.E., Zabar, S. R., & Kalet, A. L. (2013). The impact of primary care resident physician training on patient weight loss at 12 months. *Obesity Journal*, *21,* 45–50.

Levine, J. A. (2005). Measurement of energy expenditure. *Public Health Nutrition, 8*, 1123–1132.

Levine, J. A., Lanningham-Foster, L. M., McCrady, S. K., Krizan, A. C., Olson, L. R., Kane, P. H., … Clark, M. M. (2005). Interindividual variation in posture allocation: Possible role in human obesity. *Science, 307*, 584–586.

Levine, J. A., Vander Weg, M .W., Hill, J. O., & Klesges, R. C. (2006). Non-exercise activity thermogenesis: The crouching tiger hidden dragon of societal weight gain. *Arteriosclerosis, Thrombosis, and Vascular Biology, 26*, 729–736.

Lichtman, S. W., Pisarska, K., Berman, E. R., Pestone, M., Dowling, H., Offenbacher, E., … Heymsfield, S. B. (1992). Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *New England Journal of Medicine, 327*, 1893–1898.

McQuigg, M., Brown, J., Broom, J., Laws, R. A., Reckless, J. P., Noble, P. A., … Counterweight Project Team. (2005). Empowering primary care to tackle the obesity epidemic: The Counterweight Programme. *European Journal of Clinical Nutrition, 59*(Suppl. 1), S93–S100.

Morrato, E. H., Hill, J. O., Wyatt, H. R., Ghushchyan, V., & Sullivan, P. W. (2006). Are health care professionals advising patients with diabetes or at risk for developing diabetes to exercise more? *Diabetes Care, 29*, 543–548.

Ness, M., Barradas, D., Irving, J. & Manning, S. (2012). Correlates of overweight and obesity among American Indian/Alaska Native and Non-Hispanic White children and adolescents: National survey of children's health, 2007. *Maternal and Child Health Journal, 16*(Supp 2), 268–277.

Novak, C. M., Zhang, M., & Levine, J. A. (2006). Neuromedin U in the paraventricular and arcuate hypothalamic nuclei increases non-exercise activity thermogenesis. *Journal of Neuroendocrinology, 18*, 594–601.

Phelan, S., Wyatt, H. R., Hill, J. O., & Wing, R. R. (2006). Are the eating and exercise habits of successful weight losers changing? *Obesity, 14*, 710–716.

Rodearmel, S. J., Wyatt, H. R., Barry, M. J., Dong, F., Pan, D., Israel, R. G., … Hill, J. O. (2006). A family-based approach to preventing excessive weight gain. *Obesity, 14*, 1392–1401.

Thomas, S., Ness, R. B., Thurston, R. C., Matthews, K., Chang, C. C., & Hess, R. (2013). Racial differences in perception of healthy body weight in midlife women: Results from the Do Stage Transitions Result in Detectable Effects study. *Menopause*, *20*(3), 269–273.

U.S. Department of Agriculture. (2010). Choose MyPlate. Retrieved from www.ChooseMyPlate.gov

U.S. Department of Health and Human Services. (1996). *Physical activity and health: A report of the Surgeon General*. Atlanta, GA: Author.

U.S. Department of Health and Human Services. (2008). *2008 physical activity guidelines for Americans*. Retrieved from www.health.gov/paguidelines

U.S. Department of Health and Human Services. (2010). *Healthy People 2020*.Washington, DC: U.S. Government Printing Office. Retrieved from www.healthypeople.gov

U.S. Department of Health and Human Services & U.S. Department of Agriculture. (2010). *Dietary Guidelines for Americans, 2010* (7th ed.). Washington, DC: U.S. Government Printing Office. Retrieved from www.health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf

Ventham, J. C., & Reilly, J. J. (1999). Reproducibility of resting metabolic rate measurement in children. *British Journal of Nutrition, 81*, 435–437.

Wadden, T. A., Womble, L. G., Sarwer, D. B., Berkowitz, R. I., Clark, V. L., & Foster, G. D. (2003). Great expectations: "I'm losing 25% of my weight no matter what you say." *Journal of Consulting and Clinical Psychology, 71*, 1084–1089.

Ward, A. (1999). Exercise and exercise intervention in the prevention of coronary artery disease. In J. M. Rippe (Ed.), *Lifestyle medicine* (pp. 90–97). Malden, MA: Blackwell Science.

CHAPTER 12

# Special Topics in Nutrition and the Older Adult: Diet, Lifestyle, Disease, and Pharmacologic Considerations

*Roschelle Heuberger, PhD, RD, and Ronni Chernoff, PhD, RD, FAND, FASPEN*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Identify what is unique about older adult compared to middle-age or younger adults.
2. Discuss the epidemiology of the aging cohort.
3. Discuss the direct and indirect determinants of aging.
4. Evaluate the theories of human aging and determine which theory seems most likely to be accurate.
5. Delineate differences between the determinants of successful aging.
6. Link the factors that are common issues in hospitalized/institutionalized older adult and relate them to morbidity and mortality and adequate nutrition.
7. Describe pharmacologic considerations in the treatment of diseases in older persons, inclusive of the issues of polypharmacy.

## Pharmacotherapeutic Intervention in Obesity

Older adults are a diverse and extremely heterogeneous population group. Aging is a uniquely individual process that is influenced by an accumulation of life events. Differentiation between chronologic age and **senescence** must be taken into account. Some 70-year-old persons are more fit and less "aged" from a cellular and functional standpoint than are some 40-year-olds as a result of a variety of genetic, health status, and lifestyle characteristics. However, functional declines naturally occur with advancing age. Elderly people are often categorized by chronological age, with stratification so that the cohort of 65- to 74-year-olds is referred to as the "young old," 75- to 84-year-olds as "old," and those 85 years and older as the "oldest old." The oldest old group usually exhibits the physical changes most commonly associated with senescence (Singer & Manton, 1998). Older adults comprise the most rapidly growing demographic in the United States as well as in other industrialized countries. Life expectancies have increased as a result of a number of factors:

**senescence** The process of aging taking into account cellular death and dysregulation leading to functional decline.

- Declines in mortality from infectious diseases
- Increases in technologic ability to detect disease at an early juncture
- Advent of new pharmacotherapies for a variety of acute and chronic disease states
- Increases in awareness of antecedents for chronic disease among the general population
- Overall better access to health care
- Overall better nutrition and sanitation
- Overall technologic enhancements or preventative measures that may determine better health status over the life span (e.g., better automotive safety, stricter seat belt regulations)
- A better educated cohort of older adults

As a result of the population living longer and the aging of the baby boomers, the population group born between 1946 and 1964, there is a current critical need for education regarding the requirements of older adults. The interrelationships among many factors, including nutritional status, socioeconomics, lifestyle characteristics, environmental considerations, comorbid conditions, and genetics, must be evaluated concurrently in this population. An overview of the nutrition and health issues associated with an older adult population and senescence, determinants of health and nutritional status, and consequences of aging and disease follows.

## Older Adults

For each individual, aging is a unique process, so classifying people by their chronologic age may be misleading about their needs. This consideration is a challenge when developing policies designed to

meet the needs of this large proportion of the population. For government programs, such as food assistance programs, taxation, and retirement, a person older than age 65 years is considered to be an older adult. In the social sciences, the definition of *aged* is related to functional status and not chronologic age. Functional ability is most often related to psychological status; ability to perform activities of daily living (ADLs) such as bathing, cooking, and transferring; medical status; and socioeconomics.

For the specialty practice area of geriatrics, the terms *geriatric patient* and *frail elderly* are tied to functional, medical, and cognitive status. The term *frailty* actually refers to a complex syndrome that includes multiple organ systems decline, loss of physiologic reserve, and increased vulnerability to disease and death. There is also an increased risk for social isolation and institutionalization (Cramm & Nieboer, 2012; Moorhouse & Rockwood, 2012). The definition of *frail* has frequently referred to those older adults who are severely underweight, but there has been a recent shift to include normal or overweight persons in the category of frail if they have **sarcopenia**, several chronic and acute diagnoses, poor nutritional status, and a decreased ability to function (Weinrebe, Guneysu, & Welz-Barth, 2002). *Sarcopenia* refers to loss of lean body mass. This loss is related to an increasingly sedentary lifestyle, being chair- or bedbound, and having poor dietary intake. Sarcopenic obesity, characterized by a decrease in lean body mass and an increase in adiposity, may develop because of a diet poor in protein but high in calories. In recent years, this has attracted a lot of concern because of the increased metabolic burden associated with low muscle mass and obesity (Prado, Wells, Smith, Stephan, & Siervo, 2012).

**sarcopenia** Decrements in muscle mass that result in declining functional ability.

There is an increasing need to develop a uniform classification system for older adults similar to the categorization of the developmental stages of infants, children, and adolescents. Currently, the information regarding subgroups within the older adult population is lacking, and gross generalizations often result from research in this area. Each older adult must therefore be considered individually for preventative measures, nutritional recommendations, and treatment options based on the totality of the information available (Keller, Ostbye, & Goy, 2003).

## Epidemiology

The "graying" of America currently taking place with the aging of the baby boomers is considered to be a healthcare crisis, with insufficient practitioners, facilities, and accommodations for the changing demographics. There will be fewer working adults and an enormous drain on the economy with large numbers of poor elderly persons requiring extended care. Factors that may affect individual aging rates include such diverse occurrences as genetic profile (Wilmoth, 2000), food supply, social circumstances, political events, exposure to disease, climate and natural disasters, and other environmental events (Seeman & Crimmins, 2001). Efforts to promote a healthy aging agenda for the population are under way, despite the fact that this national campaign should have commenced three decades ago to maximize effects for those who are entering late life.

**Learning Point** If current trends hold, by the year 2050 one in four persons in the United States will be older than age 65 years. The oldest old are the most rapidly increasing segment of this population. Women (minority women, in particular) will soon be the dominant demographic in this oldest old category (U.S. Bureau of the Census, 2005).

During the 1990s, the population age 85 years and older increased from 3.1 million to 4.2 million, a 38% growth. This 85+ population is anticipated to grow from 4.2 million to 6.6 million in 2020, to 8.9 million by 2030, and to 19 million by 2050. The number of centenarians, people age 100 years or older, is projected to increase from 37,000 in 1990 to approximately 131,000 (middle estimate based on population projections) in 2020 (Chernoff, 2013a).

Characteristics of the portion of the U.S. population older than the age of 65 years include a number of regional differences; warmer-climate states usually have an overrepresentation of certain segments of the elderly population. Older adults are among the poorest of the poor, especially older women who were homemakers and who do not receive added benefits from having worked outside the home. Women also live longer than men, and when they reach the oldest old category they have depleted any resources they may have had and require more medication, health care, and assistance, which results in the need for greater fiscal resources. Minority women make up the poorest of the poor among this demographic.

The number of elderly adults who live alone is estimated at 10%, and 23% of households are estimated to include an elderly person. Asian and Hispanic descent is correlated with increased numbers of older adults as part of households, whereas African American and white descent correlates with increased numbers of elderly individuals who live alone (see **Table 12.1**).

There are differences in population demographics by race as well as by age and sex. By 2030, individuals who are in minority groups are projected to be 25% of the total older population; by 2050, it is projected that the minority elderly population will be 42% (Administration on Aging, 2011; Vincent & Velkoff, 2010). In 2002, non-Hispanic whites accounted for 80% of the older-than-65-years population, 86% of those 75 to 84 years, and 87% of those 85 years and older (He, Sengupta, Velkoff, & DeBarros, 2005). By 2050, the percentage of non-Hispanic whites older than age 65 years will have fallen to 77%; 12% will be African American, and 9% will be Asian (Vincent & Velkoff, 2010). American Indians and Alaska Natives represent 3.6%, Asians and Pacific Islanders are 2.9%, and Hispanics are 4.1% of the group 85 years old and older. By 2050, of those 85 years and older, 81% are projected to be white, 10% will be African American, and 6% will be Asian (He et al., 2005). Presently, there are many more elderly white people than there are elderly African Americans, despite a proportionately higher birth rate among African Americans. This discrepancy may be the result of a higher mortality rate at younger ages for African Americans as well as increased mortality from hypertension, less access to preventive healthcare services, and delays in seeing physicians until the later stages of disease (Eggers & Greenberg, 2000; Niefeld & Kasper, 2005). Although the early mortality rate for African Americans is expected to decrease, it is not expected to compensate for the gap between the races in the near future. Between 1990 and 2030, the elderly African American population is expected to grow by 159%.

Epidemiologic data on older adults are available from a number of federal, state, local, nonprofit, and private sector sources. The federal government National Health and Nutrition Examination Survey (NHANES), which has been ongoing, collected data on an oversampling of the elderly population in the United States. A variety of statistics is available regarding socioeconomics, blood values, and dietary data. Medical and behavioral measures were also obtained for the older population in the National Health and Nutrition Examination Survey series. Behavioral Risk Factor Surveillance Studies (BRFSS) also have included older adults. Conducted at the state level, these assessments are available for specific samples of elderly in most of the 48 contiguous states. Variables regarding demographics, health risk factors, and some dietary information are available.

The National Institute on Aging, a branch of the National Institutes of Health, promotes epidemiologic inquiry on older Americans, and organizations such as AARP (formerly known as the American Association for Retired Persons) and the Administration on Aging (AoA) also routinely collect information on the older-than-65-years

**TABLE 12.1** **Demographic Characteristics (United States)**

| Unadjusted Percentages for the U.S. Population | Male | Female |
|---|---|---|
| Percentage over the age of 65 years in the United States (2000) | 5% | 7% |
| Percentage of persons over the age of 85 in the United States (2000) | 0.5% | 1.0% |
| Percentage of persons over the age of 85 within the elderly population in the United States (2000) | 30% | 70% |
| Projected increases in the over-the-85-year age range among the elderly population in the United States by 2010 | +1.5% | +3.5% |
| Rates for persons over the age of 65 years in the United States | 2,000 | 2,002 |
| Percentage of older adults with a disability | 35% | 36% |
| Percentage of older adults widowed, single | 31% | 32% |
| Percentage of older adults with caretaking responsibilities | 42% | — |

Data from United States Census Bureau.

population. In addition, a number of colleges and universities across the nation have institutes or centers on aging and conduct aging research.

Corporate data from assisted living facilities or long-term care are extremely useful as an index of the utilization of different providers and resources for the aged population and, more important, provide critical data for the oldest old, who are routinely underrepresented in government surveys because of those initiatives being targeted at the noninstitutionalized (free-living) population in the United States.

Information on the oldest old has been sparse in the past, but, because of the increasing numbers of this demographic, new directives have been issued to try to capture as much information as possible on this age group to enhance care and better understand issues of concern for healthcare providers, family, and the oldest old themselves.

## Aging

### Aging Theories

Theories regarding aging include evolutionary, free radical, gene regulation and telomere (human genome), inflammation, and immune theories, among others (Cesari, Vellas, & Gambassi, 2012). Cellular aging theories have the most experimental evidence to back them. "Wear and tear" theories, as they are sometimes called, state that continuous use coupled with decreases in the division and maturation of new cells cause aging and ultimately death. **Apoptosis** eventually results in decreased numbers of viable cells. The free radical theory of aging involves cumulative damage as a result of natural free radical oxidative changes, which over time result in increased antigenicity, protein changes, and oxidative DNA damage.

**apoptosis** Preprogrammed cell death.

The sum of these processes results in cellular senescence and organ system failure, ending in death. Alterations in genetic coding suggests that aging is the result of changes in protein synthesis and messenger RNA processing. Chemical alterations in DNA over time, coupled with translational aberrations, are the cause of aging and death (Morley, Glick, & Rubenstein, 1995; Rimkus, Melinchok, McEvoy, & Yeager, 2005).

Caloric restriction, with adequate vitamin and mineral nutriture, has been associated with longevity. This is thought to be the result of decreases in oxidative stress, less metabolic free radical generation, and decrements in the abnormal compounds generated as a result of redox damage. The underlying mechanisms have yet to be fully established, and caloric restriction research has only been conducted in animal models (Cesari et al., 2012).

Multiple theories link inflammatory processes with aging; however, it seems most likely that various mechanisms are at work simultaneously (Jenny, 2012). Aging is a cumulative process that evolves over time and is not linked to a specific traumatic event or defining occurrence.

### Determinants of Successful Aging

Successful aging with little disease or disability involves many factors. Dietary considerations across the life span, such as increased intake of fiber, antioxidants, and minerals or caloric restriction, have been correlated with increased longevity and decreases in morbidity, although the literature is predominantly focused on animal studies (Maxmen, 2012). Patterns of intake have also been shown to relate to successful aging. Characteristics of eating such as eating small, frequent meals, sitting down with family, having a glass of wine with dinner, and eating until 80% full seem to have beneficial effects over the long term.

Lifestyle characteristics such as maintenance of a normal body weight, decreased exposure to known carcinogens such as tobacco or heavy metals, and use of coping mechanisms to deal with stress seem to promote healthy aging. Adequate exercise and sleep are also important. Drug and alcohol use along with life course levels of stress have been shown to have significant impact on cellular senescence.

There are other indirect determinants of successful aging with decreased cellular senescence. Some of these include mental status, mental activity, pet ownership, and social interaction. Sociodemographic characteristics also play a role, such as marital status, level of education, income, race, and ethnicity. Quality of life and perceptions of the quality of life along with spirituality, civic engagement, and coping abilities are moderating influences on the mechanisms involved in healthy longevity. Ultimately, genetic makeup and the propensity toward maintaining cellular protection against environmental influences determine the course of the individual's life span (Bamia, 2005; De Groot, Verheijden, de Henauw, Schroll, & van Staveren, 2004).

### Diet

Dietary determinants of successful aging include issues with both macro- and micronutrient intake. Caloric content across the life span, adequate (but not excessive) protein intake with moderate to high bioavailability, and high complex carbohydrate consumption are all associated with decreases in morbidity and mortality (Jungjohann, Luhrmann, Bender, Blettner, & Neuhäuser-Berthold, 2005; Millward, 2004). Decreased total fat intake, with greater proportions of monounsaturates and long-chain omega-3 fatty acids and low amounts of trans/saturated fats, is associated with decrements in vascular anomalies and chronic disease with mortality (Hjerkinn et al., 2005). Micronutrient content of the diet across the life span is directly related to disease and indirectly, but ultimately, related to mortality.

Healthy aging has been associated with increased intake of antioxidants, including phytochemicals; selenium (a component of glutathione peroxidase); vitamins A, C, and E; the carotenoids; and zinc (as part of metallothionein) (Chernoff, 2005; Faure, Ducros, Couzy, Favier, & Ferry, 2005; Gonzalez et al., 2004) (see **Figure 12.1**). In addition, bone health is improved with increased calcium, vitamin D, and vitamin K; adequate magnesium; and lower intakes of phosphorus during the course of the life span (Bischoff-Ferrari et al., 2004; Ryan-Harshman & Aldoori, 2004). Increased dietary potassium and lower levels of sodium are important for blood pressure and vascular health, and the B vitamins are critical for decrements in homocysteine levels (Cacciapuoti, 2012; Demingné, Sabboh, Rémésy, & Meneton, 2004; Gori et al., 2005; Hamilton & Hamilton, 1997).

**FIGURE 12.1** Vegetables are an excellent source of fiber, minerals, vitamins, phytochemicals, and organic constituents, all known to be beneficial for health because of a variety of effects, including their antioxidant capabilities. Increased vegetable consumption over the life span is associated with decreased cellular senescence, morbidity, and mortality.



#### Assessment Methodology

A number of methods for assessing the older adult on a large scale, such as with national nutritional monitoring systems or in community studies, have been used over the years. Quick screening tools such as the DETERMINE (Disease, Eating poorly, Tooth loss/mouth pain, Economic hardship, Reduced social contact, Multiple medications, Involuntary weight loss or gain, Needs assistance in self-care, Elder years) checklist and the Mini Nutritional Assessment have been shown to be useful in large-scale surveillance and in small-scale clinical settings (see http://www.aafp.org and http://www.mna-elderly.com to access these screening tools). Both instruments along with others designed for specific populations have been shown to be effective for flagging older adults with issues related to nutritional status and health. The Mini Nutritional Assessment, in particular, has been the subject of recent research and has been tested in a variety of populations and languages (Beck, Ovensen, & Schroll, 2001; Chernoff 2013b; Gazzotti, Albert, Pepinster, & Petermans, 2000).

Assessment of nutritional status in older adults is somewhat challenging because of the lack of appropriate standards and the heterogeneity of the older population. Anthropometric measures reflect physiologic and physical changes associated with advanced age. Although, in otherwise healthy elderly subjects, biochemical measures should be within the normal range, many older adults have multiple chronic conditions that may be a factor in biochemical measures. Serum albumin is a frequently used determination of nutritional health; however, it may be altered by cardiac, liver, or kidney disease or cancer or inflammatory processes (Sullivan, 2001). Serum albumin, along with other biochemical measures, is affected by hydration status (Mitchell, 2013).

One dimension that is lacking in general physical assessment tools is a review of the activities of daily living (ADLs) and the instrumental activities of daily living (IADLs). ADLs are a marker of an individual being able to live and manage self-care on his or her own (bathing, grooming, ambulating, toileting, feeding oneself, and dressing).

IADLs address the ability to live and manage tasks required to live independently (ability to use the telephone, shopping, preparing food, light housekeeping, doing laundry, traveling independently, managing medications, and handling finances). It has been shown that inability to complete ADLs is a harbinger of negative outcomes with any future illness (den Ouden, Schuurmans, Mueller-Schotte, van der Schouw, 2013; Mitchell, 2013).

### National Health Objectives

The National Institute on Aging (NIA) has reported that levels of disability among the older adult population in the United States have been falling since 1982 because of a variety of factors, such as better technology and increased access to health care. However, the NIA proposed the following initiatives with regard to national efforts toward bettering the older segment of U.S. society:

- Sustain decreases in disability, maintaining health and function in older adults.
- Improve strategies for promoting healthy behaviors among the older population, including the following:
  - Diet
  - Smoking
  - Alcohol abuse
  - Safety
  - Exercise
  - Evaluation of hormone replacement therapy
  - Evaluation of dietary supplements
  - Improved interaction with the healthcare system
  - Reduction in caregiver and family stress
  - Improvement in individual coping with chronic disease

Each area was identified as being critical to maintaining the current decline in morbidity and mortality, despite the increasing numbers of older adults within the population (http://www.nia.nih.gov/research/dbsr/initiative-global-aging). These recommendations to researchers and healthcare professionals were borne out of the epidemiologic evidence collected over the past few decades. In addition, public health campaigns, such as Healthy People 2020, have provided general objectives and preventive initiatives designed to promote healthy outcomes in the health, function, and quality of life of persons older than the age of 65 years. Other groups have also targeted the lay older adult, including the American Diabetes Association (ADA) and the American Cancer Institute (ACI), with media campaigns specifically targeting elderly diabetics and cancer survivors. High in priority is injury prevention, particularly fall prevention. The Affordable Care Act (ACA) of 2010 includes provisions to ensure quality care and safety in any healthcare setting. One of the emerging issues in Healthy People 2020 is the stated goals of care coordination, independence in managing care, and training for people who care for older adults (Institute of Medicine, 2008).

### Macronutrient Intake

In older adults, adequate protein intake is essential for the prevention of muscle mass losses, pressure ulcers, and decreased immunocompetence. Sarcopenia in older adults is associated with poor outcome, frailty, and falls. Investigations into the roles of essential amino acids in prevention of these issues have shown that adequate protein intake is important to the replacement of aging muscle tissue as well as in the maintenance of bone health (Wolfe, 2012). In conjunction with weight-bearing exercise, essential amino acids can increase the concentrations of insulin-like growth factor I, which has a **permissive effect** on bone and muscle cellular repair and, to some extent, replication. It should be noted that protein consumption is related to calcium and phosphorus intake, which mediates the relationships between protein intake and bone health (Santesso et al., 2012; Takeda, Yamamoto, Yamanaka-Okumura, & Taketani, 2012).

**permissive effect** Factors that aid in the pathologic effect of another factor or set of antecedents.

Other sets of variables related to protein adequacy are socioeconomics and institutionalization. Poor and institutionalized elderly adults have reduced high-quality protein intakes and often have other factors that affect muscle mass, bone health, and wound healing, for example, being chairbound, bedbound, or otherwise immobilized. The provision of protein supplements to these populations has been shown to be beneficial in decreasing morbidity (Coker & Wolfe, 2011; Collins, Kershaw, & Brockington, 2005; Ginty, 2003; Milne, Potter, & Avenell, 2005).

Fiber intake, along with adequate fluid consumption, in older persons is important for managing constipation, which results from enteromuscular changes consistent with aging. Both soluble and insoluble fiber sources are needed to slough abnormal cells from the gut, increase stool quality

and frequency, and trap bilious cholesterol, drug metabolites, and carcinogens. In addition, fiber sources reduce calories and improve blood glucose level stability (Ledikwe et al., 2004).

Fiber from whole foods also is rich in micronutrients, but intake of whole foods containing fiber often is reduced in older persons, especially in those with poor dentition. Edentulous older persons often cannot chew high-fiber whole foods and so avoid them. Poorer older persons may not be able to afford fresh, high-fiber foods, such as produce, which are more expensive. Older persons who have not had dietary patterns consistent with a high intake of high-fiber foods are reluctant to introduce them in late life and often exclude fruits, vegetables, legumes, and whole grains. Older adults also may have issues with fresh high-fiber foods because of their gas-forming potential. The addition of soluble and insoluble fiber sources to the diet of the older adult should be gradual. The use of bulking agents or fiber supplements may provide an acceptable option, and the untoward effects may be reduced by the addition of a supplemental form of normal gut flora, such as probiotics (Bhutto & Morley, 2008).

Blood glucose control is important for decreasing glycation products, which, according to theory, are a contributing factor to senescence. Antioxidants from high-fiber fruits and vegetables are important for controlling free radical damage. Another theory regarding cellular senescence and the decrease in caloric content seen in high-fiber diets is associated with the maintenance of normal body weight, a factor in healthy aging and longevity. In addition, the sloughing of abnormal cells through high-fiber dietary intake reduces carcinogenesis and disease (see **Figure 12.2**). Fiber intake also traps cholesterol and reduces atherosclerotic tendencies, associated with healthier aging.

Cholesterol, trans fats, saturated fats, and total fat intake over the life span has been shown to affect morbidity and mortality in the aged (Anderson, Suchindran, Kritchevsky, & Barrett-Connor, 2004; Cabrera, Andrade, & Dip, 2012; Singh et al., 2012). In terms of overall macronutrient intake, these compounds have been shown to interact with genetic predisposition to increase vascular adverse events and morbidity more than any other dietary constituent. Inflammatory processes that mediate the effects of genetic interactions involve a multitude of eicosanoids, **cytokines**, blood constituents such as platelets, hormones, and other cellular signals. In addition, body weight, energy expenditure, and environmental exposure serve to moderate the progression of disease in the face of increased intake (Okuyama, Ichikawa, Fujii, Ito, & Yamada, 2005). Cellular senescence as a result of increased fat and cholesterol intake may be related to free radical and nitric oxide generation and decrements in cellular repair with continued inflammatory response. Inflammatory markers such as C-reactive protein, sP-selectin, and sICAM-1 decrease with low fat intake over time.

**cytokines** A class of cellular chemical messengers often involved in cascade reactions such as with inflammatory response.

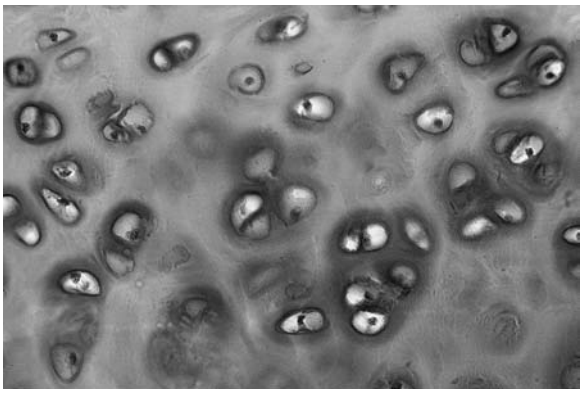


**FIGURE 12.2** Abnormal cell. Cellular abnormalities increase with age. The etiology of these changes is complex and multifactorial. Several mechanisms are thought to be integral to the disruption of cellular processing and homeostatic capability.



### Micronutrient Intake

In terms of micronutrient status, several vitamins and minerals have been linked to healthy aging. The association between maintenance of normal cellular function and vitamin or mineral status is also moderated by cumulative oxidative stress and inflammatory response. It is common knowledge that antioxidants from fruits, vegetables, and whole grains prevent oxidative damage. Because oxidative stress is related to disease states from atherosclerosis to bone and joint health, investigations into the use of foods versus vitamin and mineral supplementation have been performed (Lichtenstein & Russell, 2005; Riccioni et al., 2007). Whole fresh foods are preferable to increasing antioxidant capabilities because so many additional compounds are contained within the food in forms that make them enhance the action of select micronutrients. The trace compounds in foods include organic acids, sulfur and nitrogenous compounds

that aid in uptake, activation, and utilization of vitamins and minerals, in addition to having their own direct benefits (Boudville & Bruce, 2005).

The relationships of the B vitamins to indices of cellular senescence have been the focus of recent research. Because homocysteine has been related to oxidative damage and diseases ranging from cardiovascular disease to neurodegenerative diseases of aging, such as Alzheimer's disease, vitamin $B_{12}$, vitamin $B_6$, and folate have been studied for their impact on disease over the life span (Bottiglieri & Diaz-Arrastia, 2005; Maiese, Chong, Hou, & Shang, 2009; Riccioni et al., 2007). Although **neurodegenerative disease** is thought to have a genetic component that makes the individual more susceptible with the appropriate environmental and dietary stimuli, dietary components have been shown to turn on select genes in a wide variety of models (Fenech, 2010).

**neurodegenerative disease** Diseases that involve malfunction of the central and peripheral nervous tissue. These often are diseases that progress slowly but insidiously and result in progressive loss of function and eventual mortality. Examples include Alzheimer's disease and Parkinson's disease.

Neurodegenerative diseases, which involve atherosclerotic changes such as vascular dementia, are also linked through the cycle involving methionine conversion, with methylmalonate, homocysteine, and S-adenosyl-methionine intermediates, each playing a role in cellular dysregulation and changes to DNA blueprinting (Quadri et al., 2004).

Homocysteine is also thought to be a modifiable risk factor for neurodegenerative diseases because of its direct neurotoxicity at high levels; however, there may be unintended consequences of folic acid supplementation such as elevated glucose levels and altered lipid profiles (Chmurzynska, Malinowska, Twardowska-Rajewska, & Gawecki, 2012). Alterations in the **methylation reactions** essential for normal neurotransmitter synthesis are impaired at high levels of homocysteine and its metabolites (Tucker, Qiao, Scott, Rosenberg, & Spiro, 2005). High homocysteine levels are a result of low folate levels, and low folate levels are thought to be endemic in the elderly population in areas where mandatory folate fortification is not practiced (Wolters, Hermann, & Hahn, 2005). Decreased vitamin $B_{12}$ absorption also is common in elderly persons who have atrophic gastritis or some other risk for the reduction of intrinsic factor production from the parietal cells lining the stomach. Older adults are also more vulnerable to B vitamin insufficiency as a result of dietary choices and medication usage, which potentially interferes with bioavailability (Bross, Soch, & Smith-Knuppel, 2010; Ravaglia et al., 2005). Cellular uptake and conversion of folates and cyanocobalamin also may be diminished as a function of aging. Receptor function and transporter capabilities naturally decline with aging. Supplementation with B vitamins or the addition of B vitamins to foods as with fortification may be an option for elderly persons or may be unnecessary; there is some disagreement among experts (McCormick, 2012). In the absence of severe decrements in transport and absorption, oral supplementation can raise levels of serum B vitamins in an older adult.

**methylation reactions** The process of attaching a methyl group to an existing compound through 1 C transfer by several vitamin/mineral cofactors.

The B vitamins (folate, in particular) are related to the ability to synthesize de novo methionine and S-adenosylmethionine (SAM-e), which is a factor contributing to depressive symptoms. SAM-e is a universal methyl donor. Methylation reactions are essential for the synthesis of neurotransmitters such as serotonin in the brain and throughout the body. Serotonin receptors are not exclusive to the brain, and there are colonies of serotonin receptors throughout every organ system in the body. Serotonin is a chemical mediator for many homeostatic processes including but not limited to mood. SAM-e has been shown to have potential as an antidepressant. Folate is necessary for SAM-e participation in methylation reactions, and elevated SAM-e production can be achieved through supplementation of folate in older adults (Ramos, Allen, Haan, Green, & Miller, 2004).

Dietary intake and functional B vitamin status is thus linked to cellular senescence through a number of pathways. The impact of **hyperhomocystinemia** on increasing oxidative stressors, with associated inflammatory response, is a modifiable risk factor for aging and cellular pathology. B vitamins are essential to the regulation of homocysteine. Therefore, B vitamin status is thought to be of importance to healthy aging.

**hyperhomocystinemia** Elevated blood levels of homocysteine, the sulfur-containing amino acid metabolite.

## Dietary Patterns

Dietary patterns are important to aging processes and cellular senescence, albeit indirectly. Differences have been investigated in the relationship between consumption patterns and inflammatory response

in older persons, and the indirect relationships seem to have an effect on the progression of cellular dysregulation and ultimately disease. This can be seen in the advent of type 2 diabetes, where patterns of intake high in refined sugars and processed foods have been tied to inflammatory markers. High intake of sugared soft drinks, refined grains, diet drinks, lunch meat, and processed foods with low intake of vegetables is thought to increase C-reactive protein (CRP), interleukin-6, and **tumor necrosis factor-alpha** (Schulze et al., 2005).

**tumor necrosis factor-alpha** An indicator of inflammatory response and a mediator of several cascade mechanisms involved in immune function and homeostasis.

**Learning Point** Nutritional supplementation should be used when intake cannot provide fresh whole foods high in nutrient density (Dawson-Hughes, 2004). For certain fat-soluble vitamins such as vitamins D and E or minerals such as calcium, there may be situations in which supplementation provides benefits over obtaining these nutrients from whole foods, such as when dairy or fatty foods are not advisable as a result of the macronutrient content of the product (Deplas, Debiais, Alcalay, Bontoux, & Thomas, 2004; Nieves, 2005).

A Westernized dietary pattern of highly processed, high-fat foods has also been linked to morbidity through inflammatory processes and oxidative stress (Murumalla et al., 2012). Acculturation and the transition of other people to this consumption pattern have resulted in an increase of cellular changes and chronic diseases of aging. Changes in patterns of consumption, with fish as the dietary protein staple or omega-3-containing oils from sources other than fish, have been shown to modify inflammation through the eicosanoids and their mediating influences on the inflammatory cascade (Johnson & Fritsche, 2012; Payet et al., 2004). This decrease in inflammation over time with omega-3 fatty acid intake is directly related to cellular senescence through an accumulation of oxidative stressors.

Increased low-fat dairy consumption has also been shown to be part of a healthful dietary pattern. When supported by lifestyle counseling, increased consumption of dairy products was shown to produce favorable results (Moschonis et al., 2011). Increased levels of several essential nutrients (i.e., selenium, phosphorus) along with calcium and vitamin D may be obtained from dairy foods. Encouraging older adults to consume low-fat dairy products as part of an overall dietary pattern is important (Weaver, 2010). A caveat is that lactose intolerance increases with age, and arrangements must be made for use of lactose-free products, which are becoming more available (Savaiano, 2011).

Immune function declines also occur in old age (Pawelec, Larbi, & Derhavanessian, 2010) and are mediated by nutritional status (Pae, Meydani, & Wu, 2012). Increased infection results in increased oxidative stress. Cellular damage occurs with decrements in the immune system's ability to ward off pathologic changes such as in cancer or autoimmune disease.

Iron-deficiency anemia and pernicious anemia in older adults may be the result of factors such as "anemia of chronic disease," blood loss, or impaired nutrient absorption because of **achlorhydria**. Achlorhydria is a condition where the parietal cells lining the stomach do not produce adequate hydrochloric acid, which is needed in the absorptive process of iron, calcium, and vitamin $B_{12}$ (Bhutto & Morley, 2008). Regardless of the etiology of the anemia, more iron is needed for red blood cell synthesis and tissue oxygenation, and poor iron status caused by increased needs ultimately results in impaired immunity. Iron deficiency results in decreases in cell-mediated immune function, with decrements in T-cell proliferation and T-lymphocyte function (Ahluwalia et al., 2004; Bergman et al., 2005).

**achlorhydria** Absence or decrement in the level of hydrochloric acid in the stomach that results from decreased production by the parietal cells in the fundus of the stomach.

**Phagocytosis** and **bacteriostatic** capabilities of white blood cells are diminished (Bergman et al., 2005). Inflammatory response cytokines increase with poor iron status. It should also be noted that iron in and of itself is a potent **pro-oxidant** and must be bonded to a carrier or incorporated into a compound such as hemoglobin; it cannot travel freely without inducing free radical damage. Carrier proteins and compounds such as myoglobin and hemoglobin must be synthesized correctly and in adequate amounts. Protein must be adequate to synthesize these components, and all cofactors for cellular differentiation and maturation also must be available in addition to the iron.

**phagocytosis** The ability of white blood cells to engulf foreign bodies.

**bacteriostatic** The characteristic of immune cells, which can incapacitate bacterial invaders through engulfing, dissolution, or direct toxicity.

**pro-oxidant** A compound that normally has the potential to act as a free radical in its present oxidative state.

Similarly, older adults are susceptible to poor zinc status. Zinc-containing foods are expensive and may be difficult to obtain for older adults in

lower socioeconomic strata. Zinc needs may be elevated in older adults suffering from comorbid conditions (Belbraouet et al., 2007). Zinc malabsorption may occur because of gastrointestinal changes associated with aging or may result from a low-zinc diet. Metallothionein, the transporter for zinc, must be synthesized at optimal levels, which requires adequate protein, kilocalories, and other micronutrients. Because zinc levels are tightly controlled in human serum through negative feedback homeostasis, it often is difficult to determine whether older adults are deficient; however, it appears that deficiencies in both zinc and copper are related more to disease than to age (Belbraouet et al., 2007). Tests for zinc lack sensitivity and **specificity**. Zinc is inherently involved in the genetic regulation of immune component synthesis. Because zinc is so closely tied to immune function, there are rapid changes in cellular components such as lymphocytes and inflammatory factors such as interleukin-2 and interleukin-6 (Andree, Kim, & Kirschke, 2004).

**specificity** The ability of a measure actually to measure the component of interest.

Dietary intake of vitamins such as folic acid and vitamin C and adequacy of macronutrient intake also are essential to normal immune function. Because immunity is tied to cellular senescence through inflammatory responses, oxidative stressors, and glycation products, intake of protein, total kilocalories, carbohydrates, and vitamins such as folic acid and C is essential to healthy normal aging. Provision of adequate micro- and macronutrients to older adults has been shown to increase immune response, regardless of form. Dietary intake of nutrient-dense whole foods is always preferable, but supplementation with enriched products also produces desirable increases in immune function (Wouters-Wesseling et al., 2005). The use of prebiotics, ingredients that stimulate growth of beneficial intestinal microorganisms, and probiotics, live microorganisms believed to have beneficial effects on the host, have been touted to support immune function (Di Bartolomeo, Startek, & Van den Ende, 2012; Klaenhammer, Kleerebezem, Kopp, & Rescigno, 2012).

Nutritional status affects morbidity and mortality in older adults. Mediating factors for cellular senescence and overall homeostasis include dietary pattern, macro- and micronutrient intake and impact on cumulative oxidative stressors, increases in glycation, and damage induced by free radical production over time. Successful aging with improved long-term outcome is inherently attributable to macro- and micronutrient availability (Donini et al., 2004), although some believe it is necessary to explore further the efficacy of dietary modifications in disease progression (Meydani & Wu, 2007).

## Lifestyle Characteristics and Aging

Lifestyle characteristics of an individual can have a marked impact on cellular senescence in interaction with genetic predisposition. Many lifestyle attributes have been investigated in this regard:

- Physical activity
- **Body habitus**
- Environmental contaminant exposure
- Alcohol use
- Methylxanthine use
- Cigarette smoking
- Oral health
- Pet ownership
- Reproductive hormone levels
- Marital status
- Mental status
- Mental activity
- Socioeconomics
- Education
- Social support

**body habitus** The size and shape of the body; may also include perceptions of the size and shape of the body.

This partial listing of variables may moderate successful aging. The interactions that occur are extremely complex, and it is very difficult to assess the level of impact for any one of these factors alone, much less their interactions. Therefore, research efforts have opted to use these variables as a means of broadly classifying risk for morbidity and mortality (Brach, Simonsick, Kritchevsky, Yaffe, & Newman, 2004). The more risk factors per individual, the greater the cumulative effects in terms of cellular damage through oxidative stress, glycation, and free radical production.

### Physical Activity

Physical activity over the life span is important to healthy aging, theoretically preventing functional decline. Improvements in balance, prevention of the development of sarcopenia of old age, and improved mood and sleep are all important effects of exercise that relate to overall health in

aged adults. It should be noted that increased physical activity is associated with increased oxidative stress, normally a result of oxygen utilization in aerobic metabolism. However, physical activity is also associated with improved ability to combat the increases in free radical production, and sustained lifelong activity decreases the risk of oxidative cellular damage. Physical activity should be encouraged throughout the life span, thus improving appetite, enhancing weight control, decreasing visceral adiposity, and improving immune responsivity and glycemic control (Lee et al., 2004; Nied & Franklin, 2002).

### Body Habitus

Visceral adiposity is an extremely strong risk factor for disease, and its involvement in metabolic syndrome has been well studied (Wannamethee, Shaper, Morris, & Whincup, 2005). Insulin resistance, a hallmark of metabolic syndrome, increases over time as a result of increased levels of macronutrients in the blood and alterations in the fluidity of membranes of cells that hold the receptors for insulin, among other hormones (Jaques, Moeller, & Hankinson, 2003). Insulin resistance results in higher levels of blood glucose, which in turn results in an increase in glycation products, another factor associated with cellular senescence. Gluteal-femoral adiposity, on the other hand, does not result in the same changes in metabolism and is thought to be more "protective" than is visceral adiposity in terms of disease risks (Snijder, Visser, & Dekker, 2005). Recent evidence demonstrates that diet and exercise contribute to improved insulin sensitivity in obese older adults (Shah et al., 2009).

The caveat to the present knowledge is that less research has been done in this area on frail elderly adults and little is known about the profiles of these older adults. In the past, the term *frail* was believed to be antithetical to visceral obesity in older adults because frail elderly persons were thin and fragile (Kennedy, Chokkalingham, & Srinivasan, 2004). A perspective of frailty that includes older persons who may be obese is being recognized and discussed (Jensen, 2005; Jensen & Hsiao, 2010).

A factor that determines healthy aging is body habitus. Efforts to reduce visceral obesity in older adults, both men and women, should be undertaken. The risk for increased visceral adipose accumulation increases post menopause for women, such that it mirrors the risk for men at the same age (Chen et al., 2005). Estrogen production in women serves to increase gluteal-femoral deposition over truncal fat deposition, thus protecting women to some extent from swift development of metabolic syndrome (syndrome X) in the absence of other risk. It should be noted that body mass index (BMI) does not account for body habitus, and skinfold thickness measures have not been shown to be effective for assessing visceral adiposity (Snijder et al., 2002). These measures are very widely used despite these known issues because they are easy and inexpensive. Population standards exist for these measures, so practitioners can evaluate their patients against known references.

Pathologic processes stemming from visceral deposition result in increased cellular senescence, morbidity, and ultimately mortality. Therefore, assessment of abdominal stores should be routine for older persons. Waist circumference data are now being standardized for a variety of age, sex, and race groups (Snijder et al., 2005). In older persons waist circumference may be a better predictor of health risk than waist-to-hip ratio (Welch & Somwers, 2000).

### Alcohol Use

Alcohol intake is highly controversial with respect to morbidity, mortality, and aging in general. Differences in drinking patterns and the type of alcohol consumption have varied risks associated with them. Those risks are significantly different among races, genders, and ages and interact with lifestyle issues across the board. An example is a person who drinks one glass of red wine with dinner several nights per week for many years versus a person who engages in **binge drinking** of distilled spirits late in life as a result of depression or loss. The former is considered a healthy behavior; the latter is considered unhealthy and is associated with morbidity. The interactions among alcohol intakes at a variety of levels with diet, exercise, medications, and other issues are extremely complex. Because older adults are at risk for falls, take many different medications, and already suffer from a variety of medical conditions, it is best to recommend that alcohol intake be avoided in advanced age.

**binge drinking** The consumption of five or more standardized ethanol equivalents in one sitting. A standardized ethanol equivalent is 12 oz of beer, 4 oz of wine, or 1.5 oz of distilled spirits.

At-risk drinking tends to be more prevalent and recognized among older males because it is associated with injuries (Dharia & Slattum, 2011; Immonen, Valvanne, & Pitkala, 2011). Elderly

women may experience at-risk consumption of alcohol but tend to drink at home, whereas men tend to drink at public places and, therefore, have their alcohol consumption noticed.

### Cigarette Smoking

Cigarettes, chewing tobacco, and even pipe smoking are all considered risky health behaviors over the long term. Known carcinogens present in these products interact with a variety of other factors to influence cellular senescence, morbidity, and mortality. In smokers, an increased pro-oxidant status plus the effects of contaminants contained in tobacco (such as cadmium) require the diversion of available antioxidant mechanisms to interruption of the pathologic changes induced by tobacco products (Chen, Pu, & Lin, 2001). Even with adequate antioxidant capabilities, the number of oxidative events eventually surpasses response, and with continued tobacco use carcinogenic and inflammatory processes cause cellular alteration. Over time, an increase in a variety of disease states can be seen in tobacco users. Cancer, cardiovascular disease, stroke, and osteoporosis are significantly correlated with tobacco product usage; extent, duration, and type of usage should always be assessed in the older adult (Kato, Toniolo, Zeleniuch-Jacquotte, Shore, & Koenig, 2000). Other variables affect these tobacco effects in a **synergistic** manner; thus, poor diet quality, lack of exercise, body habitus, and heavy drinking elevate risk for morbidity. These variables often are seen clustered in an individual, creating a profile that is very strongly correlated with risk of disease. Those with less education, lower economic status, and high stress often exhibit this variable profile (Burke et al., 2001).

**synergistic** The outcome of two influential factors is greater than the sum of each alone (such as 1 + 1 = 3 times the effect).

### Oral Health

Oral health is important for consumption of foods and adequate nutrition. Persons with dentition problems or periodontal disease do not consume nutritionally dense foods such as fruits and vegetables in adequate amounts. Decreased salivation, ill-fitting dentures, and mouth sores or pain are just some of the factors involved in poor nutritional status resulting from decrements in oral intake. In addition to the lack of antioxidants, vitamins, minerals, and proteins are not adequately consumed; these include the relationship between oral health and systemic inflammatory response (Avcu et al., 2005). The oral cavity is home to many different species of bacteria.

### Pet Ownership

Evidence of the positive effects on health of pet ownership have been documented. Pet ownership can help lower blood pressure, decrease risk of cardiovascular disease, decrease depression and loneliness, and desensitize a person overall to antigens with a subsequent decrease in immune and inflammatory responses (Smith, 2012). Community-dwelling older adults may also realize a significant positive impact of pet ownership, especially dog ownership, on social interaction and physical activity, and indirectly pet owners may have better nutritional status (Gretebeck et al., 2012; McNicholas et al., 2005). Studies on cohorts of persons in specific geographic areas have found anecdotal support for these findings, but no statistically significant evidence (Parslow, Jorm, Christensen, Rodgers, & Jacomb, 2005). Three potential relationships exist to explain the benefits of pet ownership on health in older people:

- Pet ownership is associated with specific personality traits, age, and economic status, and these associated traits mediate the positive effects of pet ownership on human health.
- Pet ownership enhances social interactions and leads to decreased social isolation, thus providing an indirect mechanism for increased health.
- Pet ownership reduces stress, increases physical activity, and changes levels of hormones involved in the inflammatory process. These direct effects also may interact with the indirect effects previously outlined.

Pet ownership and its effects on overall health may require additional research to obviate findings statistically, but owning a pet should not be overlooked as a contributing factor to healthy aging.

### Reproductive Hormone Levels

Declines in circulating sex-related hormones are known to alter inflammatory mediators such as cytokines and acute phase proteins. Several inflammatory mediators, including tumor necrosis factor-alpha, interleukin-6, and interleukin-8, are modulated by these steroids. Aging-associated declines in sex hormone production may indirectly influence cellular senescence via increased oxidative stress. In addition, the protective effects of sex steroid hormones

have long been documented in terms of bone health for women, with postmenopausal women showing increased bone density losses. It has also been shown that the sex steroids in premenopausal women are protective for coronary artery disease, which can be seen in numerous surveys that have corrected for other variables known to influence risk (Krabbe, Pederson, & Brüünsgaard, 2004).

Hormone replacement therapy has become increasingly widespread among postmenopausal women, and it also is thought that gluteal-femoral adiposity is favored in these women and visceral adiposity is not. This may explain a mediating relationship in the hypothesis that estrogen and other female sex steroids reduce oxidative risk in older women.

Age-related inflammatory cytokine increases may have multiple etiologies and may interact with a number of other compounds through a variety of mechanisms. The upshot is that inflammation increases morbidity and mortality in older adults through partial mediation by declines in the sex steroid hormones (Brüünsgaard & Pedersen, 2003).

### Marital Status

Marital status is thought to have a moderating influence on healthy aging, especially in older men; it has been reported that unmarried or divorced men have a higher risk for a lack of health locus of control, indicating that they have a low likelihood of engaging in health-promoting behaviors (Lindstrőm & Rosvall, 2012). Some of the reasons may involve decreased social isolation; absent spousal aid in procuring food, cooking, and serving meals; or increased need for caregiving in general (Schone & Weinick, 1998). Nevertheless, married individuals report better health status and lower mortality rates than do unmarried individuals (Robards, Evandrou, Falkingham, & Vlachantoni, 2012; Zheng & Thomas, 2013). Widowhood is associated with increased morbidity and mortality in both older men and women and is associated with greater disability (Hokby, Reimers, & Laflamme, 2003; Iwashyna & Christakis, 2003). Nutritional status among older men is also associated with marital status: Unmarried or widowed men have poorer intakes. Living alone without caregiver support is associated with poorer dietary intake in both genders (Larrieu et al., 2004).

### Mental Status

Depression, declines in cognitive function, and neurodegenerative disease have profound effects on the nutritional status of the older adult (Morley, 2013; Salerno-Kennedy & Cashmanl, 2005). Dementia has been associated with alterations in feeding behavior with dementia patients becoming cachectic and losing weight. These changes affect morbidity, mortality, and cellular senescence, although the exact mechanisms are not fully understood (Robertson & Montagnini, 2004). Probably, a number of factors influence this effect, including decreased physical activity, increased stress hormone production, and alterations in neurotransmission leading to metabolic dysregulation, among others (Loucks, Berkamn, Gruenewald, & Seeman, 2005; Yaffe et al., 1999). In older persons loneliness, social isolation, and depression can result in poor dietary intake, frank anorexia, self-neglect, and debilitation (Abrams, Lachs, McAvay, Keohane, & Bruce, 2002; Ferry, Sidobre, Lambertin, & Barberger-Gateau, 2005; Morley, 2013). Interventions can slow the decline in nutritional status such as providing between-meal supplements and offering multiple small meals throughout the day. Tube feeding often is used inappropriately in dementia patients, and there is some evidence that tube feeding terminally demented patients may hasten their demise (Chernoff, 2006).

### Mental Activity

Mental activity is thought to delay the progression of age-related cognitive declines and improve mood. The beneficial effects of psychological well-being on morbidity and mortality have been documented in elderly adults, but the exact mechanisms are not yet fully understood (Depp & Jeste, 2006). The role of mental activity in delaying cellular senescence is thought to involve the effects of nonspecific immune and inflammatory factors, which decline with improved mood, increased physical activity, tighter social integration, and better nutritional and lifestyle characteristics (Fortes et al., 2003). Having multiple roles, motivation, "personal projects," and varied activities and having one's basic needs met have been shown to affect aging, disability, disease, and mortality (Adelman, 1994; Blazer & Sachs-Ericsson, 2005; Herzog, Regula, Markus, & Holmberg, 1998; Powell, Moss, & Winter, 2002).

### Socioeconomics

Socioeconomics affects nutritional status and overall health, especially in older adults (Donini et al., 2013). Lack of access to health care, less money

available to purchase food, and fewer resources to procure, prepare, and consume nutritious foods are all consequences of low socioeconomic status. Elderly people are often among the poorest poor and must cope with increased costs of medications on fixed incomes; with limited income available to pay for housing and other basic needs (U.S. Department of Health and Human Services, 2003), increased healthcare costs are often difficult to balance. Declines in nutrient-dense food intake among elderly people result in deficiencies in calcium, iron, zinc, vitamin $B_{12}$, and vitamin E. Foods high in these particular micronutrients also are more expensive and often are protein sources as well (Drewnowski & Schultz, 2001). Socioeconomic hardship is associated with poorer outcome in terms of nutritional status, morbidity, and mortality.

### Education

Education may be a surrogate for socioeconomic standing or may be related to general health and nutritional status. Increased education is associated with more successful aging, perhaps through more informed dietary choices and lifestyle characteristics, better access to health care, or increased mental activity. The present cohort of older adults is better educated and more knowledgeable about their healthcare options than any generation before (Chernoff, 1995). Less-educated minority persons have been shown to have increased risks for nutritional deficiency as well as poorer overall health. Increased educational attainment is also associated with increased social and psychological resources, which also may mediate the relationships (Ross & Mirowsky, 1999).

### Social Support

Social support and networks of friends, relatives, community resources, and religious organizations are associated with improved health and decreased disability, morbidity, and mortality among elderly persons (Chernoff, 2013b). This may be related to the provision of care by others, increased mental activity, healthier lifestyle, more physical activity, and less social isolation. Improved nutritional status with an extended network also is probable, with more resources available for the procurement and provision of nutritious foods. In addition, having a place within a social hierarchy may also provide benefit, just by virtue of increased resources available (Lavis, McLeod, Mustard, & Stoddard, 2003). Older adults realize benefits of increased social networks and support in terms of mental status, nutritional status, functional status, and overall successful aging (Kirby, Coleman, & Daley, 2004; Koukouli, Vlachonikolis, & Philalithis, 2002; Yeh & Liu, 2003).

## Care Management Issues

Older adults with multiple conditions often require continuous care. Whether acute or chronic, comorbidity in elderly people presents challenges in terms of healthcare costs and availability of facilities and staffing. The processes involved in cellular senescence along with overt disease often result in the need for hospitalization, institutionalization, or prolonged rehabilitation.

Malnutrition is correlated with institutionalization, hospitalization, and rehabilitative care. Poor nutritional status is a major risk factor for complications and delayed recovery. Unintentional weight loss, disease-related malnutrition, and depression-related anorexia rates are estimated to be up to 45% for older adults in the acute or subacute care setting (Brantervik et al., 2005). Identifying malnutrition in older adults who are seriously ill is challenging and multiple tools can be used. It is, however, difficult to distinguish among the disease processes, inflammatory processes, and body composition factors when evaluating projected interventions and outcomes (Sheehan et al., 2013). Sarcopenia may contribute to falls and functional decline with hospitalization and often results in further complications. This is caused in part by malnutrition, reduced physical activity, reduction in sex hormone levels, and impairment in growth and insulin-like growth hormones (Borst, 2004).

Hospitalized elderly adults have increased rates of **nosocomial** infections with malnutrition and decrements in immune function consistent with aging. Inflammation results in a hypermetabolic state, which contributes to an increase in nutritional demands while intakes remain marginal. An extended length of hospitalization worsens these effects, and the outcomes are generally poorer with longer duration of stay (Kyle, Genton, & Pichard, 2005). Generally, the type of institution also plays a role in the declines experienced by the ailing older adult. Larger institutions and/or institutions with more resources tend to offer more comprehensive

**nosocomial** Hospital-acquired infection, usually with an antibiotic-resistant strain of organism.

care and have improved or cutting-edge practices that decrease the level of the malnutrition and improve the outcome of elderly patients.

Skilled nursing facilities also differ in the amount of resources and staff provided to ill elderly residents. Recent advances in low-cost formulations that are high in protein, supplemented with vitamins and minerals, and easily digested have made it easier and more feasible to deal with nutritional support in this population; however, these advances do not address the fundamental problem of poor oral feeding and the quality-of-life issues regarding eating of pleasurable foods (Levinson et al., 2005). Holistic treatment of the issues regarding hospitalized or institutionalized frail older adults is essential, and clinical outcome in terms of morbidity and mortality should not be the only endpoints. Quality of life should be considered and comprises health and physical functioning and social and emotional functioning, both measured and perceived (Keller, 2004).

Hospitalization or institutionalization of the frail older person results in decreases in nutritional status just by virtue of changes in the eating environment. Institutional surroundings and a lack of attention to the appearance or preparation of foods affect intake and dietary choices (Gibbons & Henry, 2005). Several key macronutrients and micronutrients are affected, such as vitamin D, zinc, calcium, and protein (Meydani, 2001; Pepersack et al., 2001). In the case of vitamin D, less exposure to sunlight with no time spent outdoors complicates vitamin D and calcium nutriture in the geriatric patient (Lyman, 2005). Smaller macronutrient- and micronutrient-enriched meals, properly prepared and nicely presented, may aid in improving nutritional status and preventing further debilitation among institutionalized elderly adults (Kannus, Uusi-Rasi, Palvanen, & Parkkari, 2005; Lorefält, Wissing, & Unosson, 2005).

Common problems in comorbid hospitalized or institutionalized elderly adults include infection resulting from aspiration or nosocomial acquisition of infection, diminished cognition (Vizuete et al., 2010), and pressure ulcers (Kikawada, Iwamoto, & Takasaki, 2005). All are inherently tied to nutrition and to increases in inflammatory response and cellular senescence. It is extremely difficult to treat these conditions, and they often result in further debilitation and ultimately in increased mortality (Loeb & High, 2005; Mathus-Vliegen, 2004). Several strategies have been used to augment treatment in the compromised older adult in these environments, including nutritional support with added select amino acids, such as glutamine or arginine; use of appetite enhancers such as megestrol; and single supplement augmentation of oral intake with zinc, chromium, copper, manganese, or antioxidant vitamins such as A and C (Rabinovitz et al., 2004; Reuben, Hirsch, Zhou, & Greendale, 2005; Stechmiller, Langkamp-Henken, & Childress, 2005).

These artificial feeding and hydration strategies have met with mixed results, and the use of nutrition support or supplementation must be weighed against quality-of-life issues, such as the ability to eat for pleasure at the end of life. Implementation of best practice standards for nutrition in the frail older adult must be considered, taking into account personal preferences, involvement of a multidisciplinary healthcare team to improve oral intake, and incorporation of social interaction at mealtime (Booth, Leadbetter, Francis, & Tolson, 2005). Continuous improvement in the approach used to provide nutrition to institutionalized or hospitalized geriatric patients is essential (Pepersack, 2005). It also is important to accept that mortality is inevitable, and provision of nutrition and fluids at the end of life may not be in the best interest of the patient (Chernoff, 2006).

Indicators of the need to provide additional support to geriatric patients can be evaluated using a number of tools, including laboratory indices, the Mini Nutritional Assessment, the CMS minimum data set, anthropometry, and assessment of unintentional weight loss or gain (Chernoff, 2013b; Ranhoff, Gjoen, & Mowé, 2005; Salva, Corman, Andrieu, Salas, & Vellas, 2004; Zulkowski & Coon, 2004). Regardless of physical state, the older adult should be granted autonomy in healthcare decisions; if the older adult is unable to make decisions as a result of mental status issues, a legally appointed guardian should be allowed to direct continuance of care. The wishes of the older adult should always be in the forefront when making decisions regarding medical procedures and feeding and hydration at the end of life (Stuck et al., 2005).

Artificial feeding and provision of fluids not by oral means are burdens in terms of healthcare costs, use of medical resources, and patient suffering in elderly people at the end of life. Artificial provision of food and fluids at this stage is thought to cause continued pain and discomfort. Complications resulting from nasogastric tubes,

total parenteral nutrition, or tube feeding include increases in infection, aspiration, and inflammation. Patients often have to be restrained so that they do not dislodge feeding tubes; restraint in and of itself increases distress even in patients with severely altered mental states (Chernoff, 2006; Slomka, 2003).

Dying from dehydration is a pain-free, natural, and peaceful process. Dry mouth is the only symptom associated with this course of action and is easily treated with lip balms and ice chips. Death through dehydration reduces nausea, diarrhea, and urinary output with concomitant urinary tract infection or bedsores from catheterization, wet bed sheets, and use of bedpans. Pulmonary secretions decrease, resulting in less discomfort from shortness of breath, choking, coughing, and congestion. Measures of physical pain show that dying patients have significantly less pain and in most cases mild euphoria with dehydration (Hoefler, 2000). It is unfortunate that societal norms are such that death is viewed as something that must be staved off at all costs. The medical community has done little to inform itself and the public of the burden of keeping a dying older adult alive through artificial measures, even in situations where individuals' own wishes are overturned.

## Pharmacology in Older Adults

Polypharmacy, the use of multiple prescription medications, is common among older persons because of multiple comorbid conditions. In addition to prescribed medications, older adults report more frequent use of over-the-counter preparations and herbal supplements. Nonvitamin, nonmineral supplement use complicates the interactions among over-the-counter preparations, prescribed medications, and nutrients derived from oral intake or vitamin and mineral preparations.

**Learning Point** It is estimated that community-dwelling elderly adults take three or more prescribed medications per day, with almost half taking additional over-the-counter and supplemental products. The number of medications taken by institutionalized elderly persons is estimated at six or more per day (Bales and Ritchie, 2009).

The use of herbals, botanicals, and amino acid supplements is significantly underreported by older adults and is thought to interact with a variety of other preparations, especially the vitamin K–antagonist anticoagulants, such as warfarin and dicoumarol (Archer, 2005). Compounds from herbal products such as garlic, ginkgo biloba, and Co-Q10 mediate the clotting cascade by the same mechanisms as the vitamin K antagonists. In addition, several botanicals have known interactions with the inflammatory cascade with potent lymphocytic activation.

Aging is associated with increases in chronic and acute disease, for which multiple medications are prescribed (Soini, Routasalo, & Lagstrom, 2005). Nutritional status often is impaired as a result of the use of a variety of medications and other preparations (Eriksson et al., 2005). *Polypharmacy* is defined as "the use of excessive drugs or compounds with drug-like effects" or "the prescription of multiple medications given at one time" (Couris, Gura, & Blumberg, 2013). Although it varies among institutions, the use of seven or more prescription medications usually requires the authorization by a geriatrician or geriatrics-trained pharmacist. More than 250 different medications alter food intake as a result of sensory side effects, and hundreds more have direct effects on the absorption, disposition, and utilization of nutrients.

Polypharmacy involves a complex set of problems in that foods alter drug disposition, drugs alter other drugs' disposition, and drugs alter nutrient disposition through a variety of mechanisms (Couris et al., 2013). The addition of cellular senescence, organ system deterioration, and alterations in catabolic potential for compounds, such as medications or herbal supplements, result in reductions in total homeostatic control in older persons (Bauer, 2001). Several classes of compounds are known for their interactions and nutritional effects:

- *Anticholinergic medications:* Impair sensory perception, leading to declines in intake and increased gastrointestinal distress
- *Glycosides:* Alter nutrient absorption, increase gastrointestinal distress and anorexia, and promote electrolyte disturbances
- *Diuretics:* Affect mineral excretion, hydration, and electrolyte balance
- *Beta-adrenergic antagonists:* Alter gastrointestinal function, cause gastrointestinal distress, and impair glycogen metabolism
- *Vasodilators:* Interact with B vitamins such as pyridoxine, cause gastrointestinal distress, and depress food intake
- *Antiarrhythmic agents:* Cause gastrointestinal distress, anorexia, dysgeusia, dysosmia, and dry mouth

- *Anticoagulants:* Cause gastrointestinal distress and interact with vitamins K and E and calcium
- *Lypolipidemics:* May alter the absorption of fat-soluble vitamins and cause gastrointestinal distress and severe constipation
- *Central nervous system or psychotherapeutic agents:* Interact with a wide range of amino acids, vitamins, and minerals in addition to altering sensory perception, depressing or increasing appetite (depends on the drug), causing gastrointestinal disturbance, and altering the disposition of many metabolic intermediates (Bauer, 2001; Mallarkey, 1999)

Older adults use more than 30% of all medications prescribed, and one in three older persons has been prescribed an unnecessary, ineffective, or potentially dangerous medication. Among Medicare patients, more than 1,500 adverse events in a single year have been documented. Most adverse events are the result of interactions, and the estimated incidence of interaction increases from 6% in persons taking two or more drugs to 50% in persons taking five medications per day (Wootan & Galavis, 2005). Polypharmacy has also been linked to falls, worsening cognitive status, and poorer overall outcomes in both free-living and institutionalized older adults (Perr et al., 2005; Ziere et al., 2006). Because of the plethora of medications, each with its own set of drug–drug and drug–nutrient interactions, it is impossible to cover the entire spectrum of available information. Several guides are available for interactions between nutrients and drugs, herbals and drugs, and drugs and other drugs.

## Pharmacotherapeutic Intervention in Obesity

In light of the changing demographics in the United States, the issues of weight and weight control in older persons will become increasingly important, and little is known about the treatment efficacy of anorexigenic drugs in this population or the likelihood of interactions between these drugs and other medications, nutrients, and herbal products. More is known about appetite stimulation in this population (Lee et al., 2005). Intuitively, this population would be better served by using diet, exercise, and nutrient density as a first response to weight change (Keller, Hadley, Hadley, Wong, & Vanderkooy, 2005).

Obesity in older adults presents unique challenges, but the treatment choices warrant great concern. Most pharmacotherapeutic interventions for obesity in the older adult are contraindicated because elderly adults have multiple conditions and prescription drug use. It remains to be seen whether drug treatment can play a significant role in managing obesity among older persons in the United States.

## Summary

The concept of "successful" or "healthy" aging involves many factors. Some of these include complex interactions among nutritional status, genetics, lifestyle, and environmental considerations. Nutritional adequacy with caloric restriction over the life span is thought to be critical to the expression of genetic tendencies toward apoptosis or cellular senescence. Mediation of oxidative stresses and apoptotic events is thought to include minimizing exposure to environmental contaminants as well as alcohol, drug, and tobacco use. Other moderating factors that may directly or indirectly influence senescence include mental status, mental activity, pet ownership, marital status, economics, and education, among others. Health conditions and acute or chronic diseases requiring medication affect the aging process. Body habitus and obesity, with their concomitant influences on disease and cellular senescence, are inherently tied to overall morbidity and mortality. In summary, healthy aging involves multiple factors, each with its own set of complex interactions. Older adults must be evaluated holistically, accounting for the multitude of factors unique to the individual that influence overall quality of life and longevity.

### Case Study

**Physical Activity in Baby Boomers**

Christina Taddei, MSF, RD, LDN

Jane Smith is a 59-year-old female who recently had a routine physical at her primary care doctor's office. Her lab results revealed a higher than normal fasting glucose of 120 mg/dL, placing her in the pre-diabetic category and at risk for type 2 diabetes. Her blood pressure was also elevated at 130/85 mm Hg. Mrs. Smith has a family history of heart disease: Her father passed away from a heart attack at the age of 70 years and her mother is currently taking medication to treat hypertension.

Mrs. Smith is married and had two adult children. She has been working for the past 25 years for an insurance company. During her discussion with the doctor, she admits to living a very sedentary lifestyle and walking less than 10 minutes per day. She has a desk job where she works 8-hour days Monday through Friday. She drives back and forth to work and does not engage in any additional physical activity throughout the day. Mrs. Smith is 5 foot 3 inches tall and weighs 170 lb. Over the past 9 years, she has noticed a slow weight gain of about 20 lb. Mrs. Smith recalls weighing about 150 lb when she was 50 years old. When asked about her previous weight history, she recalls weighing around 140 lb at age 30, after having her two children. During her 20s and 30s, she was more physically active and would go for daily walks with one of her friends. On average, she would walk for 30 to 45 minutes on most days of the week. In addition, she admits to being more active when she had two young children at home. As she became older and her home life and work life became busier, she stopped walking and settled into a more sedentary lifestyle for the past 20 years.

The doctor talked to Mrs. Smith about her eating habits and lack of physical activity. Mrs. Smith is afraid of developing type 2 diabetes or a heart condition and is concerned about the extra weight she has gained over the past few years. She wants to lose 10 lb and does not want to continue her gradual yearly weight gain. Because her children are older and her job is low stress, she has time again to devote to physical activity and wants to consider how to become more physically active and find activities that she enjoys.

**Questions**

1. What should the doctor recommend to Mrs. Smith to increase her physical activity? What types of exercises should she include?
2. What would a once-a-week workout plan look like for Mrs. Smith?

## Issues for Debate

1. Discuss examples of health factors that have affected older people you have known.
2. How could some of these health factors have been improved?
3. What will be the impact of health factors on the aging baby boomer population?

## References


Abrams, R. C., Lachs, M., McAvay, G., Keohane, D. J., & Bruce M. L. (2002). Predictors of self neglect in community dwelling elders. *American Journal of Psychiatry*, *159*, 1724–1730.

Adelman, P. K. (1994). Multiple roles and psychological well being in a national sample of older adults. *Journal of Gerontology*, *49*, 277S–285S.

Administration on Aging. (2011). *A profile of older Americans: 2010*. Washington, DC: U.S. Department of Health and Human Services.

Ahluwalia, N., Sun, J., Krause, D., Mastro, A., & Handte, G. (2004). Immune function is impaired in iron-deficient, homebound, older women. *American Journal of Clinical Nutrition*, *79*, 516–521.

Anderson, J. J., Suchindran, C. M., Kritchevsky, S. B., & Barrett-Connor, E. (2004). Macronutrient intakes of elderly in the Lipid Research Clinics Program Prevalence Study. *Journal of Nutrition, Health, and Aging*, *8*, 395–399.

Andree, K. B., Kim, J., & Kirschke, C. P. (2004). Investigation of lymphocyte gene expression for use as biomarkers for zinc status in humans. *Journal of Nutrition*, *134*, 1716–1723.

Archer, S. L. (2005). Nonvitamin and nonmineral supplement use among elderly people. *Journal of the American Dietetic Association*, *105*, 63–64.

Arterburn, D. E., Crane, P. K., & Sullivan, S. D. (2004). The coming epidemic of obesity in elderly Americans. *Journal of the American Geriatric Society*, *52*, 1907–1912.

Avcu, N., Ozbek, M., Kurtoglu, D., Kansu, O., & Kansu, H. (2005). Oral findings and health status among hospitalized patients with physical disabilities, aged 60 or above. *Archives of Gerontology and Geriatrics*, *41*, 69–79.

Bales, C., & Ritchie, C. (eds.). (2009). *Handbook of clinical nutrition and aging* (2nd ed.). Totowa, NJ: Humana Press.

Bamia, C. (2005). Dietary patterns among older Europeans: The EPIC-Elderly study. *British Journal of Nutrition*, *94*, 100–113.

Bauer, L. A. (2001). *Applied clinical pharmacokinetics*. New York, NY: McGraw-Hill.

Beck, A. M., Ovesen, L., & Schroll, M. (2001). A six month prospective follow-up of 65 and older patients from general practice classified according to nutritional risk by the MNA. *European Journal of Clinical Nutrition*, *55*, 1028–1033.

Belbraouet, S., Biaudet, H., Tébi, A., Chau, N., Gray-Donald, K., & Debry, G. (2007). Serum zinc and copper status in hospitalized vs healthy elderly subjects. *Journal of the American College of Nutrition*, *26*(6), 650–654.

Bergman, M., Salman, H., Pinchasi, R., Straussberg, R., Djaldetti, M., & Bessler, H. (2005). Phagocytic capacity and apoptosis of peripheral blood cells from patients with iron deficiency anemia. *Biomedicine and Pharmacotherapy*, *59*(6), 307–311.

Bhutto, A., & Morley, J. E. (2008). The clinical significance of gastrointestinal changes with aging. *Current Opinions in Clinical Nutrition and Metabolic Care*, *11*,651–660.

Bischoff-Ferrari, H. A., Dietrich, T., Orav, E. J., Hu, F. B., Zhang, Y., Karlson, E.W., & Dawson-Hughes, B. (2004). Higher 25-hydroxyvitamin D concentrations are associated with better lower-extremity function in both active and inactive persons aged > 60 y. *American Journal of Clinical Nutrition*, *80*, 752–758.

Blazer, D. G., & Sachs-Ericsson, N. H. (2005). Perception of unmet needs as a predictor of mortality among community dwelling older adults. *American Journal of Public Health*, *95*, 299–304.

Booth, J., Leadbetter, A., Francis, M., & Tolson, D. (2005). Implementing a best practice statement in nutrition for frail older people: Part 2. *Nursing Older People*, *17*, 22–24.

Borst, S. E. (2004). Interventions for sarcopenia and muscle weakness in older people. *Age and Ageing*, *33*, 548–555.

Bottiglieri, T., & Diaz-Arrastia, R. (2005). Hyperhomocystinemia and cognitive function: More than just a casual link? *American Journal of Clinical Nutrition*, *82*, 493–494.

Boudville, A., & Bruce, D. G. (2005). Lack of meal intake compensation following nutritional supplements in hospitalised elderly women. *British Journal of Nutrition*, *93*, 879–884.

Boyle, M. A., & Holben, D. H. (2006). *Community nutrition in action: An entrepreneurial approach* (4th ed.). Belmont, CA: Wadsworth.

Brach, J. S., Simonsick, E. M., Kritchevsky, S., Yaffe, K, Newman, A. B., & Health, Aging and Body Composition Study Research Group. (2004). The association between physical function and life-style activity and exercise in the health, aging and body composition study. *Journal of the American Geriatrics Society*, *52*, 502–509.

Brantervik, A. M., Jacobsson, I. E., Grimby, A., Wallén T. C., & Bosaeus, I. G. (2005). Older hospitalized patients at risk of malnutrition: Correlation with quality of life, aid from the social welfare system and length of stay? *Age Aging*, *34*, 444–449.

Bross, M. H., Soch, K., & Smith-Knuppel, T. (2010). Anemia in older persons. *American Family Physician*, *82*(5), 480–487.

Brüünsgaard, H., & Pedersen, B. K. (2003). Age-related inflammatory cytokines and disease. *Immunology and Allergy Clinics of North America*, *23*, 15–39.

Burke, G. L., Arnold, A. M., Bild, D. E., Cushman, M., Fried, L. P., Newman, A., Nunn, C., Robbins, J., & CHS Collaborative Research Group. (2001). Factors associated with healthy aging: The cardiovascular health study. *Journal of the American Geriatrics Society*, *49*, 254–262.

Cabrera, M. A. S., Andrade, S. M., & Dip, R. M. (2012). Lipids and all-cause mortality among older adults: A 12-year follow-up study. *Scientific World Journal*. doi:10.1100/2012/930139

Cacciapuoti, F. (2012). Lowering homocysteine levels with folic acid and B-vitamins do not reduce early artherosclerosis, but could interfere with cognitive decline and Alzheimer's disease. *Journal of Thrombosis and Thrombolysis*. doi:10.1007/s11239-012-0856-x

Cesari, M., Vellas, B., & Gambassi, G. (2012). The stress of aging. *Experimental Gerontology*. http://dx.doi.org/10.1016/j.exger.2012.10.004

Chen, Y. C., Pu, Y. S., & Lin, R. S. (2001). Blood and urine levels of cadmium in relation to demographic and life style in middle age and elderly men. *Bulletin on Environment Contamination Toxicology*, *66*, 287–294.

Chen, Z., Bassford, T., Green, S. B., Cauley, J. A., Jackson, R. D., LaCroix, A. Z., … Margolis, K. L. (2005). Postmenopausal hormone therapy and body composition—a substudy of the Estrogen Plus Progestin Trial of the Women's Health Initiative. *American Journal of Clinical Nutrition*, *82*, 651–656.

Chernoff, R. (1995). Baby boomers come of age: Nutrition in the 21st century. *Journal of the American Dietetic Association*, *95*(6), 650–654.

Chernoff, R. (2005). Micronutrient requirements in older women. *American Journal of Clinical Nutrition*, *81*, 1240S–1245S.

Chernoff, R. (2006). Tube feeding patients with dementia. *Nutrition in Clinical Practice*, *21*, 142–146.

Chernoff, R. (2013a). Demographics of aging. In R. Chernoff (Ed.), *Geriatric nutrition: Health professional's handbook* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Chernoff, R. (2013b). Nutritional screen monitoring tools. In C. D. Bernadier, J. Dwyer, & D. Heber (Eds.), *Handbook of nutrition and food* (2nd ed.). Boca Raton, FL: CRC Press.

Chmurzynska, A., Malinowska, A. M., Twardowska-Rajewska, J., & Gawecki, J. (2012). Elderly women: Homocysteine reduction by short-term folic acid supplementation resulting in increased glucose concentrations and affecting lipid metabolism (C677T MTHFR polymorphism). *Nutrition*, *29*(6), 841–844. doi:http://dx.doi.org/10.1016/j.nutr.2012.09.015

Coker, R. H., & Wolfe, R. R. (2011). Bedrest and sarcopenia. *Current Opinions in Clinical Nutrition and Metabolic Care*, *15*, 7–11.

Collins, C. E., Kershaw, J., & Brockington, S. (2005). Effect of nutritional supplements on wound healing in home-nursed elderly: A randomized trial. *Nutrition*, *21*, 147–155.

Couris, R. R., Gura, K. M., & Blumberg, J. (2013). Pharmacology, nutrition, and the elderly: Interactions and implications. In R. Chernoff (Ed.), *Geriatric nutrition: Health professional's handbook* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Cramm, J. M., & Nieboer, A. P. (2012). Relationships between frailty, neighborhood security, social cohesion and sense of belonging among community-dwelling older people. *Geriatrics and Gerontology International*, *13*(3), 759–763. doi:10:1111/j.1447-0594.2012.00967.x 1-5

Dawson-Hughes, B. (2004). Racial/ethnic considerations in making recommendations for vitamin D for adult and elderly men and women. *American Journal of Clinical Nutrition, 80*, 1763S–1766S.

De Groot, L. C., Verheijden, M. W., de Henauw, S., Schroll, M., & van Staveren, W. A. (2004). Life-style, nutritional status, health, and mortality in elderly people across Europe: Our view of the longitudinal results of the SENECA study. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *59*, 1277–1284.

Demingné, C., Sabboh, H., Rémésy, C., & Meneton, P. (2004). Protective effects of high dietary potassium: Nutritional and metabolic aspects. *Journal of Nutrition*, *134*, 2903–2906.

den Ouden, M. E., Schuurmans, M. J., Mueller-Schotte, S., & van der Schouw, Y. T. (2013). Identification of high-risk individuals for the development of disability in activities of daily living: A ten-year follow-up study. *Experimental Gerontology*, *48*(4), 437–443.

Deplas, A., Debiais, F., Alcalay, M., Bontoux, D., & Thomas, P. (2004). Bone density, parathyroid hormone, calcium and vitamin D nutritional status of institutionalized elderly subjects. *Journal of Nutrition, Health & Aging, 8*, 400–404.

Depp, C. A., & Jeste, D. V. (2006). Definitions and predictors of successful aging: A comprehensive review of larger quantitative studies. *American Journal of Geriatric Psychiatry*, *14*, 6–20.

Dharia, S. P., & Slattum, P. W. (2011). Alcohol, medications and the older adult. *Consultant Pharmacist*, *26*(11), 837–844.

Di Bartolomeo, F., Startek, J. B., & Van den Ende, W. (2012). Prebiotics to fight disease: Reality or fiction? *Phytotherapy Research*,*27*(10), 1457–73. doi:10.1002/ptr.4901

Donini, L. M., De Bernardini, L., De Felice, M. R., Savina, C., Coletti, C., & Cannella, C. (2004). Effect of nutritional status on clinical outcome in a population of geriatric rehabilitation patients. *Aging Clinical and Experimental Research*, *16*, 132–138.

Donini, L. M., Scardella, P., Piombo, L., Neri, B., Asprino, R., Proietti, A. R., … Morrone, A. (2013). Malnutrition in elderly: Social and economic determinants. *Journal of Nutrition, Health, and Aging*, *17*(1), 9–15.

Drewnowski, A., & Schultz, J. M. (2001). Impact of aging on eating behaviors, food choices, nutrition and health status. *Journal of Nutrition, Health, and Aging*, *5*, 75–79.

Eggers, P. W., & Greenberg, L. G. (2000). Racial and ethnic differences in hospitalization rates among aged Medicare beneficiaries. *Health Care Financing Review*, *21*(4),91–105.

Eriksson, B. G., Dey, D. K., Hessler, R. M., et al. (2005). Relationship between MNA and SF-36 in a free-living elderly population aged 70 to 75. *Journal of Nutrition, Health, and Aging*, *9*, 212–220.

Faure, P., Ducros, V., Couzy, F., Favier, A., & Ferry, M. (2005). Rapidly exchangeable pool study of zinc in free-living or institutionalized elderly women. *Nutrition*, *21*, 831–837.

Fenech, M. (2010). Folate, DNA damage and the aging brain. *Mechanisms of Ageing and Development*, *131*(14), 236–241.

Ferry, M., Sidobre, B., Lambertin, A., & Barberger-Gateau, P. (2005). The SOLINUT study: Analysis of the interaction between nutrition and loneliness in persons aged over 70 years. *Journal of Nutrition, Health, and Aging*, *9*, 261–268.

Fortes, C., Farchi, S., Forestiere, F., Agabiti, N., Pacifici, R., Zuccaro, P., Perucci, C. A., & Ebrahim, S. (2003). Depressive symptoms lead to impaired cellular immune response. *Psychotherapy and Psychosomatics*, *72*, 253–260.

Gazzotti, C., Albert, A., Pepinster, A., & Petermans, J. (2000). Clinical usefulness of the mini-nutritional assessment (MNA) scale in geriatric patients. *Journal of Nutrition, Health, and Aging*, *4*, 176–181.

Gibbons, M. D., & Henry, C. J. (2005). Does eating environment have an effect on food intake in the elderly? *Journal of Nutrition, Health, and Aging*, *9*, 25–29.

Ginty, F. (2003). Dietary protein and bone health. *Proceedings of the Nutrition Society*, *62*, 867–876.

Gonzalez, S., Huerta, J. M., Alvarez-Uria, J., Fernandez, S., Patterson, A. M., & Lasheras, C. (2004). Serum selenium is associated with plasma homocysteine concentrations in elderly humans. *Journal of Nutrition*, *134*, 1736–1740.

Gori, A. M., Corsi, A. M., Fedi, S., Gazzini, A., Sofi, F., Bartali B., … Ferrucci, L. (2005). A proinflammatory state is associated with hyperhomocystinemia in the elderly. *American Journal of Clinical Nutrition*, *82*, 335–341.

Gretebeck, K. A., Radius, K., Black, D. R., Gretebeck, R. J., Ziemba, R., & Glickman, L. T. (2012). Dog ownership, functional ability, and walking in community dwelling older adults. *Journal of Physical Activity and Health*, *10*(5), 646–655.

Hamilton, B. P., & Hamilton, J. H. (1997). Hypertension in elderly persons. *Endocrine Practice*, *3*, 29–41.

He, W., Sengupta, M., Velkoff, V. A., & DeBarros, K. A. (2005). *65+ in the United States: 2005*. Current Population Reports, P23-209. Washington, DC: U.S. Department of Health and Human Services and U.S. Department of Commerce.

Herzog, A., Regula, F., Markus, H., & Holmberg, D. (1998). Activities and well being in older age: Effects of self concept and educational attainment. *Psychological Aging*, *13*, 179–185.

Hjerkinn, E. M., Seljeflot, I., Ellingsen, I., Berstad, P., Hjermann, I., Sandvik, L., & Arnesen, H. (2005). Influence of long-term intervention with dietary counseling, long chain n-3 fatty acid supplements, or both on circulating markers of endothelial activation in men with long-standing hyperlipidemia. *American Journal of Clinical Nutrition*, *81*, 583–589.

Hoefler, J. M. (2000). Making decisions about tube feeding for severely demented patients at the end of life: Clinical, legal and ethical considerations. *Death Studies*, *24*, 233–254.

Hokby, A., Reimers, A., & Laflamme, I. (2003). Hip fractures among older people: Do marital status and type of residence matter? *Public Health*, *117*, 196–201.

Immonen, S., Valvanne, J., & Pitkala, K. H. (2011). Prevalence of at-risk drinking among older adults and associated sociodemographic and health-related factors. *Journal of Nutrition Health and Aging*, *15*(9), 789–794.

Institute of Medicine. (2008). *Retooling for an aging America: Building the health care workforce*. Washington, DC: National Academies Press.

Iwashyna, T. J., & Christakis, N. A. (2003). Marriage, widowhood and health care use. *Social Science and Medicine*, *57*, 2137–2147.

Jaques, P. F., Moeller, S. M., & Hankinson, S. E. (2003). Weight status, abdominal adiposity, diabetes and early age related lens opacities. *American Journal of Clinical Nutrition*, *78*, 400–405.

Jenny, N. S. (2012). Inflammation in aging: Cause, effect or both? *Discovery Medicine*, *13*(73), 451–460.

Jensen, G. L. (2005). Obesity and functional decline: Epidemiology and geriatric consequences. *Clinics Geriatric Medicine*, *21*(14), 677–687.

Jensen, G. L., & Hsiao, P. Y. (2010). Obesity in older adults: Relationship to functional limitation. *Current Opinion in Clinical Nutrition and Metabolic Care*, *13*, 46–51.

Johnson, G. H., & Fritsche, K. (2012). Effect of dietary linoleic acid on markers of inflammation in healthy persons: A systematic review of randomized controlled trials. *Journal of the Academy of Nutrition and Dietetics*, *112*(7), 1029–1041.

Jungjohann, S. M., Luhrmann, P. M., Bender, R., Blettner, M., & Neuhäuser-Berthold, M. (2005). Eight-year trends in food, energy and macronutrient intake in a sample of elderly German subjects. *British Journal of Nutrition*, *93*, 361–378.

Kannus, P., Uusi-Rasi, K., Palvanen, M., & Parkkari, J. (2005). Non-pharmacological means to prevent fractures among older adults. *Annals of Medicine*, *37*, 303–310.

Kato, I., Toniolo, P., Zeleniuch-Jacquotte, A., Shore, R. E., Koenig, K. L., Akhmedkhanov, A., & Riboli, E. (2000). Diet, smoking and anthropometric indices and postmenopausal bone fractures: A prospective study. *International Journal of Epidemiology*, *29*, 85–92.

Keller, H. H. (2004). Nutrition and health-related quality of life in frail older adults. *Journal of Nutrition, Health and Aging*, *8*, 245–252.

Keller, H. H., Hadley, M., Hadley, T., Wong, S., & Vanderkooy, P. (2005). Food workshops, nutrition education, and older adults: A process evaluation. *Journal of Nutrition for the Elderly*, *24*, 5–23.

Keller, H. H., Ostbye, T., & Goy, R. (2004). Nutritional risk predicts quality of life in elderly community-living Canadians. *The Journals of Gerontology: Biological Sciences and Medical Sciences, 59*(1), 68–74.

Kennedy, R. L., Chokkalingham, K., & Srinivasan, R. (2004). Obesity in the elderly: Who should we be treating, and why, and how? *Current Opinion in Clinical Nutrition and Metabolic Care*, *7*, 3–9.

Kikawada, M., Iwamoto, T., & Takasaki, M. (2005). Aspiration and infection in the elderly: Epidemiology, diagnosis and management. *Drugs and Aging*, *22*, 115–130.

Kirby, S. E., Coleman, P. G., & Daley, D. (2004). Spirituality and well being in frail and non-frail older adults. *Journal of Gerontology*, *59*, 123–129.

Klaenhammer, T. R., Kleerebezem, M., Kopp, M. V., & Rescigno, M. (2012). The impact of probiotics and prebiotics on the immune system. *Nature Reviews Immunology*, *12*, 728–734.

Koukouli, S., Vlachonikolis, I. G., & Philalithis, A. (2002). Sociodemographic factors and self reported functional status—the significance of social support. *BMC Health Service Research*, *2*, 20.

Krabbe, K. S., Pedersen, M., & Brüünsgaard, H. (2004). Inflammatory mediators in the elderly. *Experimental Gerontology*, *39*, 687–699.

Kyle, U. G., Genton, L., & Pichard, C. (2005). Hospital length of stay and nutritional status. *Current Opinion in Clinical Nutrition and Metabolic Care*, *8*, 397–402.

Larrieu, S., Letenneur, L., Berr, C., Dartigues, J. F., Ritchie, K., Alperovitch, A., …Barberger-Gateau, P. (2004). Sociodemographic differences in dietary habits in a population based sample of elderly subjects: The 3C study. *Journal of Nutrition, Health, and Aging*, *8*, 497–502.

Lavis, J. N., McLeod, C. B., Mustard, C. A., & Stoddard, G. I. (2003). Is there a gradient in lifespan by position in the social hierarchy? *American Journal of Public Health*, *93*, 771–773.

Ledikwe, J. H., Smiciklas-Wright, H., Mitchell, D. C., Miller, C. K., & Jensen, G. L. (2004). Dietary patterns of rural older adults are associated with weight and nutritional status. *Journal of the American Geriatrics Society*, *52*,589–595.

Lee, J. S., Kritchevsky, S. B., Harris, T. B., Tylavsky, F., Rubin, S. M., & Newman, A. B. (2005). Short-term weight changes in community-dwelling older adults: The Health, Aging, and Body Composition Weight Change Substudy. *American Journal of Clinical Nutrition*, *82*, 644–650.

Lee, J. S., Kritchevsky, S. B., Tylavsky, F. A., Harris, T., Everhart, J., Simonsick, E. M., Rubin, S. M., & Newman AB; Health, Aging and Body Composition (Health ABC) Study. (2004). Weight-loss intention in the well-functioning, community-dwelling elderly: Associations with diet quality, physical activity, and weight change. *American Journal of Clinical Nutrition*, *80*, 466–474.

Levinson, Y., Dwolatzky, T., Epstein, A., Adler, B., & Epstein, L. (2005). Is it possible to increase weight and maintain the protein status of debilitated elderly residents of nursing homes? *Journals of Gerontology, Series A, Biological Science*, *60*, 878–881.

Lichtenstein, A. H., & Russell, R. M. (2005). Essential nutrients: Food or supplements? Where should the emphasis be? *Journal of the American Medical Association*, *294*, 351–358.

Lindstrőm, M., & Rosvall, M. (2012). Marital status, social capital and health locus of control: A population-based study. *Public Health*, *126*, 790–795.

Loeb, M., & High, K. (2005). The effect of malnutrition on risk and outcome of community-acquired pneumonia. *Respiratory Care Clinics of North America*, *11*, 99–108.

Lorefält, B., Wissing, U., & Unosson, M. (2005). Smaller but energy- and protein-enriched meals improve energy and nutrient intakes in elderly patients. *Journal of Nutrition, Health, and Aging*, *9*, 243–247.

Loucks, E. B., Berkamn, L. F., Gruenewald, T. L., & Seeman, T. E. (2005). Social integration is associated with fibrinogen concentration in elderly men. *Psychosomatic Medicine*, *67*, 353–358.

Lyman, D. (2005). Undiagnosed vitamin D deficiency in the hospitalized patient. *American Family Physician*, *71*, 299–304.

Maiese, K., Chong, Z. Z., Hou, J., & Shang, Y. C. (2009). The vitamin nicotinamide: Translating nutrition into clinical care. *Molecules*, *14*(9), 3446–3485.

Mallarkey, G. (1999). *Drug treatment considerations in the elderly*. Hong Kong, China: Adis International.

Mathus-Vliegen, E. M. H. (2004). Old age, malnutrition, and pressure sores: An ill-fated alliance. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *59*, M355–M360.

Maxmen, A. (2012, August 30). Calorie restriction falters in the long run: Genetics and healthy diets matter more for longevity. *Nature News and Comments*, *488*, 569.

McCormick, D. B. (2012). Vitamin/trace mineral supplements for the elderly. *Advances in Nutrition*, *3*, 822–824.

McNicholas, J., Gilbey, A., Rennie, A., Ahmedzai, S., Dono, J. A., & Ormerod, E. (2005). Pet ownership and human health: A brief review of evidence and issues. *British Medical Journal*, *331*, 1252–1254.

Meydani, M. (2001). Nutrition interventions in aging and age-associated disease. *Annals of the New York Academy of Sciences*, *928*, 226–235.

Meydani, S. N., & Wu, D. (2007). Age-associated inflammatory changes: Role of nutritional intervention. *Nutrition Reviews*, *65*(12 Pt. 2), S213–S216.

Millward, D. J. (2004). Macronutrient intakes as determinants of dietary protein and amino acid adequacy. *American Society for Nutritional Sciences*, *134*, 1588S–1596S.

Milne, A. C., Potter, J., & Avenell, A. (2005). Protein and energy supplementation in elderly people at risk from malnutrition. *Cochrane Database System Review*, (2), CD003288.

Mitchell, C. O. (2013). Nutrition assessment of the elderly. In R. Chernoff R (Ed.), *Geriatric nutrition: Health professional's handbook* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Moorhouse, P., & Rockwood, K. (2012). Frailty and its quantitative evaluation. *Journal of the Royal College of Physicians* (Edinburgh), *42*, 333–340.

Morley, J. E. (2013). Behavioral management in the person with dementia. *Journal Nutrition Health and Aging*, *17*(1), 35–38.

Morley, J. E., Glick, Z., & Rubenstein, L. Z. (1995). *Geriatric nutrition: A comprehensive review* (2nd ed.). New York, NY: Raven Press.

Moschonis, G., Kannellakis, S., Papaioannou, N., Schaafsma, A., & Manios, Y. (2011). Possible site-specific effect of an intervention combining nutrition and lifestyle counseling with consumption of fortified dairy products on bone mass: The Postmenopausal Health Study II. *Journal of Bone Mineral Metabolism*, *29*,501–506.

Murumalla, R. K., Gunasekaran, M. K., Padhan, J. K., Bencharif, K., Gence, L., Festy, F., Césari, M., Roche, R., & Hoareau, L. (2012). Fatty acids do not pay the toll: Effect of SFA and PUFA on human adipose tissue and mature adipocytes inflammation. *Lipids in Health and Disease*, *11*, 175.

Nied, R. J., & Franklin, B. (2002). Promoting and prescribing exercise for the elderly. *American Family Physician*, *65*, 419–426.

Niefeld, M. R., & Kasper, J. D. (2005). Access to ambulatory medical and long-term care services among elderly Medicare and Medicaid beneficiaries: Organizational, financial and geographic barriers. *Medical Care Research Reviews*, *62*(3), 300–319.

Nieves, J. W. (2005). Osteoporosis: The role of micronutrients. *American Journal of Clinical Nutrition, 81*, 1232S–1239S.

Okuyama, H., Ichikawa, Y., Fujii, Y., Ito, M., & Yamada, K. (2005). Changes in dietary fatty acids and life style as major factors for rapidly increasing inflammatory diseases and elderly-onset diseases. *World Review of Nutrition and Dietetics*, *95*, 52–61.

Pae, M., Meydani, S. N., & Wu, D. (2012). The role of nutrition in enhancing immunity in aging. *Aging and Disease*, *3*(1), 91–129.

Parslow, R. A., Jorm, A. F., Christensen, H., Rodgers, B., & Jacomb, P. (2005). Pet ownership and health in older adults. *Gerontology*, *51*, 40–47.

Pawelec, G., Larbi, A., & Derhavanessian, E. (2010). Senescence of the human immune system. *Journal of Comparative Pathology*, *142*(Suppl. 1), S39–S44.

Payet, M., Esmail, M. H., Polichetti, E., Le Brun, G., Adjemout, L., Donnarel, G., Portugal, H., & Pieroni, G. (2004). Docosahexaenoic acid-enriched egg consumption induces accretion of arachidonic acid in erythrocytes of elderly patients. *British Journal of Nutrition*, *91*, 789–796.

Pepersack, T. (2005). Outcomes of continuous process improvement of nutritional care program among geriatric units. *Journals of Gerontology, Series A: Biological Sciences and Medical Sciences*, *60*, 787–792.

Pepersack, T., Rotsaert, P., Benoit, F., Willems, D., Fuss, M., Bourdoux, P., & Duchateau, J. (2001). Prevalence of zinc deficiency and its clinical relevance among hospitalized elderly. *Archives of Gerontology and Geriatrics*, *33*, 243–253.

Perr, M., Menon, A. M., Deshpande, Shinde, S. B., Jiang, R., Cooper, J. W., … Lorys, R. A. (2005). Adverse outcomes associated with inappropriate drug use in nursing homes. *Annals of Pharmacotherapy*, *39*, 405–411.

Powell, L. M., Moss, M. S., & Winter, L. H. (2002). Motivation in later life: Personal projects and well being. *Psychological Aging*, *17*, 539–547.

Prado, C. M. M., Wells, J. C. K., Smith, S. R., Stephan, B. C. M., & Siervo, M. (2012). Sarcopenic obesity: A critical appraisal of the current evidence. *Clinical Nutrition*, *31*, 583–601.

Quadri, P., Fragiacomo, C., Pezzati, R., Zanda, E., Forloni, G., Tettamanti, M., & Lucca, U. (2004). Homocysteine, folate, and vitamin B-12 in mild cognitive impairment, Alzheimer's disease, and vascular dementia. *American Journal of Clinical Nutrition*, *80*, 114–122.

Rabinovitz, H., Friedensohn, A., Leibovitz, A., Gabay, G., Rocas, C., & Habot, B. (2004). Effect of chromium supplementation in elderly patients. *International Journal for Vitamin and Nutrition Research*, *74*, 178–182.

Ramos, M. I., Allen, L. H., Haan, M. N., Green, R., & Miller, J. W. (2004). Plasma folate concentrations are associated with depressive symptoms in elderly Latina women despite folic acid fortification. *American Journal of Clinical Nutrition*, *80*, 1024–1028.

Ranhoff, A. H., Gjoen, A. U., & Mowé, M. (2005). Screening for malnutrition in elderly acute medical patients: The usefulness of MNA-SF. *Journal of Nutrition, Health, and Aging*, *9*, 221–225.

Ravaglia, G. Forti, P., Maioli, F., Martelli, M., Servadei, L., Brunetti, N., … Licastro, F.. (2005). Homocysteine and folate as risk factors for dementia and Alzheimer disease. *American Journal of Clinical Nutrition*, *82*, 636–643.

Reuben, D. B., Hirsch, S. H., Zhou, K., & Greendale, G. A. (2005). The effects of megestrol acetate suspension for elderly patients with reduced appetite after hospitalization: A phase II randomized clinical trial. *Journal of the American Geriatrics Society*, *53*, 970–975.

Riccioni, G., Bucciarelli, T., Mancini, B., Di Ilio, C., Capra, V., & D'Orazio, N. (2007). The role of antioxidant vitamin supplementation in preventing cardiovascular disease. *Expert Opinions on Investigational Drugs*, *16*(1), 25–32.

Rimkus, A., Melinchok, M. D., McEvoy, K., & Yeager, A. K. (2005). *Thesaurus of aging terminology* (8th ed.). Washington, DC: AARP.

Robards, J., Evandrou, M., Falkingham, J., & Vlachantoni, A. (2012). Marital status, health and mortality. *Maturitas*, *73*(4), 295–299.

Robertson, R. G., & Montagnini, M. (2004). Geriatric failure to thrive. *American Family Physician*, *70*, 343–350.

Ross, C. E., & Mirowsky, J. (1999). Refining the association between education and health. The effects of quantity, credential and selectivity. *Demography*, *36*, 445–460.

Ryan-Harshman, M., & Aldoori, W. (2004). Bone health. New role for Vitamin K? *Canadian Family Physician*, *50*, 993–997.

Salerno-Kennedy, R., & Cashmanl, K. D. (2005). Relationship between dementia and nutrition-related factors and disorders: An overview. *International Journal for Vitamin and Nutrition Research*, *75*, 83–95.

Salva, A., Corman, B., Andrieu, S., Salas, J., Vellas, B., & International Association Of Gerontology/International Academy Of Nutrition And Aging Task Force. (2004).

Minimum data set for nutritional intervention studies in elderly people. *Journals of Gerontology, Series A: Biological Sciences and Medical Sciences*, *59*, M724–M729.

Santesso, N., Bianchi, M., Mente, A., Mustafa, R., Heels-Ansdell, D., & Schünemann, H. J. (2012). Effects of higher- versus lower-protein diets on health outcomes: A systematic review and meta-analysis. *European Journal of Clinical Nutrition*, *66*,780–788.

Savaiano, D. (2011). Lactose intolerance: An unnecessary risk for low bone density. Nestlé Nutrition workshop series. *Paediatric Programme*, *67*, 161–171.

Schone, B. S., & Weinick, R. M. (1998). Health related behaviors and the benefits of marriage for elderly persons. *Gerontologist*, *38*, 618–627.

Schulze, M. B., Hoffmann, K., Manson, J. E., Willett, W. C., Meigs, J. B., Weikert, C., … Hu, F. B. (2005). Dietary pattern, inflammation, and incidence of type 2 diabetes in women. *American Journal of Clinical Nutrition*, *82*, 675–684.

Seeman, T. E., & Crimmins, E. (2001). Social environment effects on health and aging: Integrating epidemiologic and demographic approaches and perspectives. *Annals N Y Academy Sciences*, *954*, 88–117.

Shah, K., Stufflebam, A., Hilton, T. N., Sinacore, D. R., Klein, S., & Villareal, D. T. (2009). Diet and exercise interventions reduce intrahepatic fat content and improve insulin sensitivity in obese older adults. *Obesity*, *17*(12), 2162–2168.

Sheehan, P. M., Peterson, S. J., Chen, Y., Liu, D., Lateef, O., & Braunschweig, C. A. (2013). Utilizing multiple methods to classify malnutrition among elderly patients admitted to the medical and surgical intensive care units (ICU). *Clinical Nutrition*, *32*, 1–6.

Singer, B. H., & Manton, K. G. (1998). The effects of health changes on projections of health service needs for the elderly population of the United States. *Proceedings of the National Academies of Sciences of the United States*, *95*, 15618–15622.

Singh, G. M., Danaei, G., Pelizzari, P. M., Lin, J. K., Cowan, M. J., Stevens, G. A., … Ezzati, M. (2012). The age associations of blood pressure, cholesterol, and glucose; analysis of health examination surveys from international populations. *Circulation*, *125*, 2204–2211.

Slomka, J. (2003). Withholding nutrition at the end of life: Clinical and ethical issues. *Cleveland Clinic Journal of Medicine*, *70*, 548–552.

Smith, B. (2012). The "pet effect"—health related aspects of companion. *Royal Australian College of General Practitioners*, *41*(6), 439–442.

Snijder, M. B., Visser, M., & Dekker, J. M. (2005). Low subcutaneous thigh fat is a risk factor for unfavorable glucose and lipid levels independent of high abdominal fat. The Health ABC study. *Diabetologia*, *48*, 301–308.

Soini, H., Routasalo, P., & Lagstrom, H. (2005). Nutritional status in cognitively intact older people receiving home care services—a pilot study. *Journal of Nutrition, Health, and Aging*, *9*, 249–253.

Stechmiller, J. K., Langkamp-Henken, B., & Childress, B. (2005). Arginine supplementation does not enhance serum nitric oxide levels in elderly nursing home residents with pressure ulcers. *Biology Research in Nursing*, *6*, 289–299.

Stuck, A., Amstad, H., Baumann-Hölzle, R., Fankhauser, A., Kesselring, A., Leuba, A., … Vallotton, M. (2005). Treatment and care of elderly persons who are in need of care: Medical-ethical guidelines and recommendations. *Journal of Nutrition, Health, and Aging*, *9*, 288–295.

Sullivan, D. H. (2001). What do serum proteins tell us about our elderly patients? *Journals of Gerontology: Series A, Biological Sciences and Medical Sciences*, *56A*(2), M71–M74.

Takeda, E., Yamamoto, H., Yamanaka-Okumura, H., & Taketani, Y. (2012). Dietary phosphorus in bone health and quality of life. *Nutrition Reviews*, *70*(6), 311–321.

Tucker, K. L., Qiao, N., Scott, T., Rosenberg, I., & Spiro, A., III. (2005). High homocysteine and low B vitamins predict cognitive decline in aging men: The Veterans Affairs normative aging study. *American Journal of Clinical Nutrition*, *82*, 627–635.

U.S. Bureau of the Census. (2005). 65+ in the United States. Retrieved from www.census.gov/prod/2006pubs/p23-209.pdf

U.S. Department of Health and Human Services. (2003). *Health, United States, 2003: Special excerpt: Trend tables on 65 and older population*. Hyattsville, MD: Centers for Disease Control and Prevention, National Center for Health Statistics.

Vincent, G. K., & Velkoff, V. A. (2010). *The next four decades: The older population in the United States: 2010 to 2050*. Current Population Reports P25-1138. Washington, DC: US Census Bureau.

Vizuete, A. A., Robles, F., Rodríguez-Rodríguez, E., López-Sobaler, A. M., & Ortega, R. M. (2010). Association between food and nutrient intakes and cognitive capacity in a group of institutionalized elderly people. *European Journal of Nutrition*, *49*, 293–300.

Wannamethee, S. G., Shaper, A. G., Morris, R. W., Whincup, P. H. (2005). Measures of adiposity in the identification of metabolic abnormalities in elderly men. *American Journal of Clinical Nutrition*, *81*, 1313–1321.

Weaver, C. M. (2010). Role of dairy beverages in the diet. *Physiology & Behavior*, *100*, 63–66.

Weinrebe, W., Guneysu, S., & Welz-Barth, A. (2002). Low muscle mass of the thigh is significantly correlated with delirium and worse functional outcome in older medical patients. *Journal of the American Geriatric Society*, *50*, 1310–1311.

Welch, G. W., & Somwers, M. R. (2000). The interrelationship between body topology and body composition varies with age among women. *Journal of Nutrition*, *130*, 2371–2377.

Wilmoth, J. R. (2000). Demography of longevity: Past, present, and future trends. *Experimental Gerontology*, *35*(9–10), 1111–1119.

Wolfe, R. R. (2012). The role of dietary protein in optimizing muscle mass, function and health outcomes in older individuals. *British Journal of Nutrition*, *108*(Suppl. 2), S88–S93.

Wolters, M., Hermann, S., & Hahn, A. (2005). Effect of multivitamin supplementation on the homocysteine and methylmalonic acid blood concentrations in women over the age of 60 years. *European Journal of Nutrition*, *44*, 183–192.

Wootan, J., & Galavis, J. (2005). Polypharmacy: Keeping the elderly safe. *RN*, *68*, 44–51.

Wouters-Wesseling, W., Vos, A.P., Van Hal, M., De Groot, L. C., Van Staveren, W. A., & Bindels, J. G. (2005). The effect of supplementation with an enriched drink on indices of immune function in frail elderly. *Journal of Nutrition, Health, and Aging*, *9*, 281–286.

Yaffe, K., Blackwell, T., Gore, R., Sands, L., Reus, V., & Browner, W. S. (1999). Depressive symptoms and cognitive decline in non-demented elderly women. *Archives of General Psychiatry*, *56*, 425–430.

Yeh, S. C., & Liu, Y. Y. (2003). Influence of social support on cognitive function in the elderly. *BMC Health Service Research*, *3*, 9.

Zheng, H., & Thomas, P. A. (2013). Marital status, self-rated health, and mortality: Overestimation of health or diminishing protection of marriage? *Journal of Health and Social Behavior*, *54*(1), 128–143.

Ziere, G., Dieleman, J. P., Hofman, A., Pols, H. A., van der Cammen, T. J., & Stricker, B. H. (2006). Polypharmacy and falls in the middle age and elderly population. *British Journal of Clinical Pharmacology*, *61*, 218–223.

Zulkowski, K., & Coon, P. J. (2004). Comparison of nutritional risk between urban and rural elderly. *Ostomy/Wound Management*, *50*, 46–58.

CHAPTER 13

# Special Topics in Age-Related Risks: Unique Nutrition Issues in the Older Adult

*Karen M. Funderburg, MS, RD, LD, Migy K. Mathews, MD, and Courtney Moskal, MS, RD*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. List the unique physiologic changes that occur with aging that affect nutrient intake and nutritional status.
2. List key psychosocial changes associated with aging that can affect the desire or ability to consume an adequate diet.
3. Discuss the impact that age-related changes have on nutritional status and quality of life.
4. Understand the consequences of age-related malnutrition and nutrient deficiencies on overall health status and quality of life.
5. Identify nutrition interventions that can improve nutritional status and enhance quality of life.

This chapter focuses on the unique and complex nutrition issues facing the older population. Malnutrition, key nutrient deficiencies, impaired appetite, diminished eating ability, osteoporosis, and other nutrition-related health problems are discussed. The roles of nutrition interventions that improve nutritional status and enhance quality of life are presented.

### CRITICAL Thinking

**How Are Appetite and Hunger Similar and Different?**

*Appetite* is defined as "any of the instinctive desires necessary to keep up organic life; especially the desire to eat. *Hunger* is defined as "a craving or urgent need for food or a specific nutrient; an uneasy sensation occasioned by the lack of food."

Compare loneliness to social isolation. Relate these conditions to health and nutritional status in the older adult. *Loneliness* is sadness from being alone or separated from a loved one. *Social isolation* is characterized by the lack of pleasant companionship. A state of being alone is thought of as being imposed by others and seen as negative.

## Age-Related Risks for Malnutrition

The demographic challenge of the growing population of older adults calls for solutions to reduce chronic diseases and compress morbidity. Diet presents itself as a key component of the solution. Age-related changes in physiology and metabolism affect all organ systems, with the response of the older adult often differing from that of a younger counterpart (Kinney, 2004). In addition, this age group faces socioeconomic and physiologic changes that can affect availability of food and desire to eat. Nutritional status surveys of the older population have shown a relatively low prevalence of frank nutrient deficiencies but a marked increase in risk of malnutrition and evidence of subclinical deficiencies with a direct impact on function (Blumberg, 1997). In this section we discuss key changes that increase the risk of malnutrition in the older individual; in the next section we suggest nutritional interventions and strategies to reduce the chances that an inadequate diet will be consumed.

### Impaired Appetite

The intake of food is a daily contributor to social, cultural, and psychological quality of life. Eating not only provides necessary nutrients but serves as an important part of traditions and special occasions such as holidays, birthdays, weddings, and anniversaries. Changes in the gastrointestinal tract, decreased taste and smell acuity, medication side effects, diet modifications, depression, or altered mental status are just a few conditions that can diminish the desire to eat. **Table 13.1** provides a comprehensive list of factors that may affect appetite. A waning appetite can lead to smaller meals or skipping meals altogether and poor food choices. A reduction in caloric intake and essential nutrients leads to an increased risk of illness and infection. An infection can lead to a higher metabolic rate, and increased nutritional needs, which affects weight and nutritional status. Ultimately, an impaired appetite may lead to significant risks to overall health and well-being (American Dietetic Association [ADA], 2005b). Early detection and effective strategies are needed to combat appetite loss and prevent serious health complications.

**TABLE 13.1** **Conditions that May Affect Appetite**

- Decreased or altered taste
- Decreased smell
- Decreased thirst acuity
- Hypochlorhydria
- Early satiety
- Dyspepsia
- Decreased caloric needs
- Lack of hunger
- Eating impairment
- Diet modifications
- Dining environment
- Limited food choices
- Lack of control over food choices
- Medication side effects
- Dementia
- Depression
- Loneliness
- Social isolation

### Appetite Assessment

**anorexia** Diminished appetite.

Declining food intake and **anorexia** are predictors for undernutrition in older adults in community and institutional settings (ADA, 2005b). Early detection is essential to prevent compromised nutritional status and decreased quality of life. There are nutrition screening tools designed for the older population to screen for a variety of nutrition risk factors. The tool Determine Your Nutritional Health Checklist screens for the number of meals eaten per day, diet modifications, prescription drug use, and social interaction at mealtime (Nutrition Screening Initiative, 2004). Another nutrition tool called the Mini Nutritional Assessment looks at declining food intake, psychological stress, neuropsychological problems, medication use, and number of full meals consumed per day (Nestlé Clinical Research, 1998). The main objective of both screening tools is to determine nutritional risk. Because a decline in appetite may lead to a serious health problem, it's important to have specific tools designed to assess one's food intake. When a problem is identified, early detection and treatment may prevent weight loss and improve health outcomes (Wilson et al., 2005).

A self-assessment tool to measure appetite was used in seven countries during the Survey in Europe on Nutrition and the Elderly, a Concerted Action (SENECA) study (Mathey, de Jong, de Groot, de Graaf, & Van Staveren, 2001). The SENECA study was a three-part study with the last phase occurring in 1999, when the protocol used the Appetite, Hunger, Sensory Perception (AHSP) questionnaire (de Jong, Mulder, de Graaf, & Van Staveren, 1999). (The questionnaire consists of 29 items that estimate energy intake, appetite, and hunger sensations as well as taste and smell perceptions in elderly adults. Before the European study, the tool had been validated as a reliable source of data on elderly self-assessment of appetite.) In the SENECA study, the researchers used the AHSP questionnaire in three population groups. They wanted to determine if the tool could distinguish differences in sensations of appetite, hunger, and sensory perception between healthy elderly people living independently, frail elderly people living independently, and nursing home residents (Mathey et al., 2001). As a subset of the SENECA study, data from the Dutch population were analyzed. Results of the Dutch study revealed that appetite is related to the health status of elderly adults. In healthy subjects, appetite was a good indicator of body weight. As for the frail subjects, disease state rather than appetite affected body weight (Mathey et al., 2001).

The Council for Nutritional Strategies in Long-Term Care (n.d.) developed another appetite assessment tool. The council was formed in 1998 and is an interdisciplinary panel composed of experts from academia and the medical community, including geriatricians, dietitians, pharmacists, and nurse practitioners. The council's charge is to examine issues related to diagnosis, prevention, and treatment of undernutrition in older adults and to identify evidence-based recommendations for the treatment of undernutrition in long-term care (Wilson et al., 2005). They developed the Council of Nutrition Appetite Questionnaire (CNAQ), which scores eight questions to determine a person's risk for anorexia. The questionnaire addresses appetite, hunger, early satiety, taste perception and changes, gastrointestinal tolerance of food intake, mood, and frequency of meals. The total points scored on the questionnaire identify a person's risk for impaired appetite.

Researchers at St. Louis University conducted a study to determine the reliability and validity of

the CNAQ. Study subjects were residents in nine long-term care facilities and community-dwelling elderly adults. The subjects were asked to complete the simple short CNAQ and the longer questionnaire used in the European study, the AHSP tool. Because the AHSP had been validated, the researchers used it to facilitate validation of the CNAQ. During data analysis, the reliability study indicated that questions 3, 5, 7, and 8 were reliability reducers. Therefore, only questions 1, 2, 4, and 6 were used to form the Simplified Nutritional Appetite Questionnaire (SNAQ). In this study the CNAQ and SNAQ were validated for use in older adults to identify persons at risk for significant weight loss. The application of the CNAQ and the SNAQ as clinical tools can promptly identify problems and facilitate early intervention. Because the researchers found SNAQ to have comparable reliability to the CNAQ, its four-question length may be preferred in a clinical setting (Wilson et al., 2005).

### Diet Modification

In December 2005, the ADA released an updated position paper that stated, "Food is an essential component of quality of life; an unacceptable or unpalatable diet can lead to poor food and fluid intake, resulting in weight loss and undernutrition and a spiral of negative health effects" (ADA, 2005a, p. 1955).

**Learning Point** Special diets that alter texture or consistency because of poor dentition or disease are often unpalatable. Restriction of certain nutrients, such as sodium or fat, can also create an unacceptable diet. The insult of a restrictive diet may lead to poor intake, depression, and malnutrition.

A previously prescribed dietary restriction may no longer be necessary based on the person's current health, age, and dietary intake. The need for a diet modification should be carefully evaluated. In the face of declining appetite, the decision to restrict choices may do more harm than good. Diet prescriptions should be as liberal as possible for optimum intake and quality of life. According to the ADA position paper, "Overall health goals may not warrant the use of a therapeutic diet because of the possible negative effect on quality of life. Often, a more liberalized nutrition intervention allowing a resident to participate in his or her diet-related decisions can provide for nutrient needs and allow alterations contingent on medical conditions while simultaneously increasing the desire to eat and enjoyment of food" (ADA, 2005a, p. 1956). Diet modifications along with other conditions that affect appetite may result in limited food enjoyment and compromised food intake, potentially leading to unintentional weight loss and malnutrition (ADA, 2003b).

### Physiologic Changes

Food intake decreases as physical activity and metabolic rate decline, even in healthy older adults. This decrease often is called anorexia of aging (ADA, 2005a). Physiologic changes can reduce hunger and lead to early satiety. Body composition changes result in decreases in muscle mass, bone density, total body water, and metabolic rate. Older adults are generally less active than they are in their younger years, causing a further reduction in energy requirements. These changes reduce caloric needs without reducing nutritional needs, making food selection more challenging than for the younger adult.

Changes in the gastrointestinal tract are common with aging and may include problems with **dentition**, oral health, swallowing, diarrhea, constipation, and decreased hydrochloric acid in the stomach. The decrease in stomach acid may diminish hunger and decrease vitamin $B_{12}$ absorption. Medications that reduce stomach acid may have the same effect. Other gastrointestinal changes may cause alteration in food selections as the patient attempts to compensate for losses in gastrointestinal function or chewing and swallowing abilities.

**dentition** The natural teeth, as considered collectively.

Sensory loss is common in the aging process (ADA, 2005a). Taste and smell are significant contributors to recognizing flavors and the enjoyment of food and beverages. Aging, chronic health problems, and medications can alter olfactory and taste perceptions (Morley, 2001). Smell sensation that declines with aging is called **presbyosmia**. According to the National Institutes of Health, Senior Health website (see http://nihseniorhealth.gov), problems with the sense of smell occur in approximately 30% of Americans between the ages of 70 and 80 years. The problem increases to more than 65% of people older than age 80 years (National Institute on Deafness and Other Communication Disorders, 2006). Smell plays a key role in food enjoyment. A smell disorder can decrease the appreciation of food flavors and

**presbyosmia** Loss of the sense of smell.

the desire to cook and consume a variety of foods. Taste, temperature, texture, and aroma combine to create the perceived flavor of food. Like smell, the loss of taste sensitivity can cause a loss of appetite.

The older adult is at greater risk for foodborne illness as a result of the physiologic changes of aging. Risk factors for foodborne illness are decreased taste and smell, diminished immune function, **hypochlorhydria**, and altered mental status (ADA, 2003a). Food safety education targeted for seniors is available from the Food Safety Inspection Service of the U.S. Department of Agriculture and other government agencies such as the U.S. Food and Drug Administration and the National Institutes of Health.

**hypochlorhydria** Presence of an abnormally small amount of hydrochloric acid in the stomach.

Additional physiologic changes come in the form of disabilities and diseases. Accumulating disease and disability may rob a patient of independence. The loss of the ability to shop, cook, and even eat may decrease the desire to consume food. For many people, disability leads to a more sedentary lifestyle often accompanied by social isolation (ADA, 2005b).

### Cognitive Changes

Impaired mental health can have an impact on food intake and appetite. Persons may forget to eat, may have no desire to eat, or may have difficulty swallowing because of stroke, Alzheimer's disease, or other diseases such as Parkinson's disease or cancer. Impaired cognition may decrease the ability to self-feed, alter appetite, impair movement, and affect memory. The loss of family members and friends can lead to loneliness, isolation, and depression. Persons who eat most of their meals alone are at increased risk for malnutrition. Depression, anxiety, and bereavement may result in a loss of interest in food and may trigger substance abuse.

One form of **dementia** is Alzheimer's disease. The Alzheimer's Association provides tips to improve intake on their website (see http://www.alz.org). Decreasing distraction at mealtime, providing easy-to-use dishes and utensils, serving finger foods, and limiting food choices to one or two items at a time are some helpful tips (Alzheimer's Association, 2006).

**dementia** The loss, usually progressive, of cognitive and intellectual function.

The Mini Mental State Examination (MMSE) is used to assess the cognitive impairment of the older adult. The test includes simple questions relative to orientation, registration, attention and calculation, recall, and language. It is an effective screening tool for older adults in all settings (Kurlowitz & Wallace, 1999).

### Psychosocial Changes

Nutritional risk is associated with economic hardship and loneliness. Objectives of the Elderly Nutrition Program include combating these two barriers to nutritional health. The Administration on Aging of the U.S. Department of Health and Human Services is responsible for providing funding for congregate and home-delivered meals through state-run elderly nutrition programs. The funds are authorized under Title III and Title IV of the Older Americans Act. Programs are provided through local Area Agency on Aging or Tribal Senior Services. Participants can build an informal support system and develop friendships and receive a well-balanced lunch meal, educational programs, and health screenings. Persons who qualify for homebound status can have a lunch meal delivered 5 days per week. The program participants are primarily at high nutritional risk. The success of the Elderly Nutrition Program is well documented (U.S. Department of Health and Human Services, Administration on Aging, 2009).

A study was done by Gollub and Weddle (2004) to determine whether the addition of breakfast as a second home-delivered meal could improve the well-being of at-risk older adults. Most study participants lived alone, were low income, and had trouble shopping or preparing food. A demonstration project, the Morning Meals on Wheels Program, delivered breakfast 5 days per week to subjects who also received home-delivered lunch meals for at least 6 months. A comparison group only received the five home-delivered lunch meals each week. Both groups received the same lunch items, which provided one-third of the Dietary Reference Intake. The breakfast meals also provided one-third of the Dietary Reference Intake. Several surveys were used to assess participants' perceived global quality of life, health, loneliness, food security, enjoyment of food, and depression. The study found that breakfast participants had greater energy and nutrient intakes, less food insecurity, and less depressive days than the other group. The addition of the breakfast meal reduced malnutrition risk and improved appetite, perceived health, and outlook on life (Gollub & Weddle, 2004).

### Medication Use

Increased disease leads to increased medication use. Many medications have side effects such as dry mouth, altered taste sensation, sedation, diarrhea, constipation, and decreased appetite. Medications that sedate patients may decrease waking active hours and affect mealtimes and intake.

Medications that stimulate appetite and produce weight gain continue to be explored. Current medications are megestrol, dronabinol, oxandrolone, testosterone, metoclopramide, and cyproheptadine. More research is needed to verify the benefits of these drug therapies.

## Interventions for Impaired Appetite

Treating impaired appetite requires an individual approach. The first step is to determine the root causes or factors contributing to a decreased desire to eat. Using tools that aid in assessing appetite and mental status can assist in discovering the most significant problems. Providing choices in food selection, decreasing meals eaten alone, providing assistance with meals, maintaining independence, and using flavor enhancers are first-line strategies. Exploring medication options to decrease negative side effects should also be a part of the action plan. **Table 13.2** provides a list of strategies to increase food and nutrient intake.

Multiple factors and conditions result in decreased food intake in the older adult. Often these go in accordance with impaired appetite and are just as complex. Impaired eating ability can have significant negative effects on nutritional status and quality of life. **Table 13.3** lists the common problems associated with decreased eating ability that include poor oral health and dentition, swallowing problems, and altered physical ability resulting from decreased motor skills, mental status, visual impairment, and range of motion.

**TABLE 13.2 Strategies to Increase Food and Nutrient Intake**

- Liberalized diet
- Freedom in food selection
- Eating with others
- Congregate meals or home-delivered meals
- Providing assistance
- Providing specialized utensils
- Finger foods
- Flavor enhancers
- Adding nutrients to food
- Adding nutrient-dense snacks
- Adding commercial supplements
- Pleasant eating environment with minimal distractions
- Providing praise and encouragement
- Appetite stimulants

**TABLE 13.3 Problems that Affect Eating Ability**

- Tooth loss
- Edentulousness
- Dentures
- Mouth pain
- Xerostomia
- Dysphagia
- Visual impairment
- Impaired motor skills
- Arthritis
- Altered mental status

### Oral Health Problems

Oral health problems can lead to pain, tooth loss, and alterations in the diet and have a significant impact on quality of life (Ritchie, 2002). National Health and Nutrition Examination Survey (NHANES) data from 1999 to 2002 confirm that oral health is an increasing problem with age and is compounded by poor income status and lower education level (National Center for Chronic Disease Prevention and Health Promotion, 2005). Information on tooth decay revealed that only 9% of adults aged 20 to 39 years had decay on the roots of their teeth compared with 32% in adults 60 years or older. Forty-one percent of low-income adults (100% federal poverty level) had untreated tooth decay as compared with 16% in the higher income group (200% federal poverty level). Edentulousness also was affected by income. The prevalence was 15% in adults below 100% of the federal poverty level compared with only 5% in the higher income group (200% federal poverty level).

Education level also was shown to have a significant impact on oral health. Forty-one percent of adults who had less than a high school education had tooth decay compared with 14% of adults who

had a higher than high school education. Fourteen percent of adults who had not completed high school were **edentulous** compared with 9% with a high school education and 4% with a post–high school education.

**edentulous** Toothless; having lost the natural teeth.

Other interesting oral health data from NHANES showed that smoking increases tooth decay. The data showed that 13% of current smokers were edentulous, compared with 8% of former smokers and 5% of people who had never smoked. The greatest disparities in the prevalence of root caries were seen for current smokers. Twice as many current smokers (28%) as nonsmokers (14%) had root caries (National Center for Chronic Disease Prevention and Health Promotion, 2005).

### Cultural Diversity

Non-Hispanic whites had a great prevalence of tooth decay (93%) compared with African Americans (85%) and Mexican Americans (84%). However, the percentage of untreated decay was the lowest in non-Hispanic whites at 18%, followed by 36% in Mexican Americans, and 41% in African Americans. Edentulousness was highest in African Americans at 10% and lowest in non-Hispanic whites at 6% (National Center for Chronic Disease Prevention and Health Promotion, 2005).

Persons who wear dentures may avoid certain foods because they are hard to chew. Dietary quality and nutrient intake can be compromised, particularly in persons who perceive that their dentures are ill fitting (Sahyoun & Krall, 2003). An analysis of dietary quality and selection was conducted by Sahyoun and Knall (2003) using data from 4,466 NHANES III participants. Dietary intake, dietary quality, and serum nutrient values were analyzed for denture wearers and compared with participants with natural teeth. A denture wearer is someone who wears at least a full denture on the upper or lower jaw. As part of NHANES III data collection, denture wearers completed a four-item questionnaire to assess their perception of denture use and fit. The results showed that denture wearers had significantly lower serum levels of vitamins C and E, beta-carotene, lycopene, and lutein zeaxanthin than did the dentate group. Participants who believed they had ill-fitting dentures consumed fewer fruits and vegetables and had less variety in their diet than the participants who perceived they had good-fitting dentures or participants who had at least 18 natural teeth.

Data from 181 rural residents of Pennsylvania enrolled in a managed-risk Medicare program were surveyed regarding mouth pain and chewing and swallowing problems. Participants were surveyed twice with a year between surveys. Twenty-two participants reported persistent oral health problems. Results of analysis revealed that these participants had significantly greater medical problems than the participants who reported no oral health problems. They were also more likely to have lower intakes of fiber and vitamins C, A, and $B_6$. There was no significant difference in carbohydrate, protein, and fat between the groups (Bailey, Ledikwe, Smiciklas-Wright, Mitchell, & Jensen, 2004).

Oral hygiene after each meal can reduce oral health problems. Brushing, flossing, and using antimicrobial mouthwashes can help maintain oral health and prevent tooth decay, periodontal disease, and halitosis. Residents in long-term care facilities have multiple barriers to daily oral care. They frequently have functional decline because of chronic diseases or disabilities that make routine brushing and flossing difficult or not possible (Ship, 2002). Medication side effects such as dry mouth (**xerostomia**) also can influence oral health. Residents may have ill-fitting dentures because of weight loss or bone loss. Every resident of long-term care should have access to routine dental examinations and treatment.

**xerostomia** A dryness of the mouth.

Decreases in tooth extraction because of dental caries or periodontal disease are addressed in *Healthy People 2010* in Focus Area 21, Oral Health (U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion, n.d.). Two objectives specifically target the older adult. Objective 21-4 is to reduce the proportion of adults aged 65 to 74 years who have had all their natural teeth extracted. The baseline data from 1997 showed that 26% of adults in this age group had full-mouth extractions. The 2010 objective calls to reduce the number to 20%. Objective 21-11 aims to increase the proportion of long-term care residents who use the oral healthcare systems each year. The target is 25% versus 19% from the 1997 baseline data. Federal regulations require long-term care facilities that receive Medicare and/or Medicaid funds to ensure that each resident attains and maintains the highest practicable physical, psychosocial, and mental well-being (National Center for Chronic Disease Prevention and Health Promotion, 2005).

Ironically, Medicare does not cover dental services, regardless of cause or complexity, unless the procedure itself requires inpatient hospitalization or

is necessary for another medical treatment. Coverage is not based on the value or the necessity of dental care but rather on the service required. Oral care that involves treatment, removal, or replacement of teeth or structures supporting teeth; removal of diseased teeth in an infected jaw; or preparing the mouth for dentures is not covered (Centers for Medicare and Medicaid Services, n.d.). If a person has a nondental medical condition, such as a tumor, that requires teeth to be extracted, Medicare does not cover the cost of dental appliances or dentures, even though the covered medical service resulted in the need for the teeth to be repaired or replaced. The exclusion from the Social Security Act can be found on the Centers for Medicare and Medicaid Services website (see http://www.cms.gov). The dental exclusion was included as part of the initial Medicare program. Congress did not limit the exclusion to routine dental services but included a blanket exclusion of dental services.

The expense of dental services and artificial teeth can be prohibitive for many people in their older years. Like other income barriers, a lack of dental care can profoundly affect a person's ability to eat and consequently that person's nutritional status. Prevention and treatment programs for older adults are needed and should be incorporated as an interdisciplinary team approach. Further research is needed to determine the consequences of poor oral health on overall health and nutritional status.

### Swallowing Problems

Swallowing problems can result from a variety of etiologies. Dementia, stroke, neurologic disease such as Parkinson's disease, muscle disease such as multiple sclerosis, head and neck surgery, and weakened muscles caused by aging are among the conditions that may require precautionary measures to prevent choking and **aspiration**. Impaired swallowing may lead to decreased food intake, malnutrition, dehydration, and decreased quality of life.

**aspiration** Inhalation into the airways of fluid or foreign body; when food or liquid actually enters the lungs.

The assessment and diagnosis of a swallowing problem are usually triggered by signs and symptoms in the person experiencing difficulty. These include coughing before, during, or after swallowing; frequent throat clearing; hoarse, breathy voice; drooling; pocketing of food; and attempting to swallow multiple times with each bite (Deering, Russell, & Womack, 2001). Silent aspiration can occur in which no warning signs are present.

If **dysphagia** is suspected, a swallowing evaluation by a swallowing therapist, such as a speech pathologist, should be obtained. A videofluoroscopic study or barium swallow can identify specific swallowing problems (Deering et al., 2001). The results of testing are used to determine what diet modifications are the most beneficial in protecting the person from aspiration.

**dysphagia** Difficulty in swallowing.

Modifying food and beverage consistency and positioning during swallowing are the most common interventions. Before the introduction of the National Dysphagia Diet (NDD) in 2002, there was no standardized language regarding diet consistency and thickened liquids. NDD provides a multilevel approach to diet consistency and liquid viscosity (National Institute of Dental and Craniofacial Research, 2005). Dysphagia pureed (NDD1), dysphagia mechanically altered (NDD2), and dysphagia advanced (NDD3) identify three levels of food textures that range from pudding-like to nearly normal texture solids. The four liquid viscosities are "thin," "nectar-like," "honey-like," and "spoon thick." It is vital that foods and liquids have the appropriate texture and consistency for the swallowing ability of the impaired person or aspiration can occur. Unfortunately, texture, consistency, and viscosity modifications make food less appealing and lead to decreased intake. Persons who require thickened liquids may not consume adequate liquids. Dysphagia increases the risk for dehydration and malnutrition.

### Dry Mouth

Saliva plays an important role in chewing and swallowing. It also protects teeth from decay and fights mouth infections (National Institute of Dental and Craniofacial Research, 2005). Although xerostomia is not a normal part of aging, it is often the result of certain medication side effects. Common medications used for urinary incontinence, allergies, high blood pressure, and depression can alter the function of the salivary glands. More than 400 prescription and over-the-counter medications can produce this side effect. Dry mouth also can be the result of a medical condition such as diabetes, Parkinson's disease, **Sjögren syndrome**, or head and neck radiation therapy or chemotherapy (National Institute of Dental and Craniofacial Research, 2005).

**Sjögren syndrome** Dryness of mucous membranes.

The first step in treating dry mouth is to identify the cause. Once the cause is known, a course of

treatment can be implemented. Eating moist foods and drinking water or sugarless beverages with meals are front-line solutions. Sucking on sugarless candy can stimulate the salivary glands, and keeping a water bottle close by can help moisten the mouth between meals (National Institute of Dental and Craniofacial Research, 2005).

## Malnutrition and Nutrient Deficits

In the United States it is estimated that 40% of nursing home residents and 50% of hospitalized elderly patients are malnourished (Chen, Schilling, & Lyder, 2001). In the literature there are two clinical approaches to define malnutrition in the older adult. The first definition characterizes malnutrition as any insufficient dietary intake among essential nutrients. The second approach refers to malnutrition as protein-energy undernutrition. Protein-energy undernutrition is the progressive loss of both lean body muscle mass (**sarcopenia**) and adipose tissue resulting from insufficient consumption of protein and energy, although one or the other may play the dominant role in elderly adults (Chen et al., 2001).

**sarcopenia** Progressive reduction in muscle mass with aging.

From the literature, three measurement systems have been used in identifying malnutrition in the older population, including dietary intake, biochemical indices, and **anthropometrics**. Most nutritional assessment instruments also use all three aspects of measurement plus some clinical assessment such as anorexia or comorbid conditions. The Mini Nutritional Assessment is an example of this mixing of measurement systems. It should be noted that, to date, no single measurement has emerged as optimal in defining malnutrition in the older person (Chen et al., 2001).

**anthropometrics** Measurements of the human body.

**Learning Point** Malnutrition in the older individual affects muscle mass even more than in younger individuals with the same degree of weight loss; also, correcting malnutrition in the older person is more difficult than in the younger person with a similar degree of weight loss. The importance of prevention should therefore be stressed (Kinney, 2004).

### Weight Loss

The older adult must be evaluated periodically for unintended weight loss. Weight and height should be obtained at each visit using the same scale and without shoes. Body mass index (BMI) is a ratio of body weight in kilograms divided by the height in meters squared. BMI can guide the healthcare provider in determining the overall weight status of the patient. A body mass index of less than 18.5 is considered underweight, 18.5 to 24.9 is normal, 25 to 29.9 is overweight, and higher than 30.0 is considered obese (Amin, Kuhle, & Fitzpatrick, 2003).

Weight loss exceeding 5% in 1 month or 10% in 6 months deserves a complete evaluation, including a detailed physical examination, review of age-indicated preventive measures, pertinent screening tools, psychosocial assessment, review of medications, medical history, and laboratory and radiologic testing when indicated. Risk factors for poor nutritional status and subsequent weight loss are listed in **Table 13.4**.

### Energy and Caloric Intake

Numerous studies have shown energy intake declines with age, making a nutritionally adequate diet more difficult to achieve. A reduction in **basal metabolic rate** is partly responsible for this decline in energy and caloric intake, but a reduction in lean body mass and decreased physical activity appear to be the major causes (Wahlqvist & Savige, 2000).

**basal metabolic rate** The minimal amount of energy required to sustain life in the waking state.

### Protein-Energy Malnutrition

Protein plays an important role in the maintenance of the elderly person's health. Illness or inadequate intake may result in protein-energy malnutrition, a condition more common among elderly patients, especially those in institutionalized care (Wahlqvist &

**TABLE 13.4 Risk Factors for Weight Loss**

- Alcohol or substance abuse
- Cognitive dysfunction
- Depression
- Functional limitations
- Inadequate financial resources
- Limited education
- Limited mobility
- Transportation issues
- Chronic medical illness
- Poor dentition
- Restricted diet
- Poor eating habits
- Social isolation

**decubitus ulcer** Focal ischemic necrosis of skin and underlying tissue at sites of constant pressure or recurring friction in patients immobilized by illness or disability.

Savige, 2000). Chronic deficiency of protein in the elderly person's diet may result in poor wound healing, **decubitus ulcer** development, depressed immune function, osteoporosis, and loss of muscle strength (Chernoff, 1996). Protein is rich in other essential nutrients as well, thus its inclusion in the elderly individual's diet should be encouraged.

### Vitamin D

Vitamin D deficiency has special implications for elderly adults, especially those who are institutionalized or homebound. Although dietary consumption of vitamin D through fortified dairy products, sardines, and egg yolks can provide part of the daily allowance, natural sunlight is another important source. Aging skin, deteriorating renal function, and physical inactivity (with the possible consequence of less sunlight exposure) are factors that contribute to a greater likelihood of vitamin D deficiency in older adults, therefore making them more susceptible to osteoporosis (Wahlqvist & Savige, 2000). The Dietary Reference Intake for vitamin D is 15 mg, three times higher than for the younger adult (U.S. Department of Agriculture, National Agricultural Library, n.d.).

### Thiamine

Thiamine intakes vary considerably between different elderly populations. Thiamine deficiency is usually associated with poor intakes rather than an increased need, although some studies have shown that the biochemical status of older adults can indicate the presence of thiamine deficiency despite seemingly adequate intakes (Wahlqvist & Savige, 2000). Thiamine deficiency is often responsible for symptoms of peripheral neuropathy.

### Vitamin $B_6$

A number of studies have suggested that age-related changes occur in both the absorption and metabolism of vitamin $B_6$, and as a consequence, aged adults may have a higher requirement. Vitamin $B_6$ is commonly found in meat and leafy greens, and deficiency of this vitamin often leads to impaired immune function and impaired cognitive function.

### Vitamin $B_{12}$

Vitamin $B_{12}$ deficiency is more common among older adults because of the prevalence of pernicious anemia and atrophic gastritis, which appear to increase with age. Also, the prevalence of *Helicobacter pylori* increases with age and has been shown to be associated with vitamin $B_{12}$ malabsorption, possibly because it contributes to gastric atrophy. Deficiency of vitamin $B_{12}$ increases the risk of irreversible neurologic damage and can likely contribute to homocysteine concentrations that are associated with vascular disease (Wahlqvist & Savige, 2000).

### Fluid

Aging adults are more susceptible to the risk of dehydration resulting from alterations in thirst responses, decreasing renal function, and certain medical conditions. Moreover, a reduction in total body water with age also increases the susceptibility of elderly people to dehydration (Wahlqvist & Savige, 2000).

#### Cultural Diversity

The first baby boomers turned 60 years old in 2006. This generation will shift the demographics in the United States so that by 2030 one in five Americans will be 65 years or older. A closer look reveals interesting projections about the diversity of this aging group. Minority older adults are increasing in numbers at a much higher rate than whites are. According to the Administration on Aging, between 1999 and 2030 an increase in minority elders is projected at 217% compared with 81% in whites. Dramatic increases in the numbers of Hispanic American and Asian American elders will be experienced. Native American and African American elders will also increase in numbers greater than aging whites will (U.S. Department of Health and Human Services, Administration on Aging, 2012). How will the unique challenges of an aging population be affected by the increasing cultural diversity projected in the future? How will programs and services targeted for the older adult need to change?

## Nutrition-Related Health Problems

Although nutrition-related health problems can occur at any age, the older adult is at risk for accumulation of health problems because of years of life. In this section, vascular conditions, incontinence, visual function, and osteoporosis are discussed.

### Cardiovascular Disease

Cardiovascular disease is the most common cause of death and disability in the developed world. Dietary habits may contribute to or provide protection against the risk factors associated with cardiovascular disease (Wahlqvist & Savige, 2000). Diet influences the pathogenesis of coronary artery disease in a variety of ways. The principal mechanism by which fat and cholesterol ingestion translate into increased cardiovascular risk is the induced elevation of serum lipoproteins, especially low-density lipoprotein. The initial development of fatty streaks in coronary arteries is mediated by serum lipid levels and free radical oxidation, both of which are modified by nutrients (Katz, 2000).

An inverse relationship was found between fish consumption and coronary heart disease mortality. Also, excess alcohol consumption is associated with hypertension, a risk factor for cardiovascular disease, yet moderate consumption may be protective against cardiovascular disease through its favorable effect on high-density lipoprotein cholesterol. Elevated homocysteine levels have been identified as an independent risk factor for cardiovascular disease, and it was found that elderly adults with better folate status had lower homocysteine levels. Foods that have high amounts of folate include breakfast cereal, fruit, orange juice, and leafy green vegetables.

Dietary counseling is an essential component in the primary prevention of heart disease. It is also an essential component in the clinical management of all patients with established coronary heart disease and in those with risk factors (Katz, 2000).

### Peripheral Vascular and Cerebrovascular Disease

The dietary recommendations for the prevention and modification of cardiovascular risk generally are pertinent for peripheral vascular disease and cerebrovascular disease. Peripheral vascular disease is associated with elevated plasma homocysteine levels and, therefore, may be amenable to treatment with B vitamin and folate supplementation in certain patients. Also, elevated postprandial insulin levels appear to be an independent risk factor, suggesting that dietary intervention to improve glycemic control may play a role in the prevention and control of peripheral vascular disease (Katz, 2000).

The predominant risk factor for stroke is hypertension, which can be prevented and modified by dietary interventions. Dietary sodium restriction and generous intake of potassium, magnesium, and calcium may lower blood pressure (Katz, 2000).

### Incontinence

**incontinence** Inability to control the discharge of urine or feces.

Urinary and fecal **incontinence** are common and often debilitating conditions in older people. Urinary incontinence is reported to afflict approximately half elderly adults living in institutions and 15% to 30% of community-based elderly adults (Wahlqvist & Savige, 2000). Dietary interventions that encourage an adequate intake of fluid and consuming a variety of plant foods that are a good source of dietary fiber may assist in alleviating some of the problems that can cause incontinence (Wahlqvist & Savige, 2000).

### Visual Function

**cataract** Complete or partial opacity of the ocular lens.

**macular degeneration** An eye disease that affects the macula, a part of the retina.

**Cataracts** and age-related **macular degeneration** are common causes of visual impairment in older adults. Nutrients may play a very important role in vision; for example, the antioxidants alpha-tocopherol, beta-carotene, and ascorbic acid may help to prevent cataract formation and macular degeneration (Wahlqvist & Savige, 2000). Therefore, a diet rich in fruits and green leafy vegetables should be recommended as primary prevention of age-related eye disease. In those older than age 50 years or with less than judicious diets, supplementation with vitamin C 500 mg, vitamin E 400 to 800 IU, and zinc may be helpful in the prevention of age-related eye disease (Katz, 2000).

### Osteoporosis

According to the 2004 Surgeon General's report on bone health and osteoporosis, the cost of caring for bone fractures from osteoporosis is $18 billion each year (U.S. Department of Health and Human Services, 2004). According to the National Institute of Arthritis and Musculoskeletal and Skin Diseases (2006), **osteoporosis** affects more than 10 million adults in the United States, and millions more are at risk for the disease. Risk factors for osteoporosis are numerous and include gender, ethnicity, and lifestyle. **Table 13.5** provides a comprehensive list.

**osteoporosis** Reduction in the quantity of bone.

**TABLE 13.5 Risk Factors for Osteoporosis**

- Age
- Ethnicity
  - ➤ Asian-American
  - ➤ White
- Female gender
- Early menopause
- Family history
- Low body weight
- Medications
- Smoking
- Sedentary life-style
- Heavy alcohol consumption
- Poor calcium intake for years
- Poor vitamin D intake

**Learning Point** Sodium, caffeine, and alcohol have been negatively associated with bone status. Specific nutrients that may play an important role in maintaining bone health include protein, vitamin D, calcium, vitamin K, and boron (Wahlqvist & Savige, 2000).

Dietary management is fundamental to the primary and secondary prevention of osteoporosis. The origins of osteoporosis are in childhood and adolescence, during which time adequate physical activity and dietary calcium are particularly important. Peak bone density is reached by around the end of the third decade (Katz, 2000). Many dietary factors have implications for bone health.

Dietary recommendations should focus on diversity in the diet, consumption of low-fat dairy products, calcium- and vitamin D–fortified foods, avoiding or quitting smoking, and limiting alcohol intake. In older adults at risk, vitamin D supplementation with 400 to 600 IU and calcium intake of about 1,500 mg/day are advisable (Katz, 2000). Other recommendations should encourage engaging in consistent weight-bearing physical activity, at least some of which should be outdoors in sunlight.

## Special Considerations for the Older Woman

Women face unique challenges with aging, partially because of their long life expectancy. According to the Centers for Disease Control and Prevention, a female born in 2003 has a life expectancy of 80.1 years compared with 74.8 years for a male born in the same year. White women have a longer life expectancy than do African American women, reported at 80.5 years compared with 76.1 years, respectively (National Center for Health Statistics, n.d.). Disease and disability increase with advanced aging. Women live longer but have more chronic health conditions than men do. These conditions include osteoporosis, depression, Sjögren syndrome, rheumatoid arthritis, and other autoimmune diseases (U.S. Department of Health and Human Services, n.d.-a). Older women also are more likely than older men to live in poverty, live in long-term care, or live alone (ADA, 2005b).

One of the conditions unique to women is menopause. Like adolescence, it is a natural process in a woman's life span. A woman is in menopause when she has not had a menstrual cycle for 12 consecutive months. Although normally this occurs in the late forties or early fifties, a woman who has had her ovaries surgically removed will experience the physiologic condition.

### GROUP Project

**Critical Thinking**

Perimenopause and the transition to menopause are marked by a time in a woman's life when progesterone and estrogen levels are changing in response to an aging body. These changes are accompanied by symptoms ranging from mild to severe that can disrupt a woman's daily life. Unfortunately, other "silent" but significant changes are occurring. Bone mineral density can be lost during this time, placing the woman at risk for fractures, tooth loss from declining jawbone, pain, and disability.

The National Heart, Lung, and Blood Institute and the National Cancer Institute, parts of the National Institutes of Health, sponsored the Women's Health Initiative Hormone Program that examined the effects of hormone replacement therapy on women's health. Researchers conducted two studies using hormone therapy. One study involved estrogen alone and the other tested estrogen plus progestin. The estrogen plus progestin study was stopped in 2002 when researchers determined that the risks of heart disease, stroke, blood clots, and breast cancer outweighed the benefits. The estrogen alone study was stopped in 2004 because of increased risk of stroke and blood clots. Researchers found no significant effect on the risk of breast or colorectal cancer in the estrogen-only group (National Institute on Aging, 2005).

Hormone replacement therapy can assist in preserving bone loss, reduce the risk of colorectal cancer, and assist in managing unpleasant symptoms of menopause. Considering the known risks and benefits of hormone replacement therapy, how should a woman choose which course to take?

For many women menopause brings unpleasant side effects such as hot flashes, mood swings, sleep disturbances, night sweats, depression, and weight gain. Menopause can also bring serious health concerns because it increases the risk for bone loss and heart disease. Eighty percent of adults with osteoporosis are women (National Institute of Arthritis and Musculoskeletal and Skin Diseases, 2006). Historically, hormone replacement therapy has been used to prevent bone loss and to treat the symptoms of menopause. This practice has known benefits for bone health but has been linked to breast, uterine, and ovarian cancer and heart disease. Prescribing hormone replacement therapy is no longer routine, and recommendations are based on the woman's family history and degree of symptoms. Soy isoflavones, exercise, vitamin supplements, and stress reduction techniques are some alternatives for women who choose to forgo hormone replacement therapy because of the risks.

## Summary

Aging brings unique challenges that encompass physical, mental, social, and financial changes. Special attention to food selection is important

to meet the demands of the aging process and accompanying health issues. The ultimate goal of maintaining the highest quality of life possible through the life span is as important in the older adult as with any other age. Independence, dignity, well-being, and social interaction are desired by all human beings. Regular nutrition screening with early interventions, when problems are identified, can enhance the quality of life of the older adult.

## Case Study 1

### Risk of Malnutrition

Karen M. Funderburg, MS, RD, LD, and Migy K. Mathews, MD

Mrs. Walker is a 74-year-old non-Hispanic white woman. She is 62 inches tall and weighs 112 pounds. She has lost 2 inches in height in the past 20 years. She smokes half a pack of cigarettes a day and has been a smoker for 54 years. About 18 months ago, she began to experience mouth pain. A dental examination revealed several loose teeth as a result of severe gum disease. She has had significant dental work in the past year, which has resulted in five extracts. A partial plate with false teeth was attempted but because of continued mouth pain was not worn regularly. Mrs. Walker has altered her diet as a result of her poor dentition. She no longer eats fresh fruits, salad, steak, nuts, or other hard-to-chew foods. She rarely eats out anymore. It takes her twice as long to eat as a few years ago. She plans to have a full upper jaw extraction and be fitted for a full denture when she has the money. Since her dental problems began, she has lost 11 pounds. She is depressed and frustrated by the changes she has had to make in her diet. Eating has become a chore and has lost the pleasure it once had.

#### Questions

1. Which risk factors does Mrs. Walker have for developing malnutrition?
2. What concerns you the most about this case?
3. What suggestions to do have for Mrs. Walker?

## Case Study 2

### Lack of Appetite

Karen M. Funderburg, MS, RD, LD, and Migy K. Mathews, MD

A 79-year-old woman was evaluated in an outpatient clinic. She lives with her 81-year-old husband, who is her primary caregiver. He reports that approximately 2 years ago his wife began to lose interest in the things she enjoyed, like shopping, cooking, and sewing. Gradually, she become less interested in food and has lost 18 pounds in the past year. He has tried commercial nutritional supplements but has not been successful in getting her to consume them. He stated he is not much of a cook but has been trying his best. He is frustrated and concerned about his wife's weight loss and lack of appetite. He believes he is constantly trying to get her to eat. He does not have any family support because he and his wife never had children. Her medical evaluation revealed cognitive impairment using the Mini Mental State Examination. She does not appear to have any chewing or swallowing problems but has not seen a dentist in more than 3 years. She takes one prescription medication for her arthritis.

#### Questions

1. What questions would you like to ask her husband?
2. What strategies might be appropriate for her treatment plan?
3. Do you have concerns about her husband and his ability to care for her?

## References


Alzheimer's Association. (2006). Fact sheet: Eating. Retrieved from www.alz.org/resources/factsheets/fseating.pdf

American Dietetic Association. (2003a). Position of the American Dietetic Association: Food and water safety. *Journal of the American Dietetic Association, 103*, 1203–1217.

American Dietetic Association. (2003b). Position of the American Dietetic Association: Oral health and nutrition. *Journal of the American Dietetic Association, 103*, 615–623.

American Dietetic Association. (2005a). Position of the American Dietetic Association: Liberalization of the diet prescription improves quality of life for older adults in long-term care. *Journal of the American Dietetic Association, 105*, 1955–1965.

American Dietetic Association. (2005b). Position of the American Dietetic Association: Nutrition across the spectrum of aging. *Journal of the American Dietetic Association, 105*, 616–633.

Amin, S. H., Kuhle, C. L., & Fitzpatrick, L. A. (2003). Comprehensive evaluation of the older woman. *Mayo Clinic Proceedings, 78*, 1157–1185.

Bailey, R. L., Ledikwe, J. H., Smiciklas-Wright, H., Mitchell, D. C., & Jensen, G. L. (2004). Persistent oral health problems associated with comorbidity and impaired diet quality in older adults. *Journal of the American Dietetic Association, 104*, 1273–1276.

Blumberg, J. B. (1997). Nutritional needs of seniors. *Journal of the American College of Nutrition, 16*, 517–523.

Centers for Medicare and Medicaid Services. (n.d.). Medicare dental coverage. Retrieved from www.cms.gov/Medicare/Coverage/MedicareDentalCoverage/index.html?redirect=/MedicareDentalCoverage

Chen, C. C., Schilling, L. S., & Lyder, C. H. (2001). A concept analysis of malnutrition in the elderly. *Journal of Advanced Nursing, 36*, 131–142.

Chernoff, R. (1996). Nutrition. In D. Jahnigen & R. Schrier (Eds.), *Geriatric medicine* (2nd ed.). Cambridge, MA: Blackwell Science.

Council for Nutritional Strategies in Long-Term Care. (n.d.). New Screening Tool Detects Appetite Problems in Senior

Adults: Council on Nutrition Appetite Questionnaire. Retrieved from http://medschool.slu.edu/agingsuccessfully/pdfsurveys/appetitequestionnaire.pdf

Deering, C., Russell, C., & Womack, P. (2001). *Perspectives on dysphagia: A continuing education self-study guide for dietetics professionals*. Chicago, IL: American Dietetic Association.

de Jong, N., Mulder, I., de Graaf, C., & Van Staveren, W. A. (1999). Impaired sensory functioning in elders: The relationship with its potential determinants and nutritional intake. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *54*, B324–B331.

Gollub, E. A., & Weddle, D. O. (2004). Improvement in nutritional intake and quality of life among frail homebound older adults receiving home-delivered breakfast and lunch. *Journal of the American Dietetic Association, 104*, 1227–1235.

Katz, D. L. (2000). *Nutrition in clinical practice* (1st ed.). Philadelphia, PA: Lippincott Williams and Wilkins.

Kinney, J. M. (2004). Nutritional frailty, sarcopenia and falls in the elderly. *Current Opinion in Clinical Nutrition and Metabolic Care, 7*, 15–20.

Kurlowitz, L., & Wallace, M. (1999). *The Mini Mental State Examination (MMSE). Best practices in nursing care to older adults*. New York, NY: Hartford Institute for Geriatric Nursing.

Mathey, N., de Jong, C. P. G. M., de Groot, C., de Graaf, C., & Van Steveren, W. A. (2001). Assessing appetite in Dutch elderly with the Appetite, Hunger, Sensory Perception (AHSP) questionnaire. *Journal of Nutrition, Health, and Aging, 5*, 22–28.

Morley, J. E. (2001). Decreased food intake with aging. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 56*, 81–88.

National Center for Chronic Disease Prevention and Health Promotion. (2005). Fact sheet: Key findings from NHANES 1999–2002. Retrieved from www.ct.gov/dph/lib/dph/state_health_planning/dphplans/environ_pub_hlth_tracking_plan_2005.pdf

National Center for Health Statistics. (n.d.). Health, United States, 2010: Table 22: Life expectancy at birth, at 65 years of age, and at 75 years of age according to race and sex, United States selected years, 1900–2003. Retrieved from www.cdc.gov/nchs/data/hus/2010/022.pdf

National Dysphagia Diet Task Force. (2002). *The National Dysphagia Diet: Standardization for optimal care*. Chicago, IL: American Dietetic Association.

National Eye Institute. (2005). Age-related macular degeneration. Retrieved from http://nihseniorhealth.gov/agerelatedmaculardegeneration

National Institute on Aging. (2005). Hormones and menopause. Retrieved from www.nia.nih.gov/health/publication/hormones-and-menopause

National Institute of Arthritis and Musculoskeletal and Skin Diseases. (2006). Osteoporosis. Retrieved from www.niams.nih.gov/Health_Info/Bone/Osteoporosis

National Institute on Deafness and Other Communication Disorders. (2006). Strategic plan for National Institute of Deafness and Other Communication Disorders. Retrieved from www.nidcd.nih.gov/staticresources/about/plans/strategic/strategic06-08.pdf

National Institute of Dental and Craniofacial Research. (2005). Dry mouth. Retrieved from www.nihseniorhealth.org/drymouth

Nestlé Clinical Research. (1998). Mini Nutritional Assessment. Retrieved from http://mna-elderly.com

Nutrition Screening Initiative. (2004). Determine your nutritional health checklist. Retrieved from www.hospitalmedicine.org/geriresource/toolbox/pdfs/determine.pdf

Ritchie, C. S. (2002). Oral health, taste, and olfaction. *Clinics in Geriatric Medicine, 18*, 709–717.

Sahyoun, N. R., & Krall, E. (2003). Low dietary quality among older adults with self-perceived ill-fitting dentures. *Journal of the American Dietetic Association, 103*, 1494–1499.

Ship, J. A. (2002). Improving oral health in older people. *Journal of the American Geriatrics Society, 50*, 1454–1455.

U.S. Department of Agriculture, National Agricultural Library. (n.d.). Dietary Reference Intake tables. Retrieved from http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables

U.S. Department of Health and Human Services. (n.d.-a). Sex differences in autoimmune disease. Retrieved from www.womenshealth.gov/statistics/statistics-by-topic/autoimmune-diseases.cfm

U.S. Department of Health and Human Services. (n.d.-b). Sex differences in musculoskeletal health. Retrieved from www.womenshealth.gov/publications/our-publications/the-healthy-woman/autoimmune_diseases.pdf

U.S. Department of Health and Human Services. (2004). *The 2004 Surgeon General's report on bone health and osteoporosis: What it means to you*. Retrieved from www.surgeongeneral.gov/library/reports/bonehealth/OsteoBrochure1mar05.pdf

U.S. Department of Health and Human Services, Administration on Aging. (2009). The Elderly Nutrition Program. Retrieved from www.nutrition.gov/food-assistance-programs/elderly-nutrition-program

U.S. Department of Health and Human Services, Administration on Aging. (2012). Diversity. Retrieved from www.aoa.gov/AoA_programs/Tools_Resources/diversity.aspx

U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. (n.d.). *Healthy People 2010*. Retrieved from www.healthypeople.gov/2010

Wahlqvist, M. L., & Savige, G. S. (2000). Intervention aimed at dietary and lifestyle changes to promote healthy aging. *European Journal of Clinical Nutrition, 54*, S148–S156.

Wilson, M. M., Thomas, D. R., Rubenstein, L. Z., Chibnall, J. T., Anderson, S., Baxi, A., Diebold, M. R., & Morley, J. E. (2005). Appetite assessment, simple appetite questionnaire predicts weight loss in community-dwelling adults and nursing home residents. *American Journal of Clinical Nutrition, 82*, 1074–1081.

CHAPTER 14

# Special Topics Related to the Registered Dietitian and Older Adults: Roles and Responsibilities of the Registered Dietitian in Long-Term Care

*Victoria Hammer Castellanos, PhD, RD, and Angela Sader, RD, LD, MBA*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Discuss the fundamental differences between nutrition care delivery in the long-term care setting compared with other care settings, including prioritization of resident quality of life and a regulatory environment that drives patient care.
2. Describe the clinical and management responsibilities that regularly fall to the dietitian within the long-term care setting.
3. Identify the management opportunities for long-term care dietitians within the areas of clinical care, food service management, survey management, and risk management.

## Unique Characteristics of Long-Term Care

Approximately 1.4 million Americans lived in U.S. nursing homes and skilled nursing facilities during 2009 (American Health Care Association [AHCA], 2011). A number of factors set apart the delivery of nutrition care in nursing homes compared with other care settings because of the unique culture and challenges of long-term care (Castellanos, 2004). The nursing home environment has the following characteristics:

**risk management**
Identification of areas where harm may occur to an individual, such as in the case of a fall with injury. Taking precautions to limit the number of times this situation may occur is the responsibility of facility management.

- The facility is considered to be the resident's home. Although some residents stay in the facility only for a few weeks or months, many residents live out the remainder of their lives in this setting.
- A large segment of the population is both very old and quite frail. However, a growing number of residents are somewhat younger adults who are suffering from an acute illness or injury and who expect to return home after a period of recovery or rehabilitation.
- Usually, both long-term- and short-term-stay residents suffer from a host of chronic and acute disease conditions.
- The resident's physical ability to eat enough food is often greatly reduced because of aging and/or disease processes.
- Nutrition care is, at the same time, both driven and hindered by the regulatory environment. The characteristics of skilled nursing facilities and of the residents make nutritional well-being difficult to achieve in this setting but present a rare opportunity for the nutrition professional to significantly affect both the health outcomes and quality of life of a large number of people.

## Types of Long-Term Care Centers

Long-term care (LTC) is a designation given to any facility-based operation that has accepted the responsibility of caring for patients deemed to require some form of 24-hour-a-day supervision to meet their care-related needs. The types of LTC centers are described in **Table 14.1**. The goals of all LTC facilities are similar: Each is expected to help the patients under care reach the highest level of living and activity possible through the administration of nonskilled and/or skilled services.

Whether a person is qualified to reside in an LTC facility primarily depends on a certified physician recommending that individual for LTC services. State and **federal regulations** provide guidance to physicians regarding placement decisions. The acuity level of LTC residents varies from residents who have a relatively small need for services to residents who require extensive observation, care, and oversight to ensure quality care.

**federal regulations**
Documents published by the Centers for Medicare and Medicaid Services for use in the survey process for long-term care facilities.

Significant cost is associated with LTC. Certain programs are designed to reduce the personal financial burden, including Medicare (federally funded), Medicaid (state indigent care funds), Medicare Part B (federally funded), Veterans Administration (federally funded), insurance, and hospice (federally funded). All these payer sources are designed to help individuals requiring LTC services to

**TABLE 14.1 Types of LTC Centers**

| Facility Type | Function |
|---|---|
| Skilled nursing facilities (SNFs, pronounced "sniffs") | Facilities approved to accept Medicaid and Medicare for payment when the resident qualifies. The resident population of most SNFs represents a variety of payer sources, i.e., Medicaid, Medicare, insurance, hospice, and self-pay. |
| Hospital-based facilities | LTC facilities under the umbrella of a hospital organization. These are usually SNFs but may be private placement or government sponsored. Because the billing mechanism is quite different in an acute care hospital from that of an LTC facility, the RD employed in this environment will be required to keep careful track of hours spent in each unit. |
| Private placement facilities, also called private pay facilities | Facilities in which the resident assumes all responsibility for payment of stay, either through insurance or self-pay. |
| Government-sponsored facilities | Medicaid certified but not Medicare certified |

receive those benefits even if personal funds are not available.

LTC facilities range in size from a few to more than 200 residents. Depending on their business classification, facilities may be designated as for profit or not for profit, private ownership, or publicly traded. Expectations for resident care are similar across business classifications, but internal rules and policies may vary depending on acuity of residents, number of residents involved, and reimbursement from external sources.

The exact cost of care in an LTC facility varies depending on many factors. Pricing is affected by the number of associates the individual requires to assist with care (i.e., does it require one or two persons to move the resident?), room size, age of facility, services offered, the level of care, acuity of the resident, and many other factors. It is common practice for a daily rate sheet to be provided to a potential resident that lists the services covered for a flat daily fee. This varies from facility to facility. Additional services must be reimbursed by the resident's personal funds. For example, the daily rate may not cover the cost of services provided by the beautician, cable television, and so on. Although residents receive similar care regardless of payer source, out of consideration for the resident, it is important that registered dietitians (RDs) are aware of services that may cause a financial burden to the resident. For example, if a resident is not covered under Medicare or if he or she does not have private insurance, the resident is responsible for the cost of goods and services such as adaptive eating devices and laboratory blood work. When possible, the RD should strive to achieve positive results with care approaches that are not a financial burden to the resident or family (e.g., only recommend that additional blood work be completed when the results of the laboratory analysis are clearly necessary to determine whether the current plan of care should be modified).

## Nursing Home Regulation Drives Nutrition Care

The regulatory environment has a tremendous impact on nutrition care in the LTC setting. Nursing homes are heavily regulated at both the federal and state levels. This is in contrast to community-based care and other types of LTC that are regulated only by the states; regulations for community-based care are generally sparse. The **Centers for Medicare and Medicaid Services** enforces many specific regulations related to food and nutrition care delivery in nursing homes (AHCA, 2013). The Centers for Medicare and Medicaid Services normally contracts with state health departments to complete the surveys according to federal regulations (i.e., "state survey"). Each state has additional regulations that must also be followed and that are enforced by the state surveyors.

**Centers for Medicare and Medicaid Services** The federal agency responsible for nursing home regulations and their enforcement. The Centers for Medicare and Medicaid Services is part of the U.S. Department of Health and Human Services.

The extent to which the state and federal regulations drive nutrition care delivery in the nursing home setting cannot be underemphasized. Success as an LTC practitioner requires health professionals to become intimately familiar with these regulations. To help drive this point home, we have referred to some of the relevant federal nursing home regulations throughout this chapter. A summary of the **federal nursing**

**federal nursing home regulations for nutrition services** Documents published by the Centers for Medicare and Medicaid Services for use in the survey process for long-term care facilities that focus on the nutritional oversight and delivery to the residents in the facility.

**home regulations for nutrition services** can be found at the American Health Care Association website (see http://www.ahcancal.org/searchcenter/Pages/Results.aspx?k=federal%20nursing%20home%20regulation%20for%20nutrition%20services).

## Structure of the Nutritional Services Department

The nutritional services department is composed of the RD, the dietary service manager (DSM), dietetic technician, and all staff involved in food production and service. It is the standard division of responsibilities in most nursing homes that the nutritional services department is responsible for getting the appropriate food and drinks to the residents' dining area, whereas it is the responsibility of the nursing staff to feed the resident. Licensed nurses are responsible for supervising the provision of food and fluid to residents by certified nursing assistants. During their training, certified nursing assistants are taught techniques to assist and/or feed residents with various kinds of health problems, including dysphagia (swallowing disorders). Federal regulation also allows for paid feeding assistants to help feed residents at low risk for choking, although not all states allow paid feeding assistants.

The basis for nutritional services staffing in the LTC facility begins with regulations F361 and F362, which state that the facility must have adequate personnel to provide dietary services and that a qualified dietitian is utilized in planning, managing, and implementing dietary service activities. The RD staff is then expanded to meet the requirements of the residing state and the needs of the facility residents.

State staffing requirements are highly variable. A particular state may go so far as to define the components of a nutritional assessment and identify the professionals appropriate to complete the task as a whole or parts of the task. A state may also specify the minimum number of hours an RD must be present in a facility to provide services or dictate the number of RD hours based on facility census or the number of nursing stations. Some states have requirements related to the dietary manager that affect both DSM and RD staffing. For example, if a state requires that the DSM complete an approved course by the Dietary Managers Association to become a "certified dietary manager," completion of the course requires oversight and training of the DSM by the RD. The assignment of responsibilities within the nutritional services department varies depending on facility characteristics and state regulations.

**Learning Point**

The RD is the healthcare professional responsible for overseeing and guiding facility staff in food and nutrition matters. The RD is responsible for reviewing the menu (e.g., for nutritional adequacy, appropriate therapeutic combinations, serving sizes), reviewing and providing guidance in kitchen sanitation, reviewing proper preparation of food and use of recipes, providing staff education, and providing residents with medical nutrition therapy (MNT) through assessment and appropriate interventions. The RD uses his or her expertise in food safety and sanitation as well as food production to turn out a healthy meal that meets the MNT needs of the residents.

Facilities frequently use a DSM to manage department staffing, meal production, procurement, food service sanitation, and the budget. As per regulation, the DSM must be under the oversight of an RD. In a smaller facility, and if state regulation allows, it is not uncommon for the DSM to be the staff member responsible for the collection of subjective and objective resident information to be used by the RD in his or her resident assessment. In this scenario, it is the responsibility of the RD to review the DSM's completion of the aforementioned activities. If a facility is larger or if a state requires that a licensed/registered healthcare practitioner complete documentation in the medical record, the facility may hire a dietetic technician to assist with clinical documentation. Various other staffing scenarios are also possible.

It is becoming more common for Certified Chefs to be hired by LTC facilities. Although these individuals bring a welcome breadth of cooking knowledge and customer service skill, they are not seen as the food and nutrition expert and cannot replace the RD for kitchen sanitation and production oversight.

After meeting the regulatory requirements, RD staffing in a facility is largely determined by the facility census, the level of care provided, and the average number of admissions per week. Individual facilities usually have a predetermined number of beds set aside for Medicare (i.e., short term) versus long-term residents. Residents who are admitted under Medicare after discharge from an acute care facility are usually more medically complex than long-term-stay residents. These residents are more likely to be on dialysis, have pressure ulcers, require enteral therapy or total parenteral nutrition (TPN), or require other types of MNT. Further, Medicare beds turn over

more frequently, and with each new admission it is necessary for the RD to spend a significant amount of time on the initial physical and nutritional assessment and development of comprehensive **care plans**.

**care plans** As per the Centers for Medicare and Medicaid Services regulation, a facility must use an interdisciplinary approach to develop a comprehensive plan of care for each resident that includes measurable objectives and timetables to meet a resident's medical, nursing, mental, and psychosocial needs. These care plans must be revised as the resident's status changes.

It is a common mistake to assume nutritional services staffing should be the same for equivalently sized facilities (e.g., 120 beds) in the same state. However, the facility with 25 Medicare beds (resulting in an average of seven new admissions per week) and 10 residents receiving enteral feeding requires more RD time for admission assessments, MNT documentation, and enteral therapy follow-up than does a facility with only 4 Medicare beds and 2 residents receiving enteral feeding.

### Roles of the Dietitian Within the Nutritional Services Department

Whether the services of an RD are provided by a consultant dietitian or regular employee of the company, the role of the RD in the LTC setting is central to the health and well-being of the residents and goes far beyond the responsibility of patient assessment. The RD's time in the facility is not only spent on MNT but in review of the nutritional services department and all the processes related to providing nutrition care to the residents. In the LTC setting the RD must be prepared to use skills in staff and patient education, quality management, menu planning, food production, food safety, product selection and purchasing, emergency planning, and budget management, including labor utilization.

One of the most critical functions of the dietitian in the nursing home setting is to make sure that the facility is meeting both federal and state regulations relevant to nutrition services. The nutrition professional in the nursing home setting uses a wide variety of skills and training to assist a facility in maintaining compliance with regulations and provide appropriate care to residents.

### Clinical Care Delivery in the LTC Setting

Regulations require nursing facilities to meet the nutritional needs of residents (F325–327 and F360–367) while maintaining their dignity and quality of life (F240 and F241). This also is the ethical responsibility of providers.

Providing care in the LTC setting affords tremendous opportunity for the care provider. That is to say that the nursing home has both the infrastructure and consistency of client contact that make it possible to move beyond a *reactive* medical model and to adopt a *proactive*, prevention-focused approach to nutrition care. For example, weight loss and pressure ulcer prevention committees are often created in facilities. These committees meet frequently to review individual residents who have risk factors for weight loss or pressure ulcers and to initiate interventions designed to prevent weight loss or wounds from developing.

The nutritional needs of nursing home residents are likely to be affected by both illness and advanced age. Most nursing home residents are medically frail, with at least five chronic health conditions, and suffer intermittent bouts of acute illnesses, such as infection and diarrhea. These disease states tend to increase nutrient needs. Further, some aspects of institutional living may affect nutritional needs; for example, little or no exposure to sunlight makes residents highly dependent on dietary and supplemental sources of vitamin D (Institute of Medicine, 1997).

Most nutrition-related problems in nursing homes are a consequence of undernutrition. These may include unintended weight loss, protein-energy malnutrition, pressure ulcers, and dehydration (American Dietetic Association [ADA], 2010). A host of additional nutrient deficiencies are likely to occur as an accompaniment to inadequate food intake, including vitamin and mineral deficiencies. For most nursing home residents, the risk of morbidity and mortality related to undernutrition exceeds the potential for adverse outcomes of chronic disease (ADA, 2010). Having said this, there are few data on which to base standards of nutrition care for the frail oldest adults.

There are few published data regarding the food and fluid intake patterns of LTC residents. Some reports suggest that LTC residents consume a greater proportion of their breakfast meal than they do of other meals, especially dinner (Castellanos, Surloff, & Giordan, 2003; Chapman, Samman, & Liburne, 1993; Endres, Welch, Ashraf, Banz, & Gower, 2000; Simmons & Reuben, 2000; Young, Binns, & Greenwood, 2001); however, there is typically a smaller quantity of food served at breakfast than at lunch and dinner. A study by Young and associates (2001) found that older adults with dementia tend to eat less food as the day progresses.

Also, a study looking at snack interventions found that residents consumed significantly less during the evening snack period than during the morning or afternoon snack (Simmons & Schnelle, 2004).

Inadequate hydration is a significant issue in the LTC setting (Chidester & Spangler, 1997; Kayser-Jones, Schell, Porter, Barbaccia, & Shaw, 1999). Older adults in nursing facilities are likely to be unwell, have limited access to palatable fluids, and are at greater risk for suboptimal hydration than other older people (Chidester & Spangler, 1997). Inadequate water intake can contribute to acute confusion, urinary tract infections, pressure ulcers, constipation, and adverse drug responses (American Medical Directors Association, 2002). These problems can lead to increased morbidity and mortality and can escalate healthcare costs (American Medical Directors Association, 2002; Chidester & Spangler, 1997).

Older people do not always feel thirsty, even when they are dehydrated (Rolls & Phillips, 1990). It may be difficult or impossible for older people with neurologic or musculoskeletal disabilities to obtain or consume liquids independently. In addition, it is not uncommon for nursing home residents to have medical conditions (diarrhea, vomiting, and dysphagia) or to be prescribed treatments (diuretic medications) that put them at increased risk for dehydration. Fortunately, systems put in place to ensure adequate resident assistance with eating are likely to have the added benefit of supporting adequate intake of fluids. For example, one study found that verbal prompting increased fluid intake in 71% of residents, with the range of increase of 5 to 20 oz/day (Simmons, Alessi, & Schnelle, 2001). Also, hydration programs can be implemented to ensure that fluids offered between meals or with medications are of significant volume, frequency, and palatability to appreciably increase overall fluid intake.

LTC facilities also are filled with people suffering from acute and medically complex illnesses that were once found only in hospitals. For example, TPN management and knee and hip replacement therapy are now occurring in the LTC facility instead of the hospital. At one time hospitalized people stayed in the hospital until they were well enough to be discharged home. In the current healthcare system, these "patients" are becoming "residents" of skilled nursing facilities. Thus, the LTC facility also must have the clinical staff and infrastructure to provide adequate care to severely ill people who often do not eat or drink enough to meet their nutritional needs.

Residence in a nursing home for a significant portion of a person's life is unlike a brief admission to a hospital, where limited choice and reduced quality of life are justified by short-term clinical goals. Further, research supports that therapeutic diets are at worst detrimental and at best of neutral benefit for LTC residents (Pioneer Network Food and Dining Clinical Standards Task Force, 2011). With these considerations, LTC facilities have begun to move away from standardized meals and snacks to a system driven by resident individualization and choice. A Food and Dining Clinical Standards Task Force, under the leadership of the Pioneer Network, has developed new Dining Practice Standards that have been agreed to by 12 national clinical standard-setting associations (Pioneer Network Food and Dining Clinical Standards Task Force, 2011). These Dining Practice Standards support individualized care and self-directed living versus traditional diagnosis-focused treatment for people living in nursing homes (see **Table 14.2**).

### Nutrition Care Process and Timeline

It is important to identify current nutrition problems and the risk of future nutrition problems in a timely fashion. This requires a logical and

**TABLE 14.2** **Dining Practice Standards**

- Individualized Nutrition Approaches/Diet Liberalization
- Individualized Diabetic/Calorie Controlled Diet
- Individualized Low-Sodium Diet
- Individualized Cardiac Diet
- Individualized Altered Consistency Diet
- Individualized Tube Feeding
- Individualized Real Individualized Honoring Choices Food First
- Shifting Traditional Professional Control to Individualized Support of Self-Directed Living
- New Negative Outcome

The following associations agreed to these standards: Academy of Nutrition and Dietetics, American Medical Directors Association, American Association for Long-Term Care Nursing, American Association of Nurse Assessment Coordination, American Occupational Therapy Association, American Society of Consultant Pharmacists, American Speech-Language-Hearing Association, Dietary Managers Association, Gerontological Advance Practice Nurses Association, Hartfort Institute for Geriatric Nursing, National Association of Directors of Nursing Administration in Long Term Care, and National Gerontological Nursing Association.

preplanned sequence of screening, assessments, and reviews. Many of the timeframes are dictated by federal standards, but an individual facility may choose to monitor its residents more closely than required. The policy and procedure manual in the facility should outline the timeframe for all phases of nutrition care.

Within 24 hours of admission to the facility, a member of the nutritional services department should conduct an initial interview to greet the resident, confirm the diet prescription, identify meal preferences and food dislikes, screen the resident for nutrition risk, and introduce the resident to the food service system. The DSM, dietetic technician, or RD may conduct this interview. A brief chart note should be written, and the note should state that the diet order has been confirmed and the resident has been interviewed and screened. Many times, this type of note concludes by stating that a complete **nutrition assessment** will follow.

**nutrition assessment** A thorough evaluation of the resident's food and eating preferences, medical and nutritional status, estimation of nutritional needs, and identification of medical conditions/treatments and other factors that might make the resident at risk of developing malnutrition in the future. This information is used to develop the individualized plan of nutrition care for the resident and/or to determine whether the current nutritional regimen is adequate to meet the resident's nutritional needs.

**nutrition screening** A quick review to determine whether the resident meets any known factors for nutrition risk that would require an immediate assessment by a registered dietitian.

A **nutrition screening** is a quick review to determine whether the resident meets any known factors for nutrition risk. Each facility should outline a screening process to identify which residents need immediate assessment and which residents should be given a chance to settle in to the facility before the assessment takes place. Typically, a nutrition screening ascertains weight history, albumin level, diagnosis, the presence of alternative forms of feeding, and skin condition. The nutrition screening or interview process should be sensitive enough to identify new residents with feeding tubes, intravenous lines, a diagnosis of malnutrition or dehydration, and other significant nutrition problems. An immediate assessment is warranted with certain diagnoses or the presence of a feeding tube.

Per regulations (F272 and F276), a complete nutrition assessment should be conducted within the first 14 days of admission. Many facilities improve on this standard and make it a policy to complete the assessment by the 7th day of admission. It is often advisable to wait until the resident has been in the facility for several days before completing an assessment. This short delay allows time for the resident to become accustomed to the new surroundings and for body weight and baseline laboratory data to be obtained, dining location secured, and hospital records copied for the nursing home chart. A number of resources are available to assist the RD in completing the entire assessment. The Academy of Nutrition and Dietetics (AND) has described in detail the components of the nutritional assessment, which is an essential part of the nutrition care process (Lacey & Pritchett, 2003). In addition, the AND practice group Dietitians in Health Care Communities (see http://www.dhccdpg.org) has developed written resources that are specifically designed to support the assessment process in the LTC setting.

A review of each resident must be performed a minimum of every 3 months (i.e., quarterly). A quarterly review is not as in-depth as a full initial assessment but should determine whether the current nutritional regimen is adequate to meet the resident's nutritional needs. If the resident has had a change in medical condition or suffers a significant weight loss, a full assessment must be done even if it is not scheduled. In addition, a full assessment, equivalent to the initial assessment, must be completed at the end of each year of residency.

One unique component of the medical record in nursing homes is the **Resident Assessment Instrument (RAI)**. The RAI consists of three basic components: (1) the **Minimum Data Set (MDS)**; (2) **Care Area Assessment (CAA)** process; and (3) RAI guidelines.

**Resident Assessment Instrument (RAI)** Part of the medical record in skilled nursing facilities and includes the Minimum Data Set, Care Area Assessment process, and the RAI guidelines.

**Minimum Data Set (MDS)** An electronic summary of the medical status of each resident mandated by the Centers for Medicare and Medicaid Services.

**Care Area Assessment (CAA)** The CAA is an analysis of the MDS data and provides a framework for guiding the review/assessment of 20 care areas. When an area is "triggered" by MDS data, further assessment is required to clarify the resident's functional status and determine the root cause of impairments to guide intervention.

The MDS is an electronic standardized instrument used to assess nursing home residents. It is a collection of basic physical, functional, and psychosocial information about each resident. The MDS section K, Swallowing/Nutritional Status, may be completed after the initial nutritional assessment is complete or it may be completed before the nutritional assessment because it requires only basic objective data that are independent of an assessment provided by the RD. Often facilities employ an RN as an MDS coordinator to complete the entire MDS, but it is preferable that the nutrition care professional be responsible for completing the nutrition section.

In some cases, the MDS section K may be completed by the DSM because it is the gathering of objective data from the chart. In this case, the MDS coordinator or the RD will review and sign off that section K has been completed successfully.

The CAA is an analysis of the MDS data and provides a framework for guiding the review/assessment of 20 care areas. When an area is "triggered" by MDS data, the goal is to use this information to clarify the resident's functional status and determine the root cause of impairments in order to guide intervention. Nutrition status is Care Area 12, and the following is used to trigger nutrition as an area needed for further assessment: dehydration, BMI below 18.5 or above 24.9, significant weight loss, planned or unplanned weight gain, parenteral or IV feeding, mechanically altered diet, therapeutic diet, one or more pressure ulcers stage 2 or higher.

The RAI process is taking the triggered care information and the MDS data, along with other pertinent information, to develop a care plan within 14 days of the initiated assessment. It is recommended that the same person who completed the nutrition assessment also completes the RAI process.

The care planning process is an interdisciplinary process, and the nutrition team member should communicate to those who relate directly to nutrition and contribute to nursing care plans. The nutritional department usually writes care plans that involve weight loss, dehydration, tube feedings, and nutritional deficiencies, with other disciplines adding on their approaches as necessary. Nutrition interventions should be found on all care plans that involve any diet-related approaches. For example, nutrition plays a role in many care plans seen as nursing issues, such as hypertension, pressure ulcers, congestive heart failure, and risk of aspiration. Care plans should be updated whenever there is a new intervention or, otherwise, at least quarterly.

### Clinical RD Accountability in the LTC Setting

What is expected of the clinical dietitian, including what is written in the medical record, is significantly different in a nursing home compared with an acute care facility. The paradigm is fundamentally different because of the length of time each person spends in the facility, the regulatory environment of an LTC facility, and the central role the resident and the resident's family play in determining what care will be provided.

The average length of stay in an acute care facility is days to weeks, which compares with an average length of stay of months to years in an LTC facility. This extended stay provides significantly more opportunity for the RD to have an impact on the resident's health but brings with it a proportional increase in the RD's accountability for resident care and outcomes. In the LTC setting there is sufficient opportunity to identify the full extent of the resident's nutritional issues, as well as other factors that contribute to a resident's nutritional risk, and there is a higher expectation that each nutritional issue and risk factor will be addressed. At the same time, invasive assessment and treatment methods that are used in the acute setting, such as frequent laboratory blood analysis, may be limited unless the outcome of the procedure is expected to result in a change in care that will improve the resident's quality of life.

The LTC regulations also go much further in specifying the details of clinical care. The interpretive guidelines, which have been developed to assist surveyors in evaluation and providers in compliance with the intent of the federal regulations, often are quite specific regarding how care for various conditions should be provided: "Unless contraindicated, nutritional goals for a resident with nutritional compromise who has a pressure ulcer or is at risk of developing pressure ulcers should include protein intake of approximately 1.2–1.5 gm/kg body weight daily (higher end of the range for those with larger, more extensive, or multiple wounds)" (AHCA, 2005). This is very different from the approach used by the Joint Commission, the accrediting body for hospitals, which does not identify such specific standards of care for various conditions. Thus, it is incumbent upon the LTC RD to have a detailed knowledge of the interpretive guidelines and other regulatory language and to conduct assessments and to provide care in a manner that is consistent with them.

There is also an expectation that the dietitian will be proactive and intervene *before* a problem becomes significant. For example, an RD should recognize when a resident has a nutrition risk factor and bring the issue to the care team so it can be addressed before nutritional status is compromised.

Another aspect of LTC that is different from acute care is the care plan process. In LTC, the care plans for an individual resident should be as much the resident's and family's plan of care as it is the provider's (i.e., it should be individualized to meet the needs and desires of the resident and be interdisciplinary in nature). If a resident or a responsible

family member refuses some aspect of care, relevant conversations with family members must be documented, including documentation that the risks and benefits have been explained. It is also the case that the members of the care plan team (e.g., nurses, therapists, dietitians, social workers) have significantly more authority in the LTC setting than they would in the acute care setting. The care planning team can, and often does, make recommendations to the physician based on their assessments.

### Discharge Education

Discharge education occurs in the LTC facility less often than in an acute care setting but is needed for individuals who will discharge to a setting that provides a lower level of care, either after rehabilitation or strengthening. The dietitian needs to make sure that educational materials are available and that training is provided to both nursing staff and the DSM so that, in the dietitian's absence, discharge education may be provided to the resident, family, or other caregivers. Materials often are taken from the diet manual of the facility or are available through other resources, such as the Academy of Nutrition and Dietetics, the American Heart Association, or the National Dairy Council.

### Modified Diet Menu Writing and Approval and Modified Diet Prescriptions

The nutrition care for the resident begins with a menu made up of standardized recipes in an accepted regional pattern to meet the nutritional needs of the general elderly population. Federal regulation requires that the meals meet the needs of residents in accordance with the Recommended Dietary Allowances and that planned menus are followed (F363). A complete understanding of menu and recipe mechanics and the systems related to them is key to the RD's ability to help a facility succeed in meeting its clinical and operational goals. A menu must be nutritionally sound to have a chance of meeting clinical goals, and the financial impact of a poorly planned menu is enormous. Before altering or building a menu for a facility, an RD should become familiar with the procurement process, products available through the process, nutritional content of the foods available, the budget around labor and food cost, staff skill set, equipment available, the diet manual adopted, the Dining Practice Standards, and the facility's goals regarding customer satisfaction.

If the RD is serving in the capacity of a consultant, his or her involvement in menu creation will vary based on the operational support provided by the facility; for example, whether menus are available from a corporate office, whether menus are purchased from a vendor, whether dietary software is available, and whether purchasing programs are set up with a vendor. The assistance needed by the facility may include all or part of the following:

- Writing the entire menu, including finding recipes for texture modifications and therapeutics such as dialysis diets
- Writing the occasional combination diet for which there is no menu extension, such as a purée diet that is also a consistent carbohydrate diet
- Approving menu changes in response to facility/resident preferences or a food availability issue
- Reviewing disaster menus and plans or creating disaster menus and plans for multiple scenarios, such as no electricity, partial electricity, or evacuation to a local school or church

Many resources are available regarding disaster planning, including information in diet manuals and from consultant companies.

As discussed previously, the LTC facility is viewed as the resident's home, and the approaches should be the same as they would be at home: to maintain health and to have the highest quality of life possible. One area where this philosophy dramatically affects care is in the area of therapeutic diet prescriptions. Although there may be an occasion where a therapeutic restriction is necessary, the quality of life and nutritional status of older residents in the LTC facility may be enhanced by liberalization of the diet prescription. Food consumption is frequently compromised because of a combination of acute and chronic disease conditions, such that any additional restrictions that would reduce food acceptability and intake would be counterproductive to achieving the care goals.

In addition to making diet recommendations intended to maintain each resident's health and quality of life, the RD in the LTC setting also has the responsibility to evaluate the success of those recommendations over time. Because the facility provides care to most residents over a period of weeks to months, the RD must regularly reassess the resident and determine whether the current

diet prescription is resulting in significant clinical progress or is otherwise meeting the resident's care goals.

## Increasing and Maintaining Resident Food Intake

Dining is much more than just the consumption of food; the smells, sounds, conversations, and anticipation and recall of memories inspire us to eat.

**Learning Point** One of the most important contributions of the RD is to make sure the dining environment is one that optimizes resident food intake.

A multifacility study conducted by the UCLA Borun Center examined the relationships between 16 nutrition care processes and prevalence of weight loss in nursing facilities (Simmons, Garcia et al., 2003). There were no identifiable differences in the RD medical record documentation between the low-weight-loss and the high-weight-loss facilities. Instead, the major difference between the high- and low-weight-loss facilities was the dining environment. The staff at the low-weight-loss nursing homes consistently provided verbal prompting and social interaction during meals to a greater proportion of the residents, including those most at risk for weight loss. These data suggest that it is in the area of meal service and dining that the expertise of the RD may not be fully utilized in many LTC facilities.

The provision of feeding assistance at meals (social interaction throughout the mealtime period, graduated verbal prompting that enhances self-feeding capabilities, and physical assistance to the degree required) has been found to have a dramatic effect on food intake. One study found that 50% of residents previously identified as having low food intake responded to feeding assistance by increasing their food intake more than 10%. A subset of the residents (39%) in this study were identified as "highly responsive" to the intervention and increased their meal intake over 2 days from 48% to an average of 74% consumed (Simmons, Lam, Rao, & Schnelle, 2001). Across studies, Simmons and coworkers (Simmons, Alessi et al., 2001; Simmons, Osterweil, & Schnelle, 2001; Simmons & Schnelle, 2004) consistently found that 40% to 50% of residents who had one-on-one feeding assistance significantly increased their oral food and fluid intake during mealtime.

Similarly, some residents have responded to additional feeding assistance with snacks; enhancing between-meal snack programs is one alternative for increasing dietary intake (Rahman & Simmons, 2005). A study by Simmons and Schnelle (2004) found that 70% of the residents who were not responsive to increased feeding assistance at meals did significantly increase their between-meal intake when targeted for additional attention and assistance during snacks. With the snacking intervention, calories consumed between meals (supplement 1 snacks) increased to 380 kcal/day from 96 kcal/day under usual care (Simmons & Schnelle, 2004). The second and third most preferred nutrition interventions among family members of residents were improved feeding assistance and the provision of multiple small meals and snacks, respectively (Simmons, Lam et al., 2003).

Family members of nursing home residents most prefer an intervention that includes the provision of more nutritious food (Simmons, Lam et al., 2003). This often is referred to by dietitians as a "food first" approach, and many facilities have a policy for such an offering. There is some evidence that the energy and nutrient intakes of nursing home residents can be significantly increased by increasing the energy/nutrient density of several foods across two or more meals each day (Barton, Beigg, Macdonald, & Allison, 2000; Castellanos, Surloff et al., 2003; Olin et al., 2003, 1996), where the energy density of a food is defined as the energy content per weight or volume of food served (Kral & Rolls, 2004). Increasing the nutrient density of the foods residents are already choosing to eat, instead of providing substitute foods or supplements, also allows residents both choice and control over their environment (Castellanos, Silver, Gallagher-Allred, & Smith, 2003; Simmons, Lam et al., 2003). Further, research has shown that food preferences and acceptability are important determinants of amounts consumed (de Jong, Chin-A-Paw, de Graff, de Groot, & van Staveren, 2001), particularly for residents who are cognitively intact (Simmons, Alessi et al., 2001). Often, the enhanced items are varied or rotated to limit the chance of taste fatigue for the resident.

Provision of liquid nutrition supplements is one of the nutrition interventions least preferred by residents' family members (Simmons,

Lam et al., 2003). Although the data are limited, studies suggest that some residents are willing to consume supplements and do benefit from them (Johnson, Dooley, & Gleick, 1993). However, as many as 40% of residents do not consume the liquid supplements they are provided (Johnson et al., 1993; Ross, 1999). Dietitians, however, perceived supplemental products to be second only to tube feedings in terms of effectiveness for increasing intake (Cluskey & Kim, 1997). It should be noted that there is some evidence that supplements consumed with meals cause early satiation (reduce intake of other foods provided in the meal) (Wilson, Purushothaman, & Morley, 2002); thus, the greatest benefit to residents is likely to occur when liquid supplements are provided between meals. For residents with poor appetite, replacing restrictive diets with a wider range of food choices also may improve intake and quality of life (ADA, 2010).

**Learning Point** Providing residents with a choice of preferred foods and drinks has been found to significantly increase food and fluid intake, particularly in residents who are cognitively intact (Simmons, Alessi et al., 2001).

Medications thought to stimulate appetite are sometimes used to improve food intake and weight status, but their use is controversial. It is a common belief that some older people eat better when they are provided with "small portions." Here a note of caution is warranted. Although the impact of portion size on food intake in older people has not been thoroughly studied, the few studies that do speak to this issue have found that some LTC residents eat *less*, whereas others are unaffected when portion sizes are reduced (Castellanos, Georgian, & Wellman, 2003; Cluskey & Dunton, 1999; Young et al., 2001). That is to say, there is no evidence that smaller portions enhance food intake in this population. This is consistent with a large number of studies in younger people showing that food intake decreases when smaller portions are served (Kral & Rolls, 2004).

Qualified dietitians should be responsible for managing facility-wide dining and snack programs and should optimize menus to meet the unique needs of this population. It is both a necessity and an opportunity for the nutrition professional to take responsibility for all the processes and systems in the facility that support adequate food and fluid intake of residents.

### Cultural Diversity in LTC Facilities

LTC facilities are distinctly different from both acute care and community settings in that they are both residential and institutional. That is to say that the facility is "home" to the client for at least a few weeks, and many residents live out the remaining months or years of their life in this care setting. At the same time the provider, not the individual resident, is in the position to make decisions regarding the nature of the food that is available, the manner of food service, and the timing of resident access to food. It is not difficult to imagine that these factors can have a tremendous impact, both negatively and positively, on the quality of life of the residents.

For example, the availability of culturally acceptable foods can influence a resident's food and nutrient consumption, affecting both health and quality of life. Staple foods are very culture specific, and people from a given culture prefer to eat their particular staple food (e.g., tortillas, bread, rice, beans, pasta) many times a week, if not several times a day. If a resident is not able to regularly obtain the foods that are staples of his or her culture and instead receives the staples of another culture (e.g., an Asian immigrant who receives bread at most meals instead of rice), the menu is unlikely to be acceptable to that individual. Further, the standard of a well-planned menu is determined by the cultural perspective of the evaluator. For example, a menu writer of northern European descent may not think twice that bread is served at every evening meal but would never develop a menu where rice and beans are served at every evening meal. However, this same menu writer may be responsible for a facility with a large number of Hispanic residents. Some Hispanics, depending on their country of origin, would expect to eat rice and beans at almost every noon and evening meal and perceive different beans and legumes (e.g., black, white, red, garbanzo) as entirely different foods and consider beans served on rice as entirely different from beans cooked with rice. In short, planning a single menu for a group of residents from varying cultures can be a challenging task.

To make food provision in the LTC setting more complicated, people reside in facilities because they suffer from a number of acute and chronic diseases such that they cannot be cared for at home. Federal regulations require that food be served in a form and nutrient content to meet individual needs and to support treatment and plan of care. Further, this population is known to be at high risk for deficiencies in energy, protein, fiber, and water, as evidenced by the high prevalence of unintended weight loss, compromised protein status, constipation, and dehydration. Unfortunately, any resident provided a culturally unacceptable menu is unlikely to eat well, which is likely to result in weight loss and undernutrition and a spiral of negative health effects.

Federal regulation specifies the facility's responsibilities toward creating and maintaining an environment that humanizes and individualizes each resident, including how each resident is treated at mealtime. This is consistent with the position of the ADA that nutrition care in LTC facilities must promote quality of life. Because of this, it is the standard of practice for LTC facilities to provide menus that are consistent with regional and cultural norms (e.g., rice and beans frequently may be on the menu in Miami but will be rarely if ever served in Kansas City). Facilities are also adopting new attitudes toward providing care, as described by the ADA (2005). A "resident-centered" or "person-centered" care approach involves residents in decisions about schedules, menus, and dining locations. This approach is one way to honor the relationship between food and culture in the LTC setting while at the same time meeting the intent of the federal statutes.

## Food Preparation

Food preparation and service are critical functions in the LTC facility, yet they often receive insufficient attention from the RD. Tags F362 to F365,

F368, and F369 address various aspects of the proper preparation and service of food, and the dietitian is responsible for ensuring that a facility is in compliance. However, the health and well-being of the residents and the financial viability of the company are both equally important reasons for dietitians to give adequate time and attention to the food service operation.

From both the financial and the resident outcomes points of view, the most expensive meal to a facility is the one that is not consumed. Food and dining are important quality-of-life issues for residents, and food of poor nutritional quality or food that is not consumed in adequate amounts results in a decline in the health and nutritional status of residents. Further, food service operations are resource intensive, and if the food service in an LTC facility is poorly managed, scarce resources available for resident care will be wasted. However timely and thorough a dietitian may be in completing clinical assessments and care plans, if the food leaving the kitchen is not nutritionally adequate or if it is not consumed by residents (because it is unpalatable, not served at an acceptable temperature, not in the proper form, or not served at an acceptable time), then the documentation in the medical record is of little use.

The RD has tremendous opportunities to assist the facility in improving resident care through expertise in staff management, food preparation, food service systems, training skills, and financial management. For example, to verify the use of standardized recipes and instructions and compliance with the menu, the dietitian can periodically observe food preparation. Staff can be reminded or educated in regard to cooking methods that preserve nutritional quality and texture, such as limiting water when heating vegetables and batch cooking. The tray line can also be observed periodically to verify that the menu is being followed for therapeutic diets and that alternates are available. Further, the RD can minimize food waste by making sure that items on the tray line are portioned correctly according to the recipes (e.g., correct pan size and the number of cuts per pan) and the menu (e.g., use of a no. 8 scoop for ½-cup servings). Food safety and palatability are always of paramount importance, and the RD can spot check to verify that tray line temperatures meet both **Hazard Analysis Critical Control Point (HACCP)** process and palatability requirements. Although the DSM and/or the cook should observe a dining room at every meal to determine resident acceptance, the RD should also routinely observe dining to identify any issues with the service of the meal, the dining environment, the provision of feeding assistance, and meal acceptance.

**Hazard Analysis Critical Control Point (HACCP)** A process used in cooking to identify steps and points in food preparation that are deemed as critical in preventing foodborne illness.

### Food Safety and Sanitation

The most common deficiency in LTC surveys is F371: Store, prepare, distribute, and serve food under sanitary conditions. More than 40% of LTC facilities are cited for deficient practice under this tag. Per the federal guidelines, the intent of this regulation is "to prevent the spread of food-borne illness and reduce those practices, which may result in food contamination and compromise food safety in nursing homes. Food- borne illness often is fatal to nursing home residents and can and must be avoided" (AHCA, 2005).

The federal guidelines suggest the use of the U.S. Food and Drug Administration's **Food Code** as a resource, but does not mandate its use. Since the advent of this guideline, several states have adopted the Food Code, and several counties are mandating its use, especially in reference to HACCP. HACCP is a process used in cooking to identify steps and points in food preparation deemed critical in preventing foodborne illness. Although all chapters of the Food Code are relevant, chapters 1 through 4 tend to be the most applicable to LTC settings. The Food Code is one of the most heavily used reference books in the LTC industry, and a copy should be placed in every nutritional services department and should be the foundation for all sanitation training. It should be noted that the Food Code is updated approximately every 2 years, and it is common for a state to adopt a specific version of the Food Code and not recognize newer versions (e.g., a state may use the 2005 Food Code instead of the 2009 version). Thus, it is important for a facility RD to be aware of and follow the version of the Food Code that his or her state uses.

**Food Code** Published by the U.S. Food and Drug Administration, this is a model that assists food control jurisdictions at all levels of government by providing them with a scientifically sound technical and legal basis for regulating the retail and food service segment of the industry.

Because F371 is such a problem in LTC settings, most facility administrators and nursing home companies focus efforts on avoiding this citation. These stakeholders look to the RD to assist them in making sure the facility practices help them achieve

that goal. A study by the RD Council for Quality Nursing Home Care identified the three most common surveyor observations associated with F371 in 95 surveys across nine different LTC companies (Nevins, Gluch, Castellanos, & RD Council for Quality Nursing Home Care, 2003):

- In 45.3% of F371 citations, surveyors observed problems with soiled equipment.
- In 41.1% of F371 citations, surveyors observed problems with food storage. This included a range of problems from food not labeled and dated to improper thawing and cooling of food.
- In 35.8% of F371 citations, surveyors observed problems with the cleanliness and condition of the physical structure of the kitchen. This covers dirty walls and surfaces, broken floor tiles, and holes in screens or gaps in windows.

A dietitian can significantly improve a facility's ability to achieve compliance with F371. On a routine basis the RD should review cooling and thawing practices, educate staff on proper procedures, and conduct spot checks in the kitchen. The RD should regularly inspect equipment sanitation, assist the facility in the use of cleaning schedules, and provide training on the proper use and cleaning of equipment. The RD also should make sure there is an effective system for the fulfillment of maintenance requests and completion of the routine types of cleaning normally performed by facility maintenance (e.g., refrigerator fans). Finally, the RD can review facility policies to make sure they are consistent with the Food Code currently being used by that state.

### Critical Thinking on Nutrition Issues in LTC Facilities

Nursing home regulations are intended to ensure the provision of quality care to all residents. Unfortunately, few of the nutrition regulations or the interpretive guidelines used to assist providers and surveyors are based on clinical studies because there are very few in this population or this care setting. Thus, the standard of practice in LTC often is based on consensus opinion rather than on evidence. Further, when research findings do become available, federal regulations are slow to change and accommodate these advances. When a standard of care is not clearly defined, surveyors are instructed to verify that the facility has developed its own policy and procedures, optimally based on the literature. The survey process then assesses whether the facility is following its own care standards. Although state surveyors assert that they do not mandate ***how*** care is delivered, providers often perceive the federal regulations, interpretive guidelines, and survey process as barriers to making changes in nutrition care delivery. In other words, practitioners often believe they are at increased risk of receiving a citation if they provide care in a manner that varies from what the surveyor is familiar with or expects, and that belief inhibits them from making changes, even improvements, to facility policies and procedures.

An example of a common practice that remains in place simply because it is perceived by providers to be required for compliance with regulations is the recording of meal intake estimates. It is the standard of practice in many nursing homes for nursing assistants to record estimates of meal consumption for each resident at every meal during the entire length of the resident's stay. Maintaining this practice takes a significant amount of time and effort by nursing staff and generates a tremendous amount of paperwork for the facility. It is the mistaken belief of many nurses, dietitians, and others that federal regulation requires estimates of meal intake at every meal. In point of fact, to be in compliance with the regulation the facility must record each resident's intake for several days before the MDS is completed, which is within the 14 days following admission, after a significant change of status, and annually.

Beyond the few days of meal intake estimates required to complete the MDS, the primary reason given for estimating resident food intake is to identify those individuals who are eating poorly. However, there are many reasons to justify the abandonment of daily estimates of meal intake in favor of a more accurate and objective assessment of intake adequacy:

- Meal intake estimates are inherently inaccurate (Castellanos & Andrews, 2002).
- Meal intake estimates do not reflect whether meal intake is sufficient to meet the individual resident's nutritional needs.
- Meal intake estimates divert limited resources from activities proven to be associated with quality care.
- Meal intake estimates constitute a barrier to more home-like meal service options because these options make intake estimates more difficult or impossible to complete.
- Meal intake estimates offer the illusion that there is a valid and reliable system in place to identify those residents who are eating poorly.
- Meal intake estimates provide fodder for lawyers in civil litigation if the record is incomplete or obviously erroneous, which is often.

It has been suggested that practitioners should rely on body weight measures to assess whether food intake is adequate to meet resident energy needs (Castellanos & Andrews, 2002). Body weight change is known to be a valid and reliable way to assess whether individual people are getting adequate nutrition, particularly energy (Gibson, 2005). Amount, rate, and timing of weight loss have also been found to be associated with physiologic impairment and clinical outcomes in hospitalized patients (Gibson, 2005). Further, accurate weighing and documentation are important for defining needs and monitoring MNT success (Splett, Roth-Yousey, & Vogelzang, 2003), and weight loss has been shown to be a valid indicator of quality nutrition care in the LTC setting (Simmons, Garcia et al., 2003). Although body weight change is not a valid measure of energy adequacy in all individuals (i.e., those with edema), if done correctly, it is valid and reliable for the vast majority of residents. Nursing home regulation does require weekly weights on short-term-stay residents covered under Medicare and monthly weights on all others, but most nursing homes do not go beyond these minimums to take full advantage of body weight change as an assessment tool.

In summary, there are instances when the nature of the care provided to nursing home residents is as much about avoiding citations as it is about utilizing the best care process. The practice of recording meal intake estimates is an example of how regulations, perceptions of regulations, or the survey process can become a barrier to improvements in nursing home care. For most residents weekly body weights during the entire length of their stay would be

a valid, reliable, inexpensive, and noninvasive way to identify those who are eating poorly. The availability of frequent weights over time enables both the identification of inadequate food intake and the evaluation of various interventions on weight outcomes. From a purely scientific point of view, utilization of weekly weights as an assessment tool would justify the abandonment of daily meal intake estimates, which are known to be both invalid and unreliable. Yet because daily meal estimates are expected by surveyors, because many RDs and registered nurses are of the mistaken belief that they are required, and because federal regulations do not require weekly body weights on long-term-stay residents, most facilities are reluctant to alter their current practice.

## Survey Management

Numerous times in this chapter we have referenced the regulations the LTC facility must follow when caring for residents. An annual survey to determine the facility's compliance with the regulations occurs a minimum of once every 18 months. Other surveys do occasionally take place, such as a "complaint survey" for review of a complaint made to the state about the facility. A **state survey** team can enter the facility at any time, including the middle of the night or on weekends. Occasionally, a federal survey team, whose job it is to review the state team's results for accuracy, follows them a few days later. At times the federal survey team actually accompanies the state survey team. Thus, there may be anywhere from three to nine surveyors in a facility at one time. The survey teams usually include nurses, sanitarians, and social workers and may occasionally include an RD.

**state survey** The process of the state department of health entering a facility to determine whether the Centers for Medicare and Medicaid Services and state requirements are being met.

The survey experience can be unsettling to both residents and staff. The best way to make this experience a positive one is to plan for the survey in conjunction with the DSM and be prepared to manage the survey. Some steps in planning for and managing a survey are listed in **Table 14.3**. Knowledge is power and confidence. These planning steps, and many others, can be taken to give staff the tools and confidence they need to succeed under pressure. Taking steps to manage the survey helps to keep everyone calm and focused during the actual survey. The most critical thing for the RD to do during the actual survey is to be present when the surveyor is in the kitchen or looking at nutritional issues in the chart. Information or clarification cannot be provided if facility personnel are not there to see and hear the concerns when they are identified.

## Quality Management

In most businesses quality assessment and assurance are optional and are used for business improvement. In LTC settings quality management

**TABLE 14.3 Planning for and Managing the LTC Survey**

| Planning | Managing |
|---|---|
| Educating staff about the federal and state regulations, interpretive guidelines, and investigative protocols. | Identifying the contact person in the nutritional services department to communicate with the surveyor. |
| Educating staff regarding the procedures used by surveyors. This clarifies the expectations of the survey process and how staff should respond in anticipation of the next step in the process. | Determining who will stay with the surveyor assigned to nutritional services throughout the survey process so resources, documentation, justifications/explanations, and so on can be provided as needed. |
| Ensuring systems are in place so that proper meal production and service can be practiced every day. | Responding to potential deficiency areas discovered during the survey using a predetermined approach (e.g., fix any problematic issues immediately). |
| Attending resident council meetings periodically and probing for issues or complaints that residents may bring to surveyors so they can be addressed beforehand. | Holding staff meetings at the beginning and the end of the day to review issues such as surveyor comments, ingredient availability, and staffing. |
| Participating in the facility plan for the surveyor visit, such as determining where the surveyors will be given space to work so as to minimize interference with resident activities and planning what food and beverages will be provided to them so those items can be on hand. | Determine a plan for management to follow-up on potential issues mentioned by surveyors. |

is required by the federal government (F520 and F521). Further, Section 6102(c) of the Affordable Care Act requires that all nursing homes develop **Quality Assurance Performance Improvement (QAPI) programs**, with CMS charged to develop a prototype by the end of 2011. As a result of this charge, CMS has established a 2-year national demonstration project in California, Florida, Massachusetts, and Minnesota to evaluate best practices and test the CMS technical assistance approaches. A final rule is expected sometime in 2013, depending on funding.

**Quality Assurance Performance Improvement (QAPI) programs** This is a type of quality assurance program that is mandated in nursing homes by the Affordable Care Act.

Dietitians in the LTC setting play a critical role in ensuring that the facility meets quality care standards in both clinical care and meal production. Well-designed systems must be in place to ensure appropriate and timely nutrition assessment of residents, execution of the nutrition care plan, minimal weight loss and pressure ulcers, proper sanitation, food preparation, and delivery of the meals. QAPI activities are necessary to ensure the delivery of high-quality nutritional services.

The essential steps of QAPI (1) use performance indicators to monitor a wide range of care processes and outcomes over time; this includes tracking, investigating, and monitoring adverse events; (2) set a performance standard; (3) gather individual feedback and identify deficiencies; (4) review systems and processes to determine the root cause of the issue (tools such as a fishbone diagram can be used in this review); (5) initiate performance improvement plans (PIP) to achieve performance standards; and (6) monitor progress using the "Plan, Do, Study, Act" (PDSA) method (see http://www.cms.gov).

Step 4 of the QAPI process is critical for ensuring that quality care is being delivered and that the activities required to maintain quality are being performed at an acceptable level. The efficacy of the QAPI committee depends on its ability to evaluate the systems and processes in a facility. Although QAPI may not be a responsibility of the dietitian in other care settings, the nature of the LTC setting demands that dietitians use a wide range of management skills to ensure that residents receive quality care during the entire length of their stay. Although the dietitian may not be in attendance for all QAPI meetings (e.g., if the facility RD is a consultant), the RD should review the minutes of QAPI meetings and assist the DSM in the system analysis.

Quality management also is part of the nursing home survey process. State and federal regulators use facility data from the MDS to monitor various **quality indicators**. The MDS Quality Indicator Report for Nutrition/Eating includes "prevalence of weight loss," "prevalence of tube feeding," and "prevalence of dehydration."

**quality indicators** A quality indicator report is generated from a facility-wide Minimum Data Set to be used by surveyors during the survey process. The quality indicator report is used to help surveyors identify potential quality problems in the facility. The quality indicator report for nutrition/eating includes "prevalence of weight loss," "prevalence of tube feeding," and "prevalence of dehydration."

**quality measures** Selected Minimum Data Set made available to the public by the Centers for Medicare and Medicaid Services at the Nursing Home Compare website. These data are intended to represent the quality of care provided at nursing facilities so consumers can make an informed decision. Prevalence of weight loss is one of the quality measures from each nursing home that the Centers for Medicare and Medicaid Services shares with the public.

The Centers for Medicare and Medicaid Services has made certain data available to the public so that consumers can use it to compare nursing homes. Selected MDS data from each facility are available nationally at the Nursing Home Compare website (see http://www.medicare.gov/nursinghomecompare/search.html). These data, called **quality measures**, are intended to represent the quality of care provided at nursing facilities so consumers can make an informed decision. Of particular relevance to nutrition services is the quality measure "Percent of Long-Stay Residents Who Lose Too Much Weight" (MDS 3.0 QM Users Manual). The quality measure reports the percentage of residents with a significant weight loss, excluding residents receiving hospice care; that is, residents who have experienced a weight loss of more than 5% of their body weight in 1 month or 10% of their body weight in 6 months.

It is appropriate that the facility RD take the lead in efforts to improve the facility's weight loss quality measure. The etiology of unintentional weight loss and undernutrition is multifactorial and is likely related to both psychological factors and physiologic changes (Morley, 2003). Studies have suggested that behavioral and environmental factors may be the most important determinants of food intake in the nursing home setting (Simmons, Alessi et al., 2001; Simmons, Babinou, Garcia, & Schnelle, 2002). It is critical that nutrition care processes focus on underlying causes and emphasize prevention of weight loss (Beck & Ovesen, 1998).

## Unique Role of the Consultant Dietitian in LTC

*Consultant*, as defined by *Webster's*, is a person who is called on for professional or technical advice or opinions. RD consultants are in a unique position because they not only have the clinical training necessary to provide MNT, they also are highly valued for their knowledge in the areas of food preparation, sanitation, cost management, and food service systems. If the facility RD is a consultant and not a full-time employee of the facility or company, it is important to remember that the resident is not the only customer. The clients of the consultant RD also include the facility administrator, the director of nursing services, and the DSM.

Consultants are in an LTC facility a limited number of hours per week or, in some cases, a limited number of hours per month. If the activities of a consultant are limited only to charting and pointing out sanitation or production errors, then the facility is not able to take full advantage of the expertise of the consultant in helping it to develop in-house staff and improve facility performance over time. On the other hand, if the consultant takes the time to identify and understand the current issues or concerns of the facility, he or she may be able to provide relevant staff education and/or offer several possible solutions to ongoing problems. This approach allows the facility team the opportunity to develop their own competency in the area of concern and affords staff the opportunity to choose and take ownership of the solution. Staff development and buy-in are essential for long-term correction of any problem.

An example consultant report is as follows: Cross-contamination between clean and dirty dishes in the dish room observed. An alternative consultant report that provides more information and solutions follows: At entrance conference facility expressed concern regarding sanitation. Sanitation tour completed, see attached. Provided in-service to staff on dish machine procedures. Recommend two options for change: (1) Move the hand sink closer to the dish area as discussed and/or (2) Restructure staffing to allow for one associate on each end of the dish machine, thus eliminating need to crossover from clean to dirty side.

## Summary

In summary, the role for the RD in LTC settings is much more complex than it is in many other care settings. In LTC the dietitian is expected to be proficient in MNT and have expertise in the regulatory environment, food preparation, sanitation, dining, and survey and quality management. The provision of nutrition care in this setting also requires a proactive and system-oriented approach, where residents' quality of life is a primary consideration in every decision, and care should be provided in the most home-like manner possible. The potential for the RD to make a significant difference in the quality of life of residents and to be valued by the facility are limited only by the aspirations of the individual practitioner.

## Case Study

### Increasing Appetite in Long-Term Care

Cynthia Chandler, RD, LD, CDM

Edith is a 90-year-old resident of a long-term care facility and has recently lost 12% of her body weight. Upon observation she is sleeping late and missing breakfast, not coming to the dining room for lunch, and struggling to feed herself at the evening meal, resulting in decreased oral intake. A dietitian consultation was arranged.

*Concerns:* Why is Edith sleeping so late? Is Edith able to feed herself lunch in her room? Who is Edith sitting with at dinner? Is Edith comfortable accepting assistance with feeding? Is Edith being provided foods that are "finger foods" that she can handle?

*Remarkable Findings:*

Blood glucose level random tests running 60 mg/dl at 1 pm and 72 mg/dl at 8 pm on two consecutive days
Weight: 92 pounds
Height: 5 foot 2 inches
Ideal body weight: 110 lbs (+/– 10%)
Family situation: Daughter is deceased; widow; has a cousin that visits once a month.
*Assessment:* Isolated, frail, elderly female who is becoming more reclusive and losing appetite. Blood glucose levels running lower than normal. Upon interview, Edith states food does not taste good, and it is cold by the time she is able to eat. She states that she has no appetite for the foods she is served. Edith complains of constipation and lack of energy and is embarrassed to have to be fed and wishes she could take more control of her feeding.

**Question**

1. What is a good plan that the RD can make for Edith?

## Issues to Debate

1. Does the intense regulation of LTC facilities negatively or positively affect resident care?
2. Should LTC facilities be required to have a full-time RD on staff or is the non-specificity of the current regulation acceptable (i.e., the facility must have adequate personnel to provide dietary services and a qualified dietitian is utilized in planning, managing, and implementing dietary service activities).
3. What aspects of the role of the RD in LTC would be the most challenging for an entry-level dietitian?

## References


American Dietetic Association (ADA). (2005). Position of the American Dietetic Association: Liberalization of the diet prescription improves quality of life for older adults in long-term care. *Journal of the American Dietetic Association, 105*, 1955–1965.

American Dietetic Association. (2010). Position of the American Dietetic Association: Individualized nutrition approaches for older adults in health care communities. *Journal of the American Dietetic Association, 110*, 1554–1563.

American Health Care Association. (2005). *The Long-Term Care Survey.* Washington, DC: Author.

American Health Care Association. (2011). The state of long-term health care sector. Characteristics, utilization, and government funding. Retrieved from www.ahcancal.org/research_data/trends_statistics

American Health Care Association. (2013). *The long term care survey*. Washington, DC: Author.

American Medical Directors Association. (2002). *Clinical practice guidelines: Dehydration and fluid maintenance*. Columbia, MD: Author.

Barton, A. D., Beigg, C. L., Macdonald, I. A., & Allison, S. P. (2000). A recipe for improving food intakes in elderly hospitalized patients. *Clinical Nutrition, 19*, 451–454.

Beck, A. M., & Ovesen, L. (1998). At which body mass index and degree of weight loss should hospitalized elderly patients be considered at nutritional risk? *Clinical Nutrition, 17*, 195–198.

Blaum, C. S., O'Neill, E. F., Clements, K. M., Fries, B. E., & Fiatarone, M. A. (1997). Validity of the minimum data set for assessing nutritional status in nursing home residents. *American Journal of Clinical Nutrition, 66*, 787–794.

Castellanos, V. H. (2004). Food and nutrition in nursing homes. *Generations, 28*, 65–71.

Castellanos, V. H., & Andrews, Y. N. (2002). Inherent flaws in a method commonly used to report food intake in long term care facilities. *Journal of the American Dietetic Association, 102*, 826–830.

Castellanos, V. H., Georgian, M. E., & Wellman, N. S. (2003). Fluid intake increased when larger portions were offered to nursing home residents with medications. *FASEB Journal, 17*, A285.

Castellanos, V. H., Silver, H. J., Gallagher-Allred, C., & Smith, T. (2003). Nutrition issues in home, community, and long-term care setting. *Nutrition Clinical Practice, 18*, 21–36.

Castellanos, V. H., Surloff, S. C., & Giordan, W. L. (2003). Systematic enhancement of food over short term increased calorie intake by residents with dementia. *Journal of the American Dietetic Association, 103*, A40.

Chapman, K., Samman, S., & Liburne, A. M. (1993). Are the guidelines for nutritional care and food service in nursing homes being implemented? *Australian Journal of Nutrition and Diet, 50*, 39–45.

Chidester, C., & Spangler, A. A. (1997). Fluid intake in the institutionalized elderly. *Journal of the American Dietetic Association, 97*, 23–28.

Cluskey, M., & Dunton, N. (1999). Serving meals of reduced portion size did not improve appetite among elderly in a personal-care section of a long-term-care community. *Journal of the American Dietetic Association, 99*, 33–35.

Cluskey, M., & Kim, Y. (1997). Use and perceived effectiveness of strategies for enhancing food and nutrient intakes among elderly persons in long-term care. *Journal of the American Dietetic Association, 101*, 111–114.

de Jong, N., Chin-A-Paw, M. J., de Graff, C., de Groot, L., & van Staveren, W. A. (2001). Appraisal of 4 months' consumption of nutrient-dense foods within the daily feeding pattern of frail elderly. *Journal of Aging and Health, 13*, 200–216.

ElderWeb. (n.d.). Average length of nursing home stay. Retrieved from www.elderweb.com/article/average-length-nursing-home-stay

Endres, J., Welch, P., Ashraf, H., Banz, W., & Gower, E. (2000). Acceptance of soy foods by the elderly in a long-term care facility. *Journal of Nutrition in the Elderly, 19*, 1–17.

Gibson, R. S. (2005). *Principles of nutritional assessment* (2nd ed.). New York, NY: Oxford University Press.

Institute of Medicine. (1997). *Dietary Reference Intakes for calcium, phosphorus, magnesium, vitamin d, and fluoride*. Washington, DC: National Academies Press.

Johnson, L. E., Dooley, P. A., & Gleick, J. G. (1993). Oral nutritional supplement use in elderly nursing home patients. *Journal of the American Geriatrics Society, 41*, 947–952.

Kayser-Jones, J., Schell, E. S., Porter, C., Barbaccia, J. C., & Shaw, H. (1999). Factors contributing to dehydration in nursing homes: Inadequate staffing and lack of professional supervision. *Journal of the American Geriatrics Society, 47*, 1187–1193.

Kral, T. V., & Rolls, B. J. (2004). Energy density and portion size: Their independent and combined effects on energy intake. *Physiology & Behavior, 82*, 131–138.

Lacey, K., & Pritchett, E. (2003). Nutrition care process and model: ADA adopts road map to quality care and outcomes management. *Journal of the American Dietetic Association, 103*, 1061–1072.

Morley, J. E. (2003). Anorexia and weight loss in older persons. *Journal of Gerontology, 58A*, 131–137.

Nevins, D. A., Gluch, L. A., Castellanos, V. H., & RD Council for Quality Nursing Home Care. (2003). Most common causative factors for F371 deficiencies in long-term care state surveys. *Journal of the American Dietetic Association, 103*, A42.

Olin, A. O., Armyr, I., Soop, M., Jerstrom, S., Classon, I., Cederholm, T., … Ljungqvist, O. (2003). Energy-dense meals improve energy intake in elderly residents in a nursing home. *Clinical Nutrition, 22*, 125–131.

Olin, A. O., Osterberg, P., Hadell, K., Armyr, I., Jerstrom, S., & Ljungqvist, O. (1996). Energy-enriched hospital food to improve energy intake in elderly patients. *Journal of Parenteral and Enteral Nutrition, 20*, 93–97.

Pioneer Network Food and Dining Clinical Standards Task Force. (2011). New dining practice standards. Retrieved from www.pioneernetwork.net/Providers/DiningPracticeStandards

Rahman, A. N., & Simmons, S. F. (2005). Individualizing nutritional care with between-meal snacks for nursing home residents. *Journal of the American Medical Directors Association, 6*, 215–218.

Rolls, B. J., & Phillips, P. A. (1990). Aging and disturbances of thirst and fluid balance. *Nutrition Reviews, 48*, 137–143.

Ross, F. (1999). An audit of nutritional supplement distribution and consumption on a care of the elderly ward. *Journal of the Human Nutritional Diet, 12*, 445–452.

Shatenstein, B., & Ferland, G. (2000). Absence of nutritional or clinical consequences of decentralized bulk food portioning in elderly nursing home residents with dementia in Montreal. *Journal of the American Dietetic Association, 100*, 1354–1360.

Simmons, S. F., Alessi, C., & Schnelle, J. F. (2001). An intervention to increase fluid intake in nursing home residents: Prompting and preference compliance. *Journal of the American Geriatrics Society, 49*, 926–934.

Simmons, S. F., Babinou, S., Garcia, E., & Schnelle, J. F. (2002). Quality assessment in nursing homes by systematic direct observations: Feeding assistance. *Journal of Gerontology. Series A, Biological Sciences and Medical Sciences, 57*, M665–M671.

Simmons, S. F., Garcia, E. T., Cadogan, M. P., Al-Samarrai, N. R., Levy-Storms, L. F., Osterweil, D., & Schnelle, J. F. (2003). The minimum data set weight-loss quality indicator: Does it reflect differences in care processes related to weight loss? *Journal of the American Geriatrics Society, 51*, 1410–1418.

Simmons, S. F., Lam, H. Y., Rao, G., & Schnelle, J. F. (2003). Family members' preferences for nutrition interventions to improve nursing home residents' oral food and fluid intake. *Journal of the American Geriatrics Society, 51*, 69–74.

Simmons, S. F., Osterweil, D., & Schnelle, J. F. (2001). Improving food intake in nursing home residents with feeding assistance. A staffing analysis. *Journal of Gerontology. Series A, Biological Sciences and Medical Sciences, 56*, M790–M794.

Simmons, S. F., & Reuben, D. (2000). Nutritional intake monitoring for nursing home residents: A comparison of staff documentation, direct observation, and photography methods. *Journal of the American Geriatrics Society, 48*, 209–213.

Simmons, S. F., & Schnelle, J. F. (2004). Individualized feeding assistance care for nursing home residents: Staffing requirements to implement two interventions. *Journal of Gerontology. Series A, Biological Sciences and Medical Sciences, 59A*, 966–973.

Splett, P. L., Roth-Yousey, L. L., & Vogelzang, J. L. (2003). Medical nutrition therapy for the prevention and treatment of unintentional weight loss in residential healthcare facilities. *Journal of the American Dietetic Association, 103*, 352–362.

Wilson, M.-M. G., Purushothaman, R., & Morley, J. E. (2002). Effect of liquid dietary supplements on energy intake in the elderly. *American Journal of Clinical Nutrition, 75*, 944–947.

Young, K. W., Binns, M. A., & Greenwood, C. E. (2001). Meal delivery practices do not meet needs of Alzheimer patients with increased cognitive and behavioral difficulties in a long-term care facility. *Journal of Gerontology. Series A, Biological Sciences and Medical Sciences, 56*, M656–M661.

CHAPTER 15

# Special Topics in Nutrition and Ethics: Feeding and Ethical Issues at the End of Life

*Judith Sharlin, PhD, RD, I. David Todres, MD, and Julie O'Sullivan Maillet, PhD, RD*



## CHAPTER OUTLINE

## Reader Objectives

**After studying this chapter and reflecting on the contents, you should be able to**

1. Address the appropriate uses and methods for artificial nutrition and hydration and the role of the registered dietitian at the end of life.
2. Describe the benefits, risks, and burdens associated with artificial nutrition and hydration.
3. Apply an ethical framework for decision making regarding the appropriateness of artificial nutrition and hydration.
4. Explain the legal decisions of two seminal cases.
5. Summarize the use of advance directives and how they affect decision making in health care.

## Artificial Nutrition and Hydration: Definition and Indications

Artificial nutrition and hydration at the end of life are considered legally and by healthcare professionals to be medical interventions (American Dietetic Association [AND], 2013ab). It is the patient's articulated desires for extent of medical care that should be the driving force for determining the level of nutrition intervention (AND, 2013ab). This is particularly true for artificial nutrition and hydration (ANH), which is defined as nutrition and hydration provided through a tube into the gastrointestinal tract or parenterally via intravenous feeding (Geppert, Andrews, & Druyan, 2010). Tube feeding is used for patients with a functioning gastrointestinal tract who cannot eat or who are unable to swallow. Tube feeding can include nasoenteric, gastrostomy, and jejunostomy tubes. Since medical advancements in the 1980s, percutaneous endoscopic gastrostomy (PEG) tubes have become the method of choice for long-term tube feeding, but not short-term feeding because of operative risks (Geppert et al., 2010). Parenteral feeding may be as an intravenous feeding in the arm or through a catheter in the chest. Parenteral feeding is used when insufficient nutrients can be digested or absorbed within the gastrointestinal tract. The concept of "when in doubt, feed" is applicable to most individuals. Feeding should start when the patient is medically stable and only be stopped if the feeding is no longer beneficial to the patient (AND, 2013ab). ANH has a major role in gastrointestinal diseases, after acute trauma, with chronic disabilities, and when the interprofessional team is unsure whether the disorder is reversible. However, ANH cannot cure or reverse terminal disorders (Fine, 2006). This chapter focuses on feeding at the end of life.

### Evidence about Long-Term Tube Feeding

The benefits to the use of the PEG tube in patients with certain diseases include that these tubes may prolong life through adequate nutrition and may help alleviate the discomfort of symptoms and enhance quality of life (Ganzini, 2006). PEG feeding tubes extend life by improving nutrition, lessening dehydration, reducing aspiration, and helping to heal pressure sores. Tube feeding is less invasive than parenteral nutrition. In addition, tube feeding has fewer complications and is less costly than parenteral nutrition (Hurley & McMahon, 2005; McMahon, 2004). Long-term tube feeding, like long-term parenteral feeding, allows many to live an active life. However, tube feeding in individuals with terminal illnesses does not seem to have the same value. More education is needed among healthcare professionals and families to share the evidence to allow better decision making.

In reviewing studies from 1966 through 1999 on tube feeding patients with advanced dementia, none of them showed any of the benefits to the patient: prolonged life, reduced risk of pressure sores or infections, prevention of aspiration pneumonia, or improved palliative care (Finucane, Christmas, & Travis, 1999). In addition, aspiration occurs in 25% to 40% of patients who are receiving tube feeding (McClave & Chang, 2003). A lack of evidence shows survival benefits with the PEG tube in patients suffering from terminal diseases, including dementia, Alzheimer's disease, Parkinson's disease, and vascular disease (American Society of Parenteral and Enteral Nutrition Task Force, 2010).

Feeding tubes are often placed when transferring patients from acute care to long-term care facilities to promote adequate nutrition. This even is the law in some states. Many healthcare providers now recommend slow hand feeding as an alternative (DeLegge, 2009). Although it is more labor intensive to feed patients by hand (Mitchell, Buchanan, Littlehale, & Hamel, 2003), it is more nurturing for patients. Hand feeding may provide patients with appropriate social and sensory stimulation that enhances quality of life. The Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition position papers on feeding support the use of oral consumption through hand feeding for patients with severe dementia (AND, 2013ab; American Society of Parenteral and Enteral Nutrition Task Force, 2010).

### Dehydration

There is a current debate whether terminally ill patients facing imminent death benefit from dehydration or hydration. Several small studies in the 1990s suggest that most terminally ill patients do not experience significant hunger and thirst. One study of cancer patients showed that more than two-thirds complain of thirst or dry mouth at the end of life (Huang & Ahronheim, 2000). If they say they are hungry or thirsty when the healthcare provider asks, patients often want specific foods, often comfort foods and simple oral care (Fine, 2006). Contrary to healthy individuals, thirst in advanced cancer patients appears to be unrelated to dehydration and is not relieved by fluids (Fine, 2006). Several studies in early 2006 showed that in terminally ill and advanced dementia patients quality of life was better for the patients without any form of ANH (Fine, 2006).

Dehydration causes a decrease in secretions, urinary output, edema, and ascites and increases an endogenous dynorphin that may act as a potent opiate, which may calm the patient (Printz, 1992). Fluid overload from artificial hydration increases pulmonary and gastrointestinal secretions, urinary output, and pharyngeal secretions that could lead to the choking and gurgling sounds known as the "death rattle" (Gallagher, 1989). A study showed increased peripheral edema, ascites, and pleural effusions with modest hydration and no difference in bronchial secretion or delirium (Morita et al., 2005). Most healthcare professionals in palliative care promote the use of good mouth care, sips of water, and ice chips, when desired by patients (Ersek, 2003). At the end of life, a decrease in food and fluid intake seems to be part of the natural physiology of dying (Fine, 2006). The wishes of the patient should guide the process with the health professional explaining the options.

## Meaning of Food and Drink

In patient care both food and drink have psychological and physiologic functions as well as many social meanings. Giving food and drink encompasses strong cultural and emotional values that are often equated with nurturing and caring. Providing nutrition and hydration is, however, not synonymous with eating or with feeding someone. Normally, people eat through the mouth with utensils. A medical intervention such as ANH is not considered socially normative (Slomka, 2003).

In the position and practice papers of the Academy of Nutrition and Dietetics (2013ab) concerning ethical and legal issues in nutrition, hydration, and feeding, the following guidelines were proposed for oral intake (nutrition and hydration):

1. Oral feeding should be advocated whenever possible.
2. The patient's physical and emotional enjoyment of food should be the primary consideration. Staff and family members should be encouraged to assist in feeding the patient.
3. Nutrition supplements should be used to encourage oral intake and alleviate painful symptoms associated with hunger, thirst, or malnutrition.
4. Diet restrictions should be reevaluated.
5. The patient has the right to determine whether he or she wants to consume foods outside of the "diet prescription."
6. Suboptimal oral feedings may be more appropriate than tube or parenteral feeding.

When ANH is being considered, it is important for healthcare providers to know the values and beliefs of the patient. The many cultural and religious beliefs and values about end-of-life care and feeding at the end of life cannot be overlooked by healthcare providers and must be respected (AND, 2013ab). The Academy of Nutrition and Dietetics/Commission on Dietetic Registration (2009) *Code of Ethics* should be used as a framework for all practice including obligations of the practitioner

when communicating with patients and their families (Gallagher-Allred, 2012).

## Roles of the Registered Dietitian

Registered dietitians are in a unique position, because of their education and background, to work collaboratively with the healthcare team to make nutrition, hydration, and feeding recommendations. Registered dietitians must be aware of the organization's policies and state laws regarding feeding. Registered dietitians are trained to assess patients' nutrition needs, offer nutrition counseling, and provide meals that reduce side effects of medicine and treatment. Registered dietitians also can encourage patients and families to suggest or supply their own favorite meals, if appropriate, and to assist in securing hand feeding of patients. Often, a registered dietitian's familiarity with practical aspects of food and fluid at the end of life is helpful in addressing families' concerns and questions about chewing, swallowing, and the cessation of thirst and hunger.

Registered dietitians may be able to express best what is wanted by the patient because they are the patient's and family's main conversation partner about feeding issues (AND, 2013ab). They are in a position to contribute the most accurate information from a nutrition perspective on what is warranted. Registered dietitians are trained in medical nutrition therapy and teaching and are responsible for keeping the patient's options for and outcomes of feeding at the center of care deliberations. The registered dietitian is responsible for ensuring that the care team considers all options about feeding rather than assuming that one strategy is obligatory. For example, a situation might occur with a patient where tube feeding seems to be the only choice, when, in fact, careful hand feeding may be a better alternative to provide adequate nutrition. Finally, the dietitian's role, along with the rest of the interprofessional healthcare team, is to provide a compassionate and positive experience for the patient, family, and loved ones.

## Ethical Principles as a Framework for Decision Making

Ensuring that the patient is given food and water is an important symbolic nurturing act that is deeply rooted in cultural and religious traditions. However, in some circumstances it may be morally correct to consider providing ANH inappropriate. This consideration raises strong emotional reactions and leads to anguished decision making for patients, families, and healthcare workers. Appreciating the ethical principles underlying these decisions is helpful in ensuring that the patient's care rests on a strong moral foundation (Casarett, Kapo, & Caplan, 2005; McMahon, Hurley, Kamath, & Mueller, 2005; Mueller, Hook, & Fleming, 2004).

Healthcare providers are ethically obliged to protect life and relieve suffering. Several moral principles guide providers: respect for the individual's desire/choices (autonomy), doing no harm to the person (nonmaleficence), doing what is best for the individual (beneficence), and being fair to any individual in a similar situation (justice). These principles provide a basis for patients to trust their healthcare providers. The framework for decision making includes the provider knowing what the patient's wants, knowing what the evidence says is warranted, and sharing decision making about the appropriate course of action (AND, 2013ab).

## Legal Guidance

Ethical principles may conflict with each other depending on the healthcare team's goals of maintaining life yet not prolonging death and suffering. Health professionals protect life but also must respect a patient's rights of choice and autonomy over his or her own body, such as when the patient chooses to refuse treatment that would prolong life. These and other ethical conflicts are generally resolved through discussions with all concerned or may need to be reviewed by an ethics committee. Severe conflicts may be brought to court; such legal cases are described next.

### The Schiavo and Lee Cases

To understand the ethical issues involved in long-term tube feeding, it is helpful to review the cases of Schiavo and Lee. The Schiavo case highlighted the complex interactions of medicine, ethics, law, and family dynamics in deciding on the appropriateness of ANH for Terri Schiavo. The patient had suffered a cardiac arrest in 1990 as a result of an electrolyte imbalance precipitated by an eating disorder. ANH through a feeding tube was provided for the patient. A struggle that was fought out in the courts and the public media resulted when the patient's husband declared after 15 years that he wished to discontinue ANH and the patient's parents and

siblings insisted that ANH be continued (Blendon, Benson, & Herrmann, 2005; Koch, 2005).

The facts of the case demonstrated that Schiavo was in a permanent vegetative state (PVS), a state in which there is no higher cortical functioning and therefore no cognition. Initially, PVS is considered a persistent state, and after a period of 6 months to 3 years with no change this state is then considered permanent. Multiple neurologic examinations attested to Schiavo's PVS. This is an important point because the facts have to be clear and consistent for a successful ethical discussion. The parents did not believe the results of the tests and they saw improvement in their daughter's condition. Schiavo was not in a position to state her own wishes—the principle of autonomy rooted in the precept of respect for the person—but her wishes would have been known had she left an advance directive regarding her future care. Because no advance directive was left, the principle of **substituted judgment** was applied through statements by her closest family. Legally, the designated decision maker was Terri Schiavo's husband, who requested withdrawal of the tube feeding (Blendon et al., 2005; Koch, 2005).

**substituted judgment** A legal term used to describe what a person would decide if he or she were able to decide; used when the individual is not competent to provide an autonomous decision.

When the patient's wishes cannot be ascertained (e.g., an infant or one who has always been incompetent to choose rationally), then treatment decisions are based on the patient's best interests, that is, what a reasonable person would most likely want in the same circumstances (Annas, 2004). Schiavo's parents and siblings did not accept the diagnosis of PVS and believed the condition would improve with further rehabilitative care. The parents strongly believed that withdrawal of the feeding tube would violate their daughter's Catholic belief and be tantamount to euthanasia. This belief that the withdrawal of feeding would be morally wrong had the support of an allocution by the late Pope John Paul II, who stated that for patients in PVS, "the administration of water and food even when provided by artificial means of preserving life, [is] not a medical act . . . and as such is morally obligatory" (Pope John Paul II, 2004). According to Paris (2005), this interpretation of the Catholic position on the sanctity of life is not consistent with traditional church teaching, and Paris cites the 2001 directive of the U.S. Catholic bishops that states "A person may forgo means that in the patient's judgment do not offer a reasonable hope or benefit and entail an excessive burden" (Paris, 2005).

There were multiple court hearings and repeated episodes of tube withdrawal and replacement. The case was also politicized until finally it involved the president of the United States. However, in the end the judicial court ruling of withdrawal of the tube was carried out (Quill, 2005). Schiavo died a few days after the withdrawal of tube feeding.

Surveys of the attitudes of the American public regarding the Terri Schiavo case were carried out (Blendon et al., 2005). Twelve national opinion surveys showed that most Americans opposed efforts by elected politicians to intervene in the Schiavo case. However, the public was more divided on the question of whether Schiavo's feeding tube should have been removed. A substantial minority, 24% to 42%, opposed it. Of interest too is that one-third of respondents believed she could experience pain and discomfort when the feeding tube was withdrawn.

In a 2012 case in Queens, New York, a terminally ill paralyzed patient, SungEun Grace Lee, won a court case to stop her breathing tube. Her parents argued she was being pressured by the doctors to make this decision and it was against her religion. After the court decision in her favor, Lee had a change of heart and wanted to continue with the breathing tube to make peace with God and her parents (Hartocollis, 2012). This case illustrates the complexity of individual versus family decision making, the role of religion, and the importance of healthcare providers communicating well.

### The Quinlan and Cruzan Cases

The 1976 case of Karen Ann Quinlan set the precedent of the right to terminate medical treatment when the Supreme Court allowed Quinlan's parents to turn off life support. They chose to turn off the respirator but continued the tube feeding (*In re Quinlan*, 1976).

The 1990 Nancy Cruzan case set the legal precedent for withdrawal of tube feeding (*In re Cruzan v. Director*, 1990). Cruzan was diagnosed as in a PVS. The family wanted to discontinue feeding. The hospital employees refused. Her parents brought the issue of withdrawal of tube feeding to the courts. The Missouri Supreme Court reversed a lower court decision to stop feeding because it felt that the state's obligation to preserve life superseded the right to self-determination because there was insufficient evidence of what Cruzan wanted. The Supreme Court heard the case and had two major decisions. The Court affirmed the right of the

state to determine its own definition of "clear and convincing" evidence, ruled that ANH was medical treatment and could be withheld, and declared that, as medical treatment, ANH could be stopped. Six months after the ruling, three new witnesses came forward and the tube feeding was discontinued.

### Advance Directives

The Nancy Cruzan case stimulated the public to consider that individuals could convey personal wishes regarding their care. The Patient Self-Determination Act took effect in 1991 and requires Medicare/Medicaid patients to be informed of their right to advance directives or assignments of durable power of attorney. The advance directive may take the form of a "living will" or designation of a healthcare proxy (AND, 2013ab; Gillick, 2004). By 2000, every state had passed some form of living will or healthcare proxy law.

The optimal way in which a healthcare proxy advance directive is implemented is for the patient to designate one person to act on his or her behalf to make treatment decisions if the patient is unable or incompetent to state wishes him- or herself (Annas, 2004). This is done in writing using a healthcare proxy form. It is also important for the patient to inform the designated person of how he or she wants to be treated in specific situations; for example, "do not wish to be tube fed." Doing this verbally is acceptable, or it can be written in a letter to reduce the chance of a Shiavo-type conflict.

A living will allows patients to clearly outline their wishes but may not cover all circumstances and may not be available to the healthcare team. However, with a trusted healthcare provider who is made aware of the individual's goals of care and values, the right decision can be made that is in the person's best interests. Despite the ethical advantages of an **advance directive**, however, only about one-fifth of the public has one in place (Salmond & David, 2005). A developing improvement in making patients' needs known is the Physician Orders for Life Sustaining Treatment (POLST) form. Fifteen states have authorized the use of this one-page form that is filled out by the physician and usually signed by the patient or surrogate. The form becomes part of the healthcare record and follows the patient from site to site ("Care at the End of Life," 2012).

**advance directive** Information in written or oral form provided by the patient that outlines the competent adult's wishes regarding medical treatment should he or she become incompetent in the future.

## Withdrawing or Withholding Treatment

Many believe it is more acceptable to withhold a treatment than to withdraw it. This distinction is not supported by currently accepted ethical and legal reasoning (Meisel & Cerminara, 2004).

With regard to withholding or withdrawing ANH from terminally ill or permanently unconscious patients, the Supreme Court ruled that administration of ANH is like any other medical treatment and may be withdrawn if the patient refuses the treatment or, in the case of the incapacitated patient, the appropriate standard of evidence is met. Despite the fact that withdrawing feeding is consider equal or morally better because it has been tried and then stopped if futile, many cultures and religions believe that once started feeding may not be stopped. The question is whether the feeding is morally optional or morally obligatory, and the answer varies by individual person (AND, 2013ab).

Of concern in the act of withdrawing tube feeding is the moral distinction between killing and letting die. It is legally and ethically acceptable to allow a patient to die when the burdens and risks of life-prolonging treatment clearly outweigh their benefits. Legally, an act of killing is murder if it is intended and it is prohibited in our culture. Cause of death can be ambiguous when a feeding tube is withdrawn. Some argue that withdrawal of the tube is the cause of death. However, death is caused by the underlying failure of an organ system; the fundamental cause of death is the patient's condition, not the withdrawal of treatment. The life support has on a temporary basis postponed the death. If there is no reasonable hope of recovery, further life-sustaining treatment cannot benefit the patient, and therefore it is not in the patient's interests to continue it. When it is removed, the body's own causality results in the death (Fine, 2006).

## Communication

Effective communication often helps to prevent ethical dilemmas. In communicating with the patient, the healthcare team needs to learn the patient's values, goals, and beliefs. It is also very important that the healthcare team spell out the prognosis and facts associated with the patient's condition and that they are mutually understood by all. The process of informed consent should be carefully carried out,

not rushed, and with a mutual understanding of the patient's goals of care (Brett & Rosenberg, 2001). Deep-rooted cultural and religious beliefs affect the patient's approach to the consideration of the appropriateness of long-term tube feeding (AND, 2013ab; American Society of Parenteral and Enteral Nutrition Task Force, 2010). Some patients, and especially families, consider removal of tube feeding unethical because it is a state of "starvation" (Pope John Paul II, 2004). The moral decision is the patient's decision.

Patients' families at times believe that lack of ANH causes suffering and will demand that "everything be done" to avoid this, including placement of PEG for long-term tube feeding. However, clinicians are not obligated to provide futile treatment (Snyder, Leffler, & the Ethics and Human Rights Committee, American College of Physicians, 2005). It is necessary for the healthcare team to define clearly what is meant by *futile*. This judgment must take into account the goals of care, including respecting beliefs, the proposed treatment, and the outcome, so that providers and patients are certain the goals cannot be achieved with the proposed treatment.

Presenting the issue of ANH to the patient and family requires compassion and appropriate choice of words that allow clear communication. Clinicians caring for amyotrophic lateral sclerosis patients provided examples from their clinical experience. The clinicians explained that PEG had risks, such as infection at the site of the tube, disfigurement of body shape, and diarrhea or constipation that may not always be transient. They also explained that tube feeding was cumbersome and with loss of hand movement required an assistant at every meal. The clinicians told patients that to decide against tube feeding was an option. Many patients stated they appreciated the information to make an informed choice (Mitsumoto & Rabkin, 2007).

Many healthcare providers and patients and families, unfamiliar with the ethical arguments, the laws in the state, the clinical evidence about the value of feeding in those with terminal diseases, and the fact that the Supreme Court justices ruled ANH to be a form of medical treatment and could be terminated, believe that without ANH the person would "starve to death" rather than die of the underlying terminal illness. In most instances, the patient is the ultimate decision maker, but healthcare providers need to share the facts and the options so that the patient or the healthcare proxy can make an informed decision based on nutrition needs, medical status, and cultural and spiritual needs.

## Summary

The registered dietician is responsible for assessing the nutrition and hydration needs of patients within the context of quality of life and the individual's values and beliefs. The patient should participate in the decision and be informed of the clinical evidence and the risks and benefits of the various interventions. This is the basis of informed consent. Respecting the patient's choice upholds the ethical principle of **autonomy** and in our culture often overrides the principle of **beneficence**, that is, doing good (or nonmaleficence, that is, not doing harm). When conflict occurs, the ethics team or committee, including the registered dietician, should frame the moral questions and situation, identify options, and keep the best interests of the patient at the center of the discussion. The use of advance directives is a means to ensure that the patient's wishes are respected even when the patient cannot participate in decision making.

**autonomy** The paramount ethical principle that states the competent adult has the right to make choices about treatment options.

**beneficence** The ethical principle of doing good; for example, proposing a therapy that is worthwhile for the patient (patient centered). This agrees with the ethical principle of nonmaleficence, which is "not doing any harm."

## Case Study

### Ethics

Julie O'Sullivan Maillet, PhD, RD

Mrs. S is a 75-year-old woman with progressively severe dementia. She has had repeated episodes of aspiration pneumonia and is eating and drinking limited quantity of food. She lacks decision-making capacity and does not have an advance directive as to her care wishes.

#### Questions

1. What is the moral/ethical question in this case?
2. What are the facts?
3. What other information do you need to make an ethical decision about the care of Mrs. S? (Include medical, rules, ethical, and cultural information.)
4. What are the options and what do you recommend?

## References

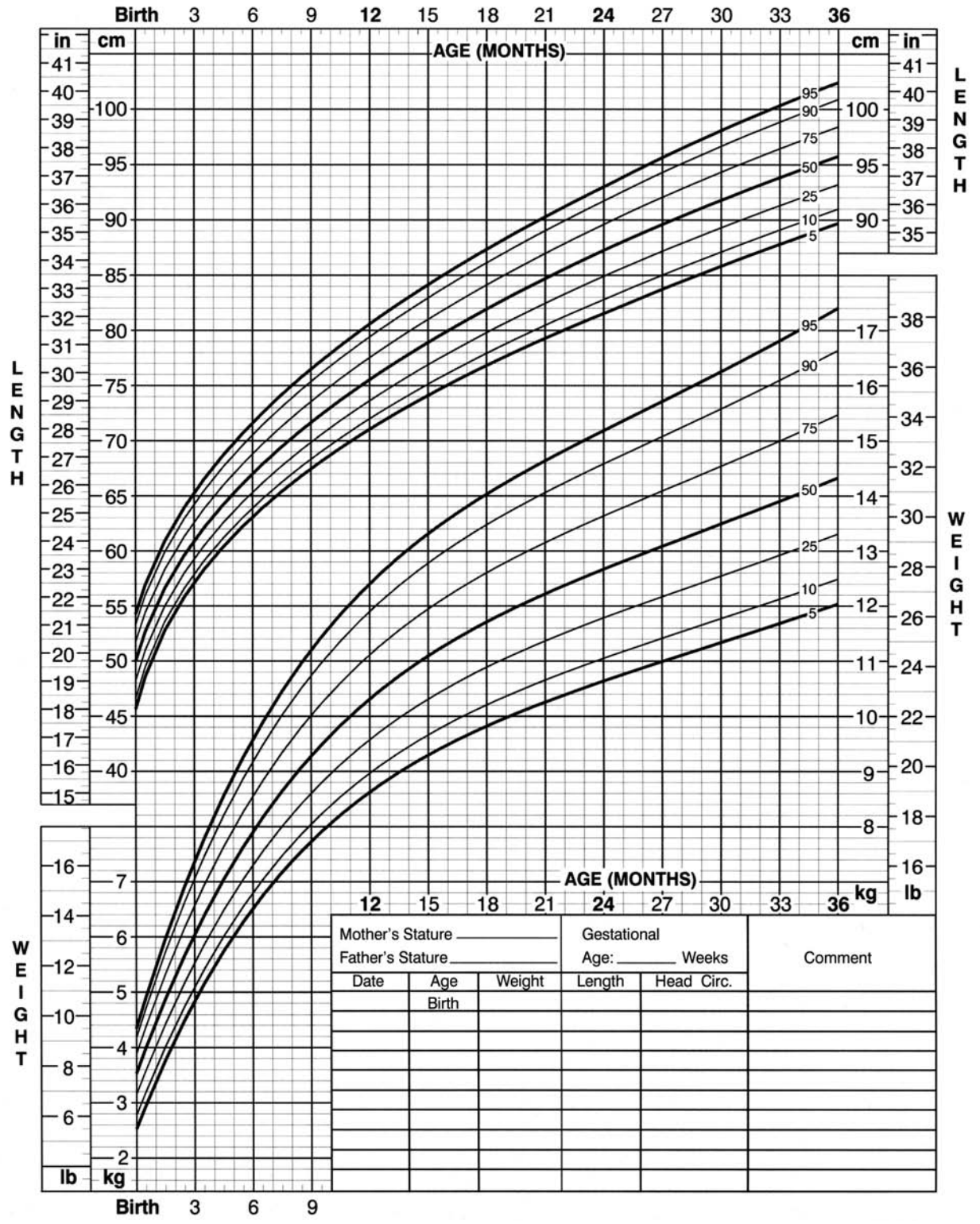

Academy of Nutrition and Dietetics. (2013a). Academic Nutrition and Dietetics position paper. Ethical and legal issues in feeding and hydration. *Journal of the Academy of Nutrition and Dietetics, 113(6)*, 828–833.

Academy of Nutrition and Dietetics. (2013b). Academic Nutrition and Dietetics practice paper. Ethical and legal

issues in feeding and hydration. Published on the Academy's website, www.eatright.org, June 2013.
American Dietetic Association/Commission on Dietetic Registration. (2009). Code of ethics for the profession of dietetics and process for consideration of ethical issues. *Journal of the American Dietetic Association*, 109(8), 1461–1467.
American Society of Parenteral and Enteral Nutrition Task Force. (2010). A.S.P.E.N. ethics position paper. Retrieved from http://nutritioncare.org/Index.aspx?id=5850
Annas, G. J. (2004). *The rights of patients* (3rd ed.). New York: New York University Press.
Annas, G. J. (2005). "Culture of life" politics at the bedside: The case of Terri Schiavo. *New England Journal of Medicine, 352*, 1710–1715.
Blendon, R. J., Benson, J. M., & Herrmann, M. J. (2005). The American public and the Terri Schiavo case. *Archives of Internal Medicine, 65*, 2580–2584.
Brett, A. S., & Rosenberg, J. C. (2001). The adequacy of informed consent for placement of gastrostomy tubes. *Archives of Internal Medicine, 161*, 745–748.
Care at the end of life [Editorial]. (2012, November 25). *New York Times*, p. SR10.
Casarett, D., Kapo, J., & Caplan, A. (2005). Appropriate use of artificial nutrition and hydration: Fundamental principles and recommendations. *New England Journal of Medicine, 353*, 2607–2612.
*In re Cruzan v. Director MDH, et al.* (1990). In the Supreme Court of the United States. No. 88-1503. Nancy Beth Cruzan, by her parents and co-guardians, Lester l. Cruzan, et ux, petitioners, v. Director, Missouri Department of Health, et al. *Issues in Law and Medicine*, *5*(4), 453–478.
DeLegge, M. H. (2009). Tube feeding in patients with dementia: Where are we? *Nutrition in Clinical Practice*, *24*(2), 214–216.
Ersek, M. (2003). Artificial nutrition and hydration: Clinical issues. *Journal of Hospice Palliative Care Nursing, 5*, 221–230.
Fine, R. L. (2006). Ethical issues in artificial nutrition and hydration. *Nutrition in Clinical Practice*, *21*, 118–125.
Finucane, T. E., Christmas, C., & Travis, K. (1999). Tube feeding in patients with advanced dementia: A review of the evidence. *Journal of the American Medical Association, 282*, 1365–1370.
Gallagher, C. R. (1989). *Nutritional care of the terminally ill*. Rockville, MD: Aspen.
Gallagher-Allred, C. R. (2012). Communication and education for families dealing with end-of-life decisions. *Journal of the Academy of Nutrition and Dietetics*, *112*(2), 309–310.
Ganzini, L. (2006). Artificial nutrition and hydration at the end of life: Ethics and evidence. *Palliative and Supportive Care, 4*, 135–143.
Geppert, C. M. A., Andrews, M. R., & Druyan, M. E. (2010). Ethical issues in artificial nutrition and hydration: A review. *Journal of Enteral and Parenteral Nutrition*, *34*(1), 79–88.
Gillick, M. R. (2004). Advance care planning. *New England Journal of Medicine, 350*, 7–8.
Hartocollis, A. (2012, October 7). Changing her mind, a Queens woman decides to remain on life support. *New York Times*, p. A27.
Huang, Z. B., & Ahronheim, J. C. (2000). Nutrition and hydration in terminally ill patients: An update. *Clinics in Geriatric Medicine*, 16, 313–325.
Hurley, D. L., & McMahon, M. M. (2005). Diabetes mellitus. In R. H. Rolandelli, R. Bankhead, J. I. Boulatta, & C. W. Compher (Eds.), *Clinical nutrition: Enteral and tube feeding* (4th ed., pp. 498–505). Philadelphia, PA: W. B. Saunders.
Koch, T. (2005). The challenge of Terri Schiavo: Lessons for bioethics. *Journal of Medical Ethics*, *31*, 376–378.
McClave, S. A., & Chang, W. K. (2003). Complications of enteral access. *Gastrointestinal Endoscopy*, *58*, 739–751.
McMahon, M. M. (2004). Parenteral nutrition. In L. Goldman & D. Aussiello (Eds.), *Cecil's textbook of medicine* (Vol. 2, 22nd ed., pp. 1322–1326). Philadelphia, PA: W. B. Saunders.
McMahon, M. M., Hurley, D. L., Kamath, P. S., & Mueller, P. S. (2005). Medical and ethical aspects of long-term enteral tube feeding. *Mayo Clinic Proceedings*, *80*, 1461–1476.
Meisel, A., & Cerminara, K. L. (2004). *The right to die: The law of end-of-life decision making* (3rd ed.). New York, NY: Aspen.
Mitchell, S. L., Buchanan, J. L., Littlehale, S., & Hamel, M. B. (2003). Tube feeding versus hand feeding nursing home residents in advanced dementia. *Journal of the American Geriatrics Society*, *51*, 129–131.
Mitsumoto, H., & Rabkin, J. G. (2007). Palliative care for patients with amyotrophic lateral sclerosis. *Journal of the American Medical Association*, *298*, 207–216.
Morita, T., Hyodo, I., Yoshimi, T., Ikenaga, M., Tamura, Y., Yoshizawa, A., . . . Japan Palliative Oncology Study Group. (2005). Association between hydration volume and symptoms in terminally ill cancer patients with abdominal malignancies. *Annals of Oncology*, *16*, 640–647.
Mueller, P. S., Hook, C. C., & Fleming, K. C. (2004). Ethical issues in geriatrics: A guide for clinicians. *Mayo Clinic Proceedings*, *79*, 554–562.
Paris, J. J. (2005). To feed or not to feed: Terri Schiavo and the use of artificial nutrition and fluids. *Southern Medical Journal*, *98*, 757–758.
Pope John Paul II. (2004). Address to the participants in the International Congress on Life Sustaining Treatments and Vegetative States: Scientific advances and ethical dilemmas. *Origins*, *33*, 738–740.
Printz, L. A. (1992). Terminal dehydration, a compassionate treatment. *Archives of Internal Medicine*, *152*, 697–700.
Quill, T. E. (2005). Terri Schiavo: A tragedy compounded. *New England Journal of Medicine*, *352*, 1630–1633.
*In re Quinlan.* (1976). 70 N.J. 10, 355. A.2d 647.
Salmond, S. W., & David, E. (2005). Attitudes toward advance directives and advance directive completion rates. *Orthopaedic Nursing*, *24*(2), 117–127.
Slomka, J. (2003). Withholding nutrition at the end of life: Clinical and ethical issues. *Cleveland Clinic Journal of Medicine*, *70*, 548–552.
Snyder, L., Leffler, C., & the Ethics and Human Rights Committee, American College of Physicians. (2005). Ethics manual. *Annals of Internal Medicine*, *142*, 560–582.

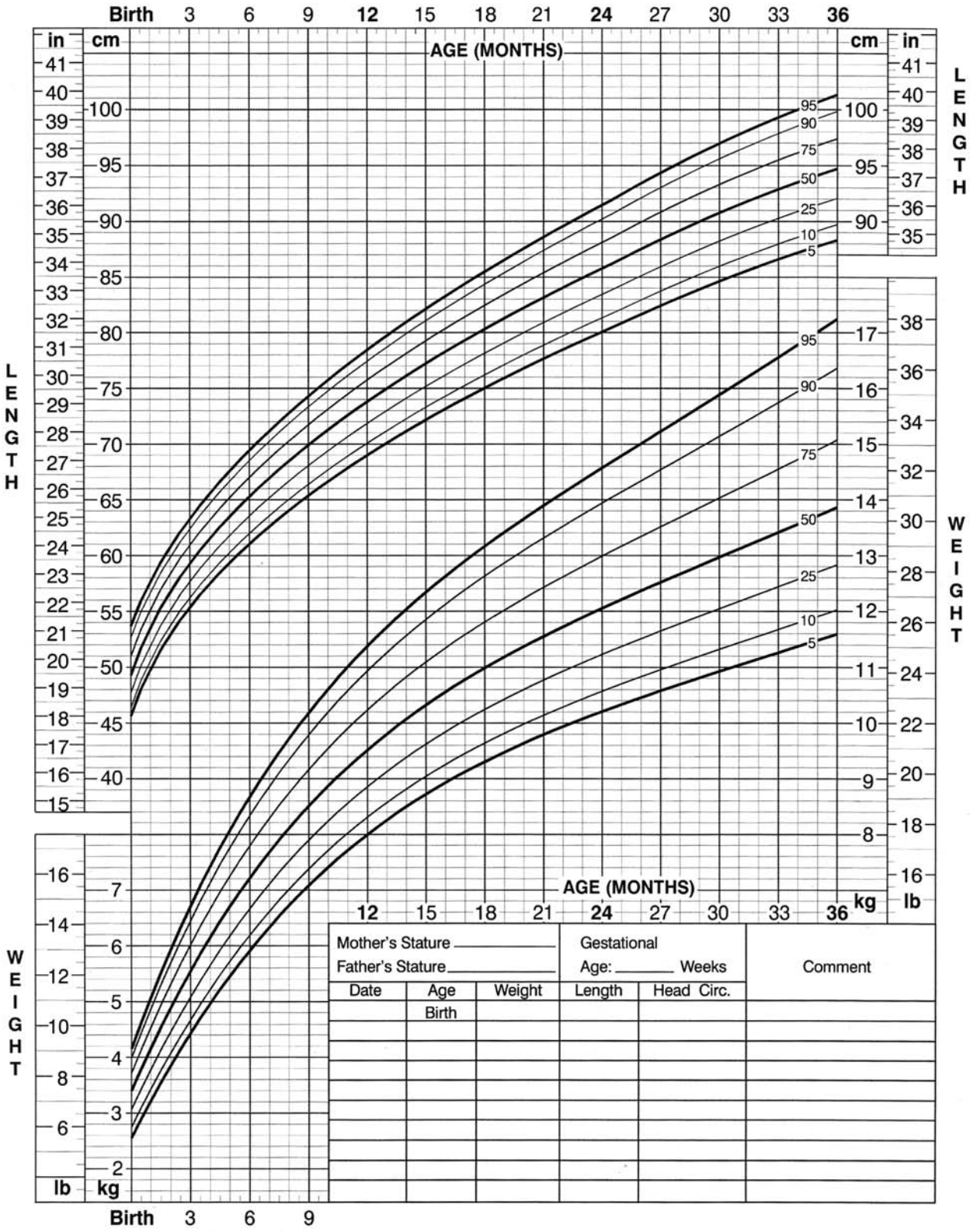
# APPENDIX 1

# Centers for Disease Control and Prevention Growth Charts




Boys, birth to 36 months: length-for-age and weight-for-age percentiles.

Courtesy of CDC.




Girls, birth to 36 months: length-for-age and weight-for-age percentiles.

Courtesy of CDC.

# APPENDIX 2 Dietary Reference Intakes (DRIs)

## Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Vitamins

### Food and Nutrition Board, Institute of Medicine, National Academies

| Life Stage Group | Vit A (µg/d)[a] | Vit C (mg/d) | Vit D (µg/d)[b,c] | Vit E (mg/d)[d] | Vit K (µg/d) | Thia-min (mg/d) | Ribo-flavin (mg/d) | Niacin (mg/d)[e] | Vit $B_6$ (mg/d) | Folate (µg/d)[f] | Vit $B_{12}$ (µg/d) | Pantothenic Acid (mg/d) | Biotin (µg/d) | Choline (mg/d)[g] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | | | | | | | | |
| 0–6 mo | 400* | 40* | 5* | 4* | 2.0* | 0.2* | 0.3* | 2* | 0.1* | 65* | 0.4* | 1.7* | 5* | 125* |
| 7–12 mo | 500* | 50* | 5* | 5* | 2.5* | 0.3* | 0.4* | 4* | 0.3* | 80* | 0.5* | 1.8* | 6* | 150* |
| *Children* | | | | | | | | | | | | | | |
| 1–3 y | 300 | 15 | 5* | 6 | 30* | 0.5 | 0.5 | 6 | 0.5 | 150 | 0.9 | 2* | 8* | 200* |
| 4–8 y | 400 | 25 | 5* | 7 | 55* | 0.6 | 0.6 | 8 | 0.6 | 200 | 1.2 | 3* | 12* | 250* |
| *Males* | | | | | | | | | | | | | | |
| 9–13 y | 600 | 45 | 5* | 11 | 60* | 0.9 | 0.9 | 12 | 1.0 | 300 | 1.8 | 4* | 20* | 375* |
| 14–18 y | 900 | 75 | 5* | 15 | 75* | 1.2 | 1.3 | 16 | 1.3 | 400 | 2.4 | 5* | 25* | 550* |
| 19–30 y | 900 | 90 | 5* | 15 | 120* | 1.2 | 1.3 | 16 | 1.3 | 400 | 2.4 | 5* | 30* | 550* |
| 31–50 y | 900 | 90 | 5* | 15 | 120* | 1.2 | 1.3 | 16 | 1.3 | 400 | 2.4 | 5* | 30* | 550* |
| 51–70 y | 900 | 90 | 10* | 15 | 120* | 1.2 | 1.3 | 16 | 1.7 | 400 | 2.4[i] | 5* | 30* | 550* |
| >70 y | 900 | 90 | 15* | 15 | 120* | 1.2 | 1.3 | 16 | 1.7 | 400 | 2.4[i] | 5* | 30* | 550* |
| *Females* | | | | | | | | | | | | | | |
| 9–13 y | 600 | 45 | 5* | 11 | 60* | 0.9 | 0.9 | 12 | 1.0 | 300 | 1.8 | 4* | 20* | 375* |
| 14–18 y | 700 | 65 | 5* | 15 | 75* | 1.0 | 1.0 | 14 | 1.2 | 400[i] | 2.4 | 5* | 25* | 400* |
| 19–30 y | 700 | 75 | 5* | 15 | 90* | 1.1 | 1.1 | 14 | 1.3 | 400[i] | 2.4 | 5* | 30* | 425* |
| 31–50 y | 700 | 75 | 5* | 15 | 90* | 1.1 | 1.1 | 14 | 1.3 | 400[i] | 2.4 | 5* | 30* | 425* |
| 51–70 y | 700 | 75 | 10* | 15 | 90* | 1.1 | 1.1 | 14 | 1.5 | 400 | 2.4[h] | 5* | 30* | 425* |
| >70 y | 700 | 75 | 15* | 15 | 90* | 1.1 | 1.1 | 14 | 1.5 | 400 | 2.4[h] | 5* | 30* | 425* |
| *Pregnancy* | | | | | | | | | | | | | | |
| 14–18 y | 750 | 80 | 5* | 15 | 75* | 1.4 | 1.4 | 18 | 1.9 | 600[j] | 2.6 | 6* | 30* | 450* |
| 19–30 y | 770 | 85 | 5* | 15 | 90* | 1.4 | 1.4 | 18 | 1.9 | 600[j] | 2.6 | 6* | 30* | 450* |
| 31–50 y | 770 | 85 | 5* | 15 | 90* | 1.4 | 1.4 | 18 | 1.9 | 600[j] | 2.6 | 6* | 30* | 450* |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Lactation* | | | | | | | | | | | | | | |
| 14–18 y | 1,200 | 115 | 5* | 19 | 75* | 1.4 | 1.6 | 17 | 2.0 | 500 | 2.8 | 7* | 35* | 550* |
| 19–30 y | 1,300 | 120 | 5* | 19 | 90* | 1.4 | 1.6 | 17 | 2.0 | 500 | 2.8 | 7* | 35* | 550* |
| 31–50 y | 1,300 | 120 | 5* | 19 | 90* | 1.4 | 1.6 | 17 | 2.0 | 500 | 2.8 | 7* | 35* | 550* |

**NOTE:** This table (taken from the DRI reports, see www.nap.edu) presents Recommended Dietary Allowances (RDAs) and Adequate Intakes (AIs). The latter are followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98 percent) individuals in a group. For healthy breast-fed infants, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

[a] As retinol activity equivalents (RAEs). 1 RAE =1 μg retinol, 12 μg β-carotene, 24 μg α-carotene, or 24 μg β-cryptoxanthin. The RAE for dietary provitamin A carotenoids is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A is the same as RE.

[b] As cholecalciferol. 1 μg cholecalciferol = 40 IU vitamin D.

[c] In the absence of adequate exposure to sunlight.

[d] As α-tocopherol. α-Tocopherol includes *RRR*-α-tocopherol, the only form of α-tocopherol that occurs naturally in foods, and the 2R-stereoisomeric forms of α-tocopherol (*RRR*-, *RSR*-, *RRS*-, and *RSS*-α-tocopherol) that occur in fortified foods and supplements. It does not include the 2S-stereoisomeric forms of α-tocopherol (*SRR*-, *SSR*-, *SRS*-, and *SSS*-α-tocopherol), also found in fortified foods and supplements.

[e] As niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan; 0–6 months = preformed niacin (not NE).

[f] As dietary folate equivalents (DFE). 1 DFE = 1 μg food folate = 0.6 μg of folic acid from fortified food or as a supplement consumed with food = 0.5 μg of a supplement taken on an empty stomach.

[g] Although AIs have been set for choline, there are few data to assess whether a dietary supply of choline is needed at all stages of the life cycle, and it may be that the choline requirement can be met by endogenous synthesis at some of these stages.

[h] Because 10 to 30 percent of older people may malabsorb food-bound $B_{12}$, it is advisable for those older than 50 years to meet their RDA mainly by consuming foods fortified with $B_{12}$ or a supplement containing $B_{12}$.

[i] In view of evidence linking folate intake with neural tube defects in the fetus, it is recommended that all women capable of becoming pregnant consume 400 μg from supplements or fortified foods in addition to intake of food folate from a varied diet.

[j] It is assumed that women will continue consuming 400 μg from supplements or fortified food until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptional period—the critical time for formation of the neural tube.

Data from Dietary *Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); and *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc* (2001). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Elements

### Food and Nutrition Board, Institute of Medicine, National Academies

| Life Stage Group | Calcium (mg/d) | Chromium (µg/d) | Copper (µg/d) | Fluoride (mg/d) | Iodine (µg/d) | Iron (mg/d) | Magnesium (mg/d) | Manganese (mg/d) | Molybdenum (µg/d) | Phosphorus (mg/d) | Selenium (µg/d) | Zinc (mg/d) | Potassium (g/d) | Sodium (g/d) | Chloride (g/d) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | | | | | | | | | |
| 0–6 mo | 210* | 0.2* | 200* | 0.01* | 110* | 0.27* | 30* | 0.003* | 2* | 100* | 15* | 2* | 0.4* | 0.12* | 0.18* |
| 7–12 mo | 270* | 5.5* | 220* | 0.5* | 130* | 11 | 75* | 0.6* | 3* | 275* | 20* | 3 | 0.7* | 0.37* | 0.57* |
| *Children* | | | | | | | | | | | | | | | |
| 1–3 y | 500* | 11* | 340 | 0.7* | 90 | 7 | 80 | 1.2* | 17 | 460 | 20 | 3 | 3.0* | 1.0* | 1.5* |
| 4–8 y | 800* | 15* | 440 | 1* | 90 | 10 | 130 | 1.5* | 22 | 500 | 30 | 5 | 3.8* | 1.2* | 1.9* |
| *Males* | | | | | | | | | | | | | | | |
| 9–13 y | 1,300* | 25* | 700 | 2* | 120 | 8 | 240 | 1.9* | 34 | 1,250 | 40 | 8 | 4.5* | 1.5* | 2.3* |
| 14–18 y | 1,300* | 35* | 890 | 3* | 150 | 11 | 410 | 2.2* | 43 | 1,250 | 55 | 11 | 4.7* | 1.5* | 2.3* |
| 19–30 y | 1,000* | 35* | 900 | 4* | 150 | 8 | 400 | 2.3* | 45 | 700 | 55 | 11 | 4.7* | 1.5* | 2.3* |
| 31–50 y | 1,000* | 35* | 900 | 4* | 150 | 8 | 420 | 2.3* | 45 | 700 | 55 | 11 | 4.7* | 1.5* | 2.3* |
| 51–70 y | 1,200* | 30* | 900 | 4* | 150 | 8 | 420 | 2.3* | 45 | 700 | 55 | 11 | 4.7* | 1.3* | 2.0* |
| >70 y | 1,200* | 30* | 900 | 4* | 150 | 8 | 420 | 2.3* | 45 | 700 | 55 | 11 | 4.7* | 1.2* | 1.8* |
| *Females* | | | | | | | | | | | | | | | |
| 9–13 y | 1,300* | 21* | 700 | 2* | 120 | 8 | 240 | 1.6* | 34 | 1,250 | 40 | 8 | 4.5* | 1.5* | 2.3* |
| 14–18 y | 1,300* | 24* | 890 | 3* | 150 | 15 | 360 | 1.6* | 43 | 1,250 | 55 | 9 | 4.7* | 1.5* | 2.3* |
| 19–30 y | 1,000* | 25* | 900 | 3* | 150 | 18 | 310 | 1.8* | 45 | 700 | 55 | 8 | 4.7* | 1.5* | 2.3* |
| 31–50 y | 1,000* | 25* | 900 | 3* | 150 | 18 | 320 | 1.8* | 45 | 700 | 55 | 8 | 4.7* | 1.5* | 2.3* |
| 51–70 y | 1,200* | 20* | 900 | 3* | 150 | 8 | 320 | 1.8* | 45 | 700 | 55 | 8 | 4.7* | 1.3* | 2.0* |
| >70 y | 1,200* | 20* | 900 | 3* | 150 | 8 | 320 | 1.8* | 45 | 700 | 55 | 8 | 4.7* | 1.2* | 1.8* |
| *Pregnancy* | | | | | | | | | | | | | | | |
| 14–18 y | 1,300* | 29* | 1,000 | 3* | 220 | 27 | 400 | 2.0* | 50 | 1,250 | 60 | 12 | 4.7* | 1.5* | 2.3* |
| 19–30 y | 1,000* | 30* | 1,000 | 3* | 220 | 27 | 350 | 2.0* | 50 | 700 | 60 | 11 | 4.7* | 1.5* | 2.3* |
| 31–50 y | 1,000* | 30* | 1,000 | 3* | 220 | 27 | 360 | 2.0* | 50 | 700 | 60 | 11 | 4.7* | 1.5* | 2.3* |
| *Lactation* | | | | | | | | | | | | | | | |
| 14–18 y | 1,300* | 44* | 1,300 | 3* | 290 | 10 | 360 | 2.6* | 50 | 1,250 | 70 | 13 | 5.1* | 1.5* | 2.3* |
| 19–30 y | 1,000* | 45* | 1,300 | 3* | 290 | 9 | 310 | 2.6* | 50 | 700 | 70 | 12 | 5.1* | 1.5* | 2.3* |
| 31–50 y | 1,000* | 45* | 1,300 | 3* | 290 | 9 | 320 | 2.6* | 50 | 700 | 70 | 12 | 5.1* | 1.5* | 2.3* |

**NOTE:** This table presents Recommended Dietary Allowances (RDAs) and Adequate Intakes (AIs). The latter are followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98 percent) individuals in a group. For healthy breast-fed infants, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

Data from *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc* (2001); and *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate* (2004). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels (UL[a]), Vitamins

### Food and Nutrition Board, Institute of Medicine, National Academies

| Life Stage Group | Vitamin A (µg/d)[b] | Vitamin C (mg/d) | Vitamin D (µg/d) | Vitamin E (mg/d)[c,d] | Vitamin K | Thiamin | Ribo-flavin | Niacin (mg/d)[d] | Vitamin $B_6$ (mg/d) | Folate (µg/d)[d] | Vitamin $B_{12}$ | Pantothenic Acid | Biotin | Choline (g/d) | Carot-enoids[e] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | | | | | | | | | |
| 0–6 mo | 600 | ND[f] | 25 | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| 7–12 mo | 600 | ND | 25 | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| *Children* | | | | | | | | | | | | | | | |
| 1–3 y | 600 | 400 | 50 | 200 | ND | ND | ND | 10 | 30 | 300 | ND | ND | ND | 1.0 | ND |
| 4–8 y | 900 | 650 | 50 | 300 | ND | ND | ND | 15 | 40 | 400 | ND | ND | ND | 1.0 | ND |
| *Males, Females* | | | | | | | | | | | | | | | |
| 9–13 y | 1,700 | 1,200 | 50 | 600 | ND | ND | ND | 20 | 60 | 600 | ND | ND | ND | 2.0 | ND |
| 14–18 y | 2,800 | 1,800 | 50 | 800 | ND | ND | ND | 30 | 80 | 800 | ND | ND | ND | 3.0 | ND |
| 19–70 y | 3,000 | 2,000 | 50 | 1,000 | ND | ND | ND | 35 | 100 | 1,000 | ND | ND | ND | 3.5 | ND |
| >70 y | 3,000 | 2,000 | 50 | 1,000 | ND | ND | ND | 35 | 100 | 1,000 | ND | ND | ND | 3.5 | ND |
| *Pregnancy* | | | | | | | | | | | | | | | |
| 14–18 y | 2,800 | 1,800 | 50 | 800 | ND | ND | ND | 30 | 80 | 800 | ND | ND | ND | 3.0 | ND |
| 19-50 y | 3,000 | 2,000 | 50 | 1,000 | ND | ND | ND | 35 | 100 | 1,000 | ND | ND | ND | 3.5 | ND |
| *Lactation* | | | | | | | | | | | | | | | |
| 14–18 y | 2,800 | 1,800 | 50 | 800 | ND | ND | ND | 30 | 80 | 800 | ND | ND | ND | 3.0 | ND |
| 19–50 y | 3,000 | 2,000 | 50 | 1,000 | ND | ND | ND | 35 | 100 | 1,000 | ND | ND | ND | 3.5 | ND |

[a]UL =The maximum level of daily nutrient intake that is likely to pose no risk of adverse effects. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to lack of suitable data, ULs could not be established for vitamin K, thiamin, riboflavin, vitamin B12, pantothenic acid, biotin, carotenoids. In the absence of ULs, extra caution may be warranted in consuming levels above recommended intakes.

[b]As preformed vitamin A only.

[c]As α-tocopherol; applies to any form of supplemental a-tocopherol.

[d]The ULs for vitamin E, niacin, and folate apply to synthetic forms obtained from supplements, fortified foods, or a combination of the two.

[e]β-Carotene supplements are advised only to serve as a provitamin A source for individuals at risk of vitamin A deficiency.

[f]ND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intake.

Data from *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); and *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc* (2001). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels (UL[a]), Elements

### Food and Nutrition Board, Institute of Medicine, National Academies

| Life Stage Group | Arsenic[b] | Boron (mg/d) | Calcium (g/d) | Chromium | Copper (µg/d) | Fluoride (mg/d) | Iodine (µg/d) | Iron (mg/d) | Magnesium (mg/d)[c] | Manganese (mg/d) | Molybdenum (µg/d) | Nickel (mg/d) | Phosphorus (g/d) | Potassium | Selenium (µg/d) | Silicon[d] | Sulfate | Vanadium (mg/d)[e] | Zinc (mg/d) | Sodium (g/d) | Chloride (g/d) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | | | | | | | | | | | | | | | |
| 0–6 mo | ND[f] | ND | ND | ND | ND | 0.7 | ND | 40 | ND | ND | ND | ND | ND | ND | 45 | ND | ND | ND | 4 | ND | ND |
| 7–12 mo | ND | ND | ND | ND | ND | 0.9 | ND | 40 | ND | ND | ND | ND | ND | ND | 60 | ND | ND | ND | 5 | ND | ND |
| *Children* | | | | | | | | | | | | | | | | | | | | | |
| 1–3 y | ND | 3 | 2.5 | ND | 1,000 | 1.3 | 200 | 40 | 65 | 2 | 300 | 0.2 | 3 | ND | 90 | ND | ND | ND | 7 | 1.5 | 2.3 |
| 4–8 y | ND | 6 | 2.5 | ND | 3,000 | 2.2 | 300 | 40 | 110 | 3 | 600 | 0.3 | 3 | ND | 150 | ND | ND | ND | 12 | 1.9 | 2.9 |
| *Males, Females* | | | | | | | | | | | | | | | | | | | | | |
| 9–13 y | ND | 11 | 2.5 | ND | 5,000 | 10 | 600 | 40 | 350 | 6 | 1,100 | 0.6 | 4 | ND | 280 | ND | ND | ND | 23 | 2.2 | 3.4 |
| 14–18 y | ND | 17 | 2.5 | ND | 8,000 | 10 | 900 | 45 | 350 | 9 | 1,700 | 1.0 | 4 | ND | 400 | ND | ND | ND | 34 | 2.3 | 3.6 |
| 19–70 y | ND | 20 | 2.5 | ND | 10,000 | 10 | 1,100 | 45 | 350 | 11 | 2,000 | 1.0 | 4 | ND | 400 | ND | ND | 1.8 | 40 | 2.3 | 3.6 |
| >70 y | ND | 20 | 2.5 | ND | 10,000 | 10 | 1,100 | 45 | 350 | 11 | 2,000 | 1.0 | 3 | ND | 400 | ND | ND | 1.8 | 40 | 2.3 | 3.6 |
| *Pregnancy* | | | | | | | | | | | | | | | | | | | | | |
| 14–18 y | ND | 17 | 2.5 | ND | 8,000 | 10 | 900 | 45 | 350 | 9 | 1,700 | 1.0 | 3.5 | ND | 400 | ND | ND | ND | 34 | 2.3 | 3.6 |
| 19–50 y | ND | 20 | 2.5 | ND | 10,000 | 10 | 1,100 | 45 | 350 | 11 | 2,000 | 1.0 | 3.5 | ND | 400 | ND | ND | ND | 40 | 2.3 | 3.6 |
| *Lactation* | | | | | | | | | | | | | | | | | | | | | |
| 14–18 y | ND | 17 | 2.5 | ND | 8,000 | 10 | 900 | 45 | 350 | 9 | 1,700 | 1.0 | 4 | ND | 400 | ND | ND | ND | 34 | 2.3 | 3.6 |
| 19–50 y | ND | 20 | 2.5 | ND | 10,000 | 10 | 1,100 | 45 | 350 | 11 | 2,000 | 1.0 | 4 | ND | 400 | ND | ND | ND | 40 | 2.3 | 3.6 |

[a]UL = The maximum level of daily nutrient intake that is likely to pose no risk of adverse effects. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to lack of suitable data, ULs could not be established for arsenic, chromium, silicon, potassium, and sulfate. In the absence of ULs, extra caution may be warranted in consuming levels above recommended intakes.
[b]Although the UL was not determined for arsenic, there is no justification for adding arsenic to food or supplements.
[c]The ULs for magnesium represent intake from a pharmacological agent only and do not include intake from food and water.
[d]Although silicon has not been shown to cause adverse effects in humans, there is no justification for adding silicon to supplements.
[e]Although vanadium in food has not been shown to cause adverse effects in humans, there is no justification for adding vanadium to food and vanadium supplements should be used with caution. The UL is based on adverse effects in laboratory animals and this data could be used to set a UL for adults but not children and adolescents.
[f]ND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intake.

Data from *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc* (2001); and *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate* (2004). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Estimated Energy Requirements (EER) for Men and Women 30 Years of Age[a]

### Food and Nutrition Board, Institute of Medicine, National Academies

| Height (m [in]) | PAL[b] | Weight for BMI[c] of 18.5 kg/m$_2$ (kg [lb]) Weight for BMI of 24.99 kg/m$_2$ (kg [lb]) 41.6 (92) 56.2 (124) | | EER, Men[d] (kcal/day) EER, Women[d] (kcal/day) BMI of 18.5 kg/m$_2$ | | | | |
|---|---|---|---|---|---|---|---|---|
| 1.50 (59) | Sedentary | | | BMI of 24.99 kg/m$_2$ BMI of 18.5 kg/m$_2$ BMI of 24.99 kg/m$_2$ 1,848 2,080 | | | | |
| | Low active | | | 1,625 1,762 2,009 2,267 1,803 1,956 2,215 2,506 2,025 2,198 2,554 | | | | |
| | Active | | | 2,898 2,291 2,489 | | | | |
| | Very active | | | | | | | |
| 1.65 (65) | Sedentary | 50.4 (111) | 68.0 (150) | 2,068 | 2,349 | 1,816 | | |
| | Low active | | | 2,254 | 2,566 | 2,016 | 1,982 | 2,202 |
| | Active | | | 2,490 | 2,842 | 2,267 | 2,477 | 2,807 |
| | Very active | | | 2,880 | 3,296 | 2,567 | | |
| 1.80 (71) | Sedentary | 59.9 (132) | 81.0 (178) | 2,301 | 2,635 | 2,015 | | |
| | Low active | | | 2,513 | 2,884 | 2,239 | | |
| | Active | | | 2,782 | 3,200 | 2,519 | 2,211 | 2,459 |
| | Very active | | | 3,225 | 3,720 | 2,855 | 2,769 | 3,141 |

[a]For each year below 30, add 7 kcal/day for women and 10 kcal /day for men. For each year above 30, subtract 7 kcal/day for women and 10 kcal/day for men.
[b]PAL = physical activity level.
[c]BMI = body mass index.
[d]Derived from the following regression equations based on doubly labeled water data:
Adult man: EER = 662 – 9.53 × age (y) + PA × (15.91 × wt [kg] + 539.6 × ht [m])
Adult woman: EER = 354 – 6.91 × age (y) + PA × (9.36 × wt [kg] + 726 × ht [m])
Where PA refers to coefficient for PAL
PAL = total energy expenditure ÷ basal energy expenditure PA = 1.0 if PAL ≥ 1.0, < 1.4 (sedentary) PA = 1.12 if PAL ≥ 1.4, < 1.6 (low active) PA = 1.27 if PAL ≥ 1.6 < 1.9 (active) PA = 1.45 if PAL ≥ 1.9 < 2.5 (very active)

Data from Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: National Academies Press.

## Dietary Reference Intakes (DRIs): Acceptable Macronutrient Distribution Ranges

### Food and Nutrition Board, Institute of Medicine, National Academies

| Range (percent of energy) Macronutrient | Children, 1–3 y | Children, 4–18 y | Adults |
|---|---|---|---|
| Fat | 30–40 | 25–35 | 20–35 |
| *n*-6 polyunsaturated fatty acids[a] (linoleic acid) | 5–10 | 5–10 | 5–10 |
| *n*-3 polyunsaturated fatty acids[a] (α-linolenic acid) | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 |
| Carbohydrate | 45–65 | 45–65 | 45–65 |
| Protein | 5–20 | 10–30 | 10–35 |

[a]Approximately 10% of the total can come from longer-chain *n*-3 or *n*-6 fatty acids.

Data from Institute of Medicine, Food and Nutrition Board. (2005). *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: National Academies Press.

## Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Macronutrients

### Food and Nutrition Board, Institute of Medicine, National Academies

| Total Life Stage Group | Water[a] (L/d) | Carbohydrate (g/d) | Total Fiber (g/d) | Fat (g/d) | Linoleic Acid (g/d) | a-Linolenic Acid (g/d) | Protein[b] (g/d) |
|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | |
| 0–6 mo | 0.7* | 60* | ND | 31* | 4.4* | 0.5* | 9.1* |
| 7–12 mo | 0.8* | 95* | ND | 30* | 4.6* | 0.5* | 11.0[c] |
| *Children* | | | | | | | |
| 1–3 y | 1.3* | 130 | 19* | ND | 7* | 0.7* | 13 |
| 4–8 y | 1.7* | 130 | 25* | ND | 10* | 0.9* | 19 |
| *Males* | | | | | | | |
| 9–13 y | 2.4* | 130 | 31* | ND | 12* | 1.2* | 34 |
| 14–18 y | 3.3* | 130 | 38* | ND | 16* | 1.6* | 52 |
| 19–30 y | 3.7* | 130 | 38* | ND | 17* | 1.6* | 56 |
| 31–50 y | 3.7* | 130 | 38* | ND | 17* | 1.6* | 56 |
| 51–70 y | 3.7* | 130 | 30* | ND | 14* | 1.6* | 56 |
| >70 y | 3.7* | 130 | 30* | ND | 14* | 1.6* | 56 |
| *Females* | | | | | | | |
| 9–13 y | 2.1* | 130 | 26* | ND | 10* | 1.0* | 34 |
| 14–18 y | 2.3* | 130 | 26* | ND | 11* | 1.1* | 46 |
| 19–30 y | 2.7* | 130 | 25* | ND | 12* | 1.1* | 46 |
| 31–50 y | 2.7* | 130 | 25* | ND | 12* | 1.1* | 46 |
| 51–70 y | 2.7* | 130 | 21* | ND | 11* | 1.1* | 46 |
| >70 y | 2.7* | 130 | 21* | ND | 11* | 1.1* | 46 |
| *Pregnancy* | | | | | | | |
| 14–18 y | 3.0* | 175 | 28* | ND | 13* | 1.4* | 71 |
| 19–30 y | 3.0* | 175 | 28* | ND | 13* | 1.4* | 71 |
| 31–50 y | 3.0* | 175 | 28* | ND | 13* | 1.4* | 71 |
| *Lactation* | | | | | | | |
| 14–18 y | 3.8* | 210 | 29* | ND | 13* | 1.3* | 71 |
| 19–30 y | 3.8* | 210 | 29* | ND | 13* | 1.3* | 71 |
| 31–50 y | 3.8* | 210 | 29* | ND | 13* | 1.3* | 71 |

**NOTE:** This table presents Recommended Dietary Allowances (RDAs) and Adequate Intakes (AIs). The latter are followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98 percent) individuals in a group. For healthy infants fed human milk, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover the needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

[a]*Total* water includes all water contained in food, beverages, and drinking water.
[b]Based on 0.8 g/kg body weight for the reference body weight.
[c]Change from 13.5 in prepublication copy due to calculation error.

Data from *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids* (2002/2005); *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate* (2005). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Additional Macronutrient Recommendations

### Food and Nutrition Board, Institute of Medicine, National Academies

| Macronutrient | Recommendation |
|---|---|
| Dietary cholesterol | As low as possible while consuming a nutritionally adequate diet |
| Trans fatty acids | As low as possible while consuming a nutritionally adequate diet |
| Saturated fatty acids | As low as possible while consuming a nutritionally adequate diet |
| Added sugars | Limit to no more than 25% of total energy |

Data from *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids* (2002/2005); *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate* (2005). These reports may be accessed via http://www.nap.edu.

## Dietary Reference Intakes (DRIs): Estimated Average Requirements for Groups

### Food and Nutrition Board, Institute of Medicine, National Academies

| Life Stage Group | CHO (g/d) | Protein (g/d)[a] | Vit A (µg/d)[b] | Vit C (mg/d) | Vit E (mg/d)[c] | Thiamin (mg/d) | Ribo-flavin (mg/d) | Niacin (mg/d)[d] | Vit $B_6$ (mg/d) | Folate (µg/d)[b] | Vit $B_{12}$ (µg/d) | Copper (µg/d) | Iodine (µg/d) | Iron (mg/d) | Mag-nesium (mg/d) | Molyb-denum (µg/d) | Phos-phorus (mg/d) | Sele-nium (µg/d) | Zinc (mg/d) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Infants* | | | | | | | | | | | | | | | | | | | |
| 7–12 mo | | 9* | | | | | | | | | | | | 6.9 | | | | | 2.5 |
| *Children* | | | | | | | | | | | | | | | | | | | |
| 1–3 y | 100 | 11 | 210 | 13 | 5 | 0.4 | 0.4 | 5 | 0.4 | 120 | 0.7 | 260 | 65 | 3.0 | 65 | 13 | 380 | 17 | 2.5 |
| 4–8 y | 100 | 15 | 275 | 22 | 6 | 0.5 | 0.5 | 6 | 0.5 | 160 | 1.0 | 340 | 65 | 4.1 | 110 | 17 | 405 | 23 | 4.0 |
| *Males* | | | | | | | | | | | | | | | | | | | |
| 9–13 y | 100 | 27 | 445 | 39 | 9 | 0.7 | 0.8 | 9 | 0.8 | 250 | 1.5 | 540 | 73 | 5.9 | 200 | 26 | 1,055 | 35 | 7.0 |
| 14–18 y | 100 | 44 | 630 | 63 | 12 | 1.0 | 1.1 | 12 | 1.1 | 330 | 2.0 | 685 | 95 | 7.7 | 340 | 33 | 1,055 | 45 | 8.5 |
| 19–30 y | 100 | 46 | 625 | 75 | 12 | 1.0 | 1.1 | 12 | 1.1 | 320 | 2.0 | 700 | 95 | 6 | 330 | 34 | 580 | 45 | 9.4 |
| 31–50 y | 100 | 46 | 625 | 75 | 12 | 1.0 | 1.1 | 12 | 1.1 | 320 | 2.0 | 700 | 95 | 6 | 350 | 34 | 580 | 45 | 9.4 |
| 51–70 y | 100 | 46 | 625 | 75 | 12 | 1.0 | 1.1 | 12 | 1.4 | 320 | 2.0 | 700 | 95 | 6 | 350 | 34 | 580 | 45 | 9.4 |
| .70 y | 100 | 46 | 625 | 75 | 12 | 1.0 | 1.1 | 12 | 1.4 | 320 | 2.0 | 700 | 95 | 6 | 350 | 34 | 580 | 45 | 9.4 |
| *Females* | | | | | | | | | | | | | | | | | | | |
| 9–13 y | 100 | 28 | 420 | 39 | 9 | 0.7 | 0.8 | 9 | 0.8 | 250 | 1.5 | 540 | 73 | 5.7 | 200 | 26 | 1,055 | 35 | 7.0 |
| 14–18 y | 100 | 38 | 485 | 56 | 12 | 0.9 | 0.9 | 11 | 1.0 | 330 | 2.0 | 685 | 95 | 7.9 | 300 | 33 | 1,055 | 45 | 7.3 |
| 19–30 y | 100 | 38 | 500 | 60 | 12 | 0.9 | 0.9 | 11 | 1.1 | 320 | 2.0 | 700 | 95 | 8.1 | 255 | 34 | 580 | 45 | 6.8 |

(*continued*)

## Dietary Reference Intakes (DRIs): Estimated Average Requirements for Groups

### Food and Nutrition Board, Institute of Medicine, National Academies *(continued)*

| Life Stage Group | CHO (g/d) | Protein (g/d)[a] | Vit A (µg/d)[b] | Vit C (mg/d) | Vit E (mg/d)[c] | Thiamin (mg/d) | Ribo-flavin (mg/d) | Niacin (mg/d)[d] | Vit $B_6$ (mg/d) | Folate (µg/d)[b] | Vit $B_{12}$ (µg/d) | Copper (µg/d) | Iodine (µg/d) | Iron (mg/d) | Mag-nesium (mg/d) | Molyb-denum (µg/d) | Phos-phorus (mg/d) | Sele-nium (µg/d) | Zinc (mg/d) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 31–50 y | 100 | 38 | 500 | 60 | 12 | 0.9 | 0.9 | 11 | 1.1 | 320 | 2.0 | 700 | 95 | 8.1 | 265 | 34 | 580 | 45 | 6.8 |
| 51–70 y | 100 | 38 | 500 | 60 | 12 | 0.9 | 0.9 | 11 | 1.3 | 320 | 2.0 | 700 | 95 | 5 | 265 | 34 | 580 | 45 | 6.8 |
| .70 y | 100 | 38 | 500 | 60 | 12 | 0.9 | 0.9 | 11 | 1.3 | 320 | 2.0 | 700 | 95 | 5 | 265 | 34 | 580 | 45 | 6.8 |
| *Pregnancy* | | | | | | | | | | | | | | | | | | | |
| 14–18 y | 135 | 50 | 530 | 66 | 12 | 1.2 | 1.2 | 14 | 1.6 | 520 | 2.2 | 785 | 160 | 23 | 335 | 40 | 1,055 | 49 | 10.5 |
| 19–30 y | 135 | 50 | 550 | 70 | 12 | 1.2 | 1.2 | 14 | 1.6 | 520 | 2.2 | 800 | 160 | 22 | 290 | 40 | 580 | 49 | 9.5 |
| 31–50 y | 135 | 50 | 550 | 70 | 12 | 1.2 | 1.2 | 14 | 1.6 | 520 | 2.2 | 800 | 160 | 22 | 300 | 40 | 580 | 49 | 9.5 |
| *Lactation* | | | | | | | | | | | | | | | | | | | |
| 14–18 y | 160 | 60 | 885 | 96 | 16 | 1.2 | 1.3 | 13 | 1.7 | 450 | 2.4 | 985 | 209 | 7 | 300 | 35 | 1,055 | 59 | 10.9 |
| 19–30 y | 160 | 60 | 900 | 100 | 16 | 1.2 | 1.3 | 13 | 1.7 | 450 | 2.4 | 1,000 | 209 | 6.5 | 255 | 36 | 580 | 59 | 10.4 |
| 31–50 y | 160 | 60 | 900 | 100 | 16 | 1.2 | 1.3 | 13 | 1.7 | 450 | 2.4 | 1,000 | 209 | 6.5 | 265 | 36 | 580 | 59 | 10.4 |

**NOTE:** This table presents Estimated Average Requirements (EARs), which serve two purposes: for assessing adequacy of population intakes, and as the basis for calculating Recommended Dietary Allowances (RDAs) for individuals for those nutrients. EARs have not been established for vitamin D, vitamin K, pantothenic acid, biotin, choline, calcium, chromium, fluoride, manganese, or other nutrients not yet evaluated via the DRI process.

[a]For individual at reference weight (Table 1-1). *indicates change from prepublication copy due to calculation error.

[b]As retinol activity equivalents (RAEs). 1 RAE = 1 µg retinol, 12 µg β-carotene, 24 µg α-carotene, or 24 µg β-cryptoxanthin. The RAE for dietary provitamin A carotenoids is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A is the same as RE.

[c]As α-tocopherol. α-Tocopherol includes *RRR*-α-tocopherol, the only form of α-tocopherol that occurs naturally in foods, and the 2R-stereoisomeric forms of α-tocopherol (*RRR*-, *RSR*-, *RRS*-, and *RSS*-α-tocopherol) that occur in fortified foods and supplements. It does not include the 2S-stereoisomeric forms of α-tocopherol (*SRR*-, *SSR*-, SRS-, and *SSS*-α-tocopherol), also found in fortified foods and supplements.

[d]As niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan.

[e]As dietary folate equivalents (DFE). 1 DFE = 1 µg food folate = 0.6 µg of folic acid from fortified food or as a supplement consumed with food = 0.5 µg of a supplement taken on an empty stomach.

Data from *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc* (2001), and *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids* (2002). These reports may be accessed via www.nap.edu.

# APPENDIX 3 Body Mass Index for Adults

## BMI Table

**Body Mass Index Table**

| | Normal | | | | | | Overweight | | | | | Obese | | | | | | | | | | Extreme Obesity | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **BMI** | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 32 | 33 | 34 | 35 | 36 | 37 | 38 | 39 | 40 | 41 | 42 | 43 | 44 | 45 | 46 | 47 | 48 | 49 | 50 | 51 | 52 | 53 | 54 |
| **Height (inches)** | **Body Weight (pounds)** | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 58 | 91 | 96 | 100 | 105 | 110 | 115 | 119 | 124 | 129 | 134 | 138 | 143 | 148 | 153 | 158 | 162 | 167 | 172 | 177 | 181 | 186 | 191 | 196 | 201 | 205 | 210 | 215 | 220 | 224 | 229 | 234 | 239 | 244 | 248 | 253 | 258 |
| 59 | 94 | 99 | 104 | 109 | 114 | 119 | 124 | 128 | 133 | 138 | 143 | 148 | 153 | 158 | 163 | 168 | 173 | 178 | 183 | 188 | 193 | 198 | 203 | 208 | 212 | 217 | 222 | 227 | 232 | 237 | 242 | 247 | 252 | 257 | 262 | 267 |
| 60 | 97 | 102 | 107 | 112 | 118 | 123 | 128 | 133 | 138 | 143 | 148 | 153 | 158 | 163 | 168 | 174 | 179 | 184 | 189 | 194 | 199 | 204 | 209 | 215 | 220 | 225 | 230 | 235 | 240 | 245 | 250 | 255 | 261 | 266 | 271 | 276 |
| 61 | 100 | 106 | 111 | 116 | 122 | 127 | 132 | 137 | 143 | 148 | 153 | 158 | 164 | 169 | 174 | 180 | 185 | 190 | 195 | 201 | 206 | 211 | 217 | 222 | 227 | 232 | 238 | 243 | 248 | 254 | 259 | 264 | 269 | 275 | 280 | 285 |
| 62 | 104 | 109 | 115 | 120 | 126 | 131 | 136 | 142 | 147 | 153 | 158 | 164 | 169 | 175 | 180 | 186 | 191 | 196 | 202 | 207 | 213 | 218 | 224 | 229 | 235 | 240 | 246 | 251 | 256 | 262 | 267 | 273 | 278 | 284 | 289 | 295 |
| 63 | 107 | 113 | 118 | 124 | 130 | 135 | 141 | 146 | 152 | 158 | 163 | 169 | 175 | 180 | 186 | 191 | 197 | 203 | 208 | 214 | 220 | 225 | 231 | 237 | 242 | 248 | 254 | 259 | 265 | 270 | 278 | 282 | 287 | 293 | 299 | 304 |
| 64 | 110 | 116 | 122 | 128 | 134 | 140 | 145 | 151 | 157 | 163 | 169 | 174 | 180 | 186 | 192 | 197 | 204 | 209 | 215 | 221 | 227 | 232 | 238 | 244 | 250 | 256 | 262 | 267 | 273 | 279 | 285 | 291 | 296 | 302 | 308 | 314 |
| 65 | 114 | 120 | 126 | 132 | 138 | 144 | 150 | 156 | 162 | 168 | 174 | 180 | 186 | 192 | 198 | 204 | 210 | 216 | 222 | 228 | 234 | 240 | 246 | 252 | 258 | 264 | 270 | 276 | 282 | 288 | 294 | 300 | 306 | 312 | 318 | 324 |
| 66 | 118 | 124 | 130 | 136 | 142 | 148 | 155 | 161 | 167 | 173 | 179 | 186 | 192 | 198 | 204 | 210 | 216 | 223 | 229 | 235 | 241 | 247 | 253 | 260 | 266 | 272 | 278 | 284 | 291 | 297 | 303 | 309 | 315 | 322 | 328 | 334 |
| 67 | 121 | 127 | 134 | 140 | 146 | 153 | 159 | 166 | 172 | 178 | 185 | 191 | 198 | 204 | 211 | 217 | 223 | 230 | 236 | 242 | 249 | 255 | 261 | 268 | 274 | 280 | 287 | 293 | 299 | 306 | 312 | 319 | 325 | 331 | 338 | 344 |
| 68 | 125 | 131 | 138 | 144 | 151 | 158 | 164 | 171 | 177 | 184 | 190 | 197 | 203 | 210 | 216 | 223 | 230 | 236 | 243 | 249 | 256 | 262 | 269 | 276 | 282 | 289 | 295 | 302 | 308 | 315 | 322 | 328 | 335 | 341 | 348 | 354 |
| 69 | 128 | 135 | 142 | 149 | 155 | 162 | 169 | 176 | 182 | 189 | 196 | 203 | 209 | 216 | 223 | 230 | 236 | 243 | 250 | 257 | 263 | 270 | 277 | 284 | 291 | 297 | 304 | 311 | 318 | 324 | 331 | 338 | 345 | 351 | 358 | 365 |
| 70 | 132 | 139 | 146 | 153 | 160 | 167 | 174 | 181 | 188 | 195 | 202 | 209 | 216 | 222 | 229 | 236 | 243 | 250 | 257 | 264 | 271 | 278 | 285 | 292 | 299 | 306 | 313 | 320 | 327 | 334 | 341 | 348 | 355 | 362 | 369 | 376 |
| 71 | 136 | 143 | 150 | 157 | 165 | 172 | 179 | 186 | 193 | 200 | 208 | 215 | 222 | 229 | 236 | 243 | 250 | 257 | 265 | 272 | 279 | 286 | 293 | 301 | 308 | 315 | 322 | 329 | 338 | 343 | 351 | 358 | 365 | 372 | 379 | 386 |
| 72 | 140 | 147 | 154 | 162 | 169 | 177 | 184 | 191 | 199 | 206 | 213 | 221 | 228 | 235 | 242 | 250 | 258 | 265 | 272 | 279 | 287 | 294 | 302 | 309 | 316 | 324 | 331 | 338 | 346 | 353 | 361 | 368 | 375 | 383 | 390 | 397 |
| 73 | 144 | 151 | 159 | 166 | 174 | 182 | 189 | 197 | 204 | 212 | 219 | 227 | 235 | 242 | 250 | 257 | 265 | 272 | 280 | 288 | 295 | 302 | 310 | 318 | 325 | 333 | 340 | 348 | 355 | 363 | 371 | 378 | 386 | 393 | 401 | 408 |
| 74 | 148 | 155 | 163 | 171 | 179 | 186 | 194 | 202 | 210 | 218 | 225 | 233 | 241 | 249 | 256 | 264 | 272 | 280 | 287 | 295 | 303 | 311 | 319 | 326 | 334 | 342 | 350 | 358 | 365 | 373 | 381 | 389 | 396 | 404 | 412 | 420 |
| 75 | 152 | 160 | 168 | 176 | 184 | 192 | 200 | 208 | 216 | 224 | 232 | 240 | 248 | 256 | 264 | 272 | 279 | 287 | 295 | 303 | 311 | 319 | 327 | 335 | 343 | 351 | 359 | 367 | 375 | 383 | 391 | 399 | 407 | 415 | 423 | 431 |
| 76 | 156 | 164 | 172 | 180 | 189 | 197 | 205 | 213 | 221 | 230 | 238 | 246 | 254 | 263 | 271 | 279 | 287 | 295 | 304 | 312 | 320 | 328 | 336 | 344 | 353 | 361 | 369 | 377 | 385 | 394 | 402 | 410 | 418 | 426 | 435 | 443 |

Courtesy of the National Heart, Lung, and Blood Institute.

# APPENDIX 4 Review for the Registration Examination for Dietitians

*Karlyn Grimes, MS, RD, LDN*



## Registration Examination Test Specifications (Effective January 1, 2012)

The Registration Examination for Dietitians is designed to evaluate a dietitian's ability to perform at the entry level. The content domains and assigned weights reflect the results of the 2010 Dietetics Practice Audit. The Registration Examination for Dietitians study outline is located at http://www.cdrnet.org/vault/2459/web/files/2011%20RD%20Study%20Outline%20FINAL.pdf. Following are the four domains covered in the registration examination and the assigned weights for each domain. The focus of this examination review is Domain II, Nutrition Care for Individuals and Groups. Domain II relates closely to the material covered in this textbook, and this domain represents the highest assigned weight of all domains (50%).

**DOMAIN I Principles of Dietetics (12%)**

- Food science and nutrient composition of foods
- Nutrition and supporting sciences
- Education and communication
- Research
- Management concepts

**DOMAIN II Nutrition Care for Individuals and Groups (50%)**

- Screening and assessment
- Diagnosis
- Planning and intervention
- Monitoring and evaluation

**DOMAIN III Management of Food and Nutrition Programs and Services (21%)**

- Function of management
- Human resources
- Financial management
- Marketing and public relations
- Quality improvement

**DOMAIN IV Foodservice Systems (17%)**

- Menu planning
- Procurement, production, distribution, service
- Safety and sanitation
- Equipment and facility planning
- Sustainability

## Medical Nutrition Therapy for Various Chronic Diseases and Conditions Throughout the Life Cycle

The following list of common chronic diseases and medical conditions is well known to the world of medical nutrition therapy. You may encounter a number of these conditions throughout your career in the field of dietetics. It is highly recommended that you become familiar with the basic nutritional recommendations for these conditions.

### Concepts to Consider When Applying Medical Nutrition Therapy to Chronic Disease

- Knowledge of the etiology and physiology of each disease or condition
- Side effects or complications that can influence the nutritional status of individuals possessing each disease or condition
- Potential side effects from drugs commonly used for each disease or condition
- Medical nutrition therapy recommendations for each disease or condition (i.e., adjustments in calorie, carbohydrate, protein, fat, and vitamin and mineral recommendations)

### Common Chronic Diseases and Conditions

- AIDS/HIV infection
- Allergies, food and environmental
- Burns
- Cancer (all types)
- Cerebrovascular accident (stroke)
- Celiac disease
- Chronic obstructive pulmonary disease
- Congestive heart failure
- Coronary artery disease, arteriosclerosis, atherosclerosis
- Crohn's disease
- Cystic fibrosis
- Diabetes, type 1 and type 2
- Diverticular disease
- Disordered eating, such as anorexia and bulimia nervosa
- Failure to thrive
- Gastrectomy
- Gastroesophageal reflux disease
- Hepatic cirrhosis hepatitis
- Hypercholesterolemia/hyperlipidemia
- Hypertension
- Hyperthyroidism/hypothyroidism
- Lactose intolerance
- Myocardial infarction
- Obesity
- Osteoporosis
- Pancreatitis
- Peripheral vascular disease
- Renal disease, acute, chronic, end stage
- Short bowel syndrome
- Trauma
- Ulcerative colitis

## Review of Common Medical Abbreviations

When evaluating medical records, it is essential to recognize and interpret common medical abbreviations to fully understand the scope of an individual's medical condition. The following table defines a variety of abbreviations used in medical dictation.

| Abbreviation | Meaning |
|---|---|
| a | Before |
| a.c. | Before meals |
| ad lib | As desired |
| ADLs | Activities of daily living |
| AF | Osmotic fluid, acid fat |
| AKA | Above knee amputation |
| Alb | Albumin |
| Amb | Ambulate, ambulatory |
| AODM | Adult onset diabetes mellitus |
| AP | Anteroposterior |
| ARF | Acute renal failure |
| ASCVD | Arteriosclerotic cardiovascular disease |
| ASHD | Arteriosclerotic heart disease |
| BEE | Basal energy expenditure |
| b.i.d. | Twice a day |
| BKA | Below knee amputation |
| BMR | Basal metabolic rate |
| BP | Blood pressure |
| Bx | Biopsy |
| CAD | Coronary artery disease |
| CAT, CT | Computed tomography |
| CBC | Complete blood count |
| CCU | Coronary care unit |
| CF | Cystic fibrosis |
| CHD | Coronary heart disease |
| CHF | Congestive heart failure |

| Abbreviation | Meaning |
|---|---|
| COPD | Chronic obstructive pulmonary disease |
| CRF | Chronic renal failure |
| CVA | Cerebrovascular accident |
| CXR | Chest X-ray |
| DAT | Diet as tolerated |
| DKA | Diabetic ketoacidosis |
| DM | Diabetes mellitus |
| DOB | Date of birth |
| DOE | Dyspnea on exertion |
| Dx | Diagnosis |
| ECG, EKG | Electrocardiogram |
| EEG | Electroencephalogram |
| e.g. | For example |
| ETOH | Ethanol |
| ESRD | End-stage renal disease |
| FBS | Fasting blood sugar |
| FTT | Failure to thrive |
| F/U | Follow-up |
| FUO | Fever of undetermined origin |
| Fx | Fracture |
| GI | Gastrointestinal |
| H/H | Hematocrit/hemoglobin |
| Hct | Hematocrit |
| Hb | Hemoglobin |
| HBP | High blood pressure |
| HEENT | Head, ears, eyes, nose, throat |
| HOB | Head of bed |
| H&P | History and physical examination |
| HR | Heart rate |
| HS | Hour of sleep (bedtime) |
| HTN | Hypertension |
| IBW | Ideal body weight |
| ICU | Intensive care unit |
| IDDM | Insulin-dependent diabetes mellitus |
| i.e. | That is |
| I&O | Intake and output |
| IVF | Intravenous fluid |
| kg | Kilogram |
| KUB | Kidney, ureter, bladder |
| LBW | Low body weight |
| LLE | Left lower extremity |
| LLL | Left lower lobe |
| LLQ | Left lower quadrant |
| LML | Left mid lobe |
| LMP | Last menstrual period |
| LUE | Left upper extremity |
| MAC | Midarm circumference |
| MCH | Mean corpuscular volume |
| MI | Myocardial infarction |
| MCH | Mean corpuscular hemoglobin |
| MCHC | Mean corpuscular hemoglobin concentration |
| NAD | No active disease |
| NIDDM | Non-insulin-dependent diabetes mellitus |
| NKA | No known allergies |
| NKDA | No known drug allergies |
| NPO | Nothing by mouth |
| N&V | Nausea and vomiting |
| OB | Obstetrics |
| OOB | Out of bed |
| OR | Operating room |
| OS | Left eye |
| OU | Both eyes, each eye |
| p.c. | After meals |
| PCM | Protein calorie malnutrition |
| PID | Pelvic inflammatory disease |
| PKU | Phenylketonuria |
| PMH | Past medical history |
| p.o. | By mouth, orally, post operative, phone order |
| PPN | Peripheral parenteral nutrition |
| p.r.n. | As needed, whenever necessary |
| PTA | Prior to admission |
| PUD | Peptic ulcer disease |
| PVD | Peripheral vascular disease |
| q | Every |
| q.d. | Every day |
| q.h. | Every hour |
| q.i.d. | Four times a day |
| q.o.d. | Every other day |
| RDA | Recommended Dietary Allowance |
| RLE | Right lower extremity |
| RLL | Right lower lobe |
| RLQ | Right lower quadrant |
| RO, R/O | Rule out |

(*continued*)

| Abbreviation | Meaning |
|---|---|
| ROM | Range of motion |
| RUE | Right upper extremity |
| RUL | Right upper lobe |
| RUQ | Right upper quadrant |
| Rx | Prescription |
| s | Without |
| SBS | Short bowel syndrome |
| SGA | Small for gestational age |
| SOAP | Subjective, objective, assessment, plan |
| SOB | Shortness of breath |
| S/P | Status post |
| S&S | Signs and symptoms |
| Sx | Symptoms |
| TB | Tuberculosis |
| TF | Tube feeding |
| TG, trig. | Triglycerides |
| TIA | Transient ischemic attack |
| TIBC | Total iron binding capacity |
| t.i.d. | Three times a day |
| TPN | Total parenteral nutrition |
| TPR | Temperature, pulse, respiration |
| TSH | Thyroid-stimulating hormone |
| Tx | Treatment |
| UBW | Usual body weight |
| URI | Upper respiratory infection |
| US | Ultrasound |
| UTI | Urinary tract infection |
| VS | Vital signs |
| WDWN | Well developed well nourished |
| WNL | Within normal limits |
| w/o | Without |
| # | Number or pounds |

***Note:*** Abbreviations vary in different institutions.

## Summary of Common Modified and Mechanically Altered Diets

Medical facilities providing nourishment to patients generally offer a variety of diets differing in nutrient and textural quality. The precise name of each diet can vary from institution to institution, but generally common dietary principles apply to those diets geared at managing specific acute and chronic diseases and conditions.

| Diet | Description | Indications | Diet Principles/Comments |
|---|---|---|---|
| **Modified Diets** | | | |
| Regular/house | Provides a well-balanced diet without restrictions or texture modifications. This diet can be adjusted to provide small or large portions. | Offered to healthy persons with no known health or medical contraindications. | A nutritionally balanced diet providing the amounts of energy, protein, vitamins, minerals, and other nutrients sufficient to meet the needs of all individuals age 4 years and older. |
| No added salt (NAS) | Based on the house diet with moderate restrictions in the sodium content of the meals. The diet eliminates or limits foods high in sodium, including processed foods. Generally, delivers a maximum of 4 g of sodium per day. | Recommended for individuals with essential hypertension, fluid retention, impaired liver or kidney function, and cardiovascular disease. The appropriate degree of sodium restriction depends on disease severity, presence and amount of edema, overall organ function, and current drug therapy. | The primary purpose of a sodium-restricted diet is to restore normal sodium balance to the body by effecting loss of excess sodium and water from extracellular compartments. |

| Diet | Description | Indications | Diet Principles/Comments |
|---|---|---|---|
| **Modified Diets** | | | |
| Two-gram sodium | Based on the NAS diet with greater restrictions on the sodium content of the diet. Primary restrictions include foods high in sodium, including processed foods, fast foods, convenience foods, canned foods, and sodium-containing compounds used in food manufacturing, such as monosodium glutamate (MSG), baking powder, sodium chloride, baking soda, disodium phosphate, sodium propionate, and sodium benzoate. Salt packets are generally eliminated from all trays. | Recommended for individuals with the conditions listed under NAS diets who have advanced to a more severe state. Two-gram sodium diets are also beneficial for individuals with ascites associated with liver disease, congestive heart failure, and those receiving adrenocortical therapy. The appropriate level of sodium restriction depends on disease severity, presence and amount of edema, and current drug therapy. | The primary purpose of a sodium-restricted diet is to restore normal sodium balance to the body by effecting loss of excess sodium and water from extracellular compartments. |
| Calorie-controlled diets (1,200-, 1,500-, and 1,800-calorie diets) | A calorie-restricted version of the house diet providing ~45–65% of total calories from carbohydrates, 10–35% of total calories from protein, and 20–35% of total calories from fat, and adhering to dietary guidelines developed by the American Diabetes Association. Fat and sugar restrictions are commonly implemented to promote calorie control. | Recommended for individuals who are overweight, obese, and/or have a diagnosis of cardiovascular disease, pre-diabetes, or type 2 diabetes. | A calorie-restricted diet that is nutritionally adequate while providing suboptimal calories. Calorie restriction encourages the body to utilize fat stores to meet daily energy needs. Generally, vitamin and mineral supplements are recommended for diets providing fewer than 1,500 calories. |
| No concentrated sweets (NCS) | NCS dietary patterns provide most foods allowed on the regular/house diet, but aim to provide calorie- and sugar-restricted dietary items, including unsweetened fruit-based desserts, sugar substitutes, diet syrup, and diet jelly. Skim milk often replaces whole milk. | Recommended for individuals with type 1 or type 2 diabetes, gestational diabetes, impaired glucose tolerance, or impaired fasting glucose. | Prescribed for individuals with a stable diabetic condition or with a need for a mild calorie restriction. The goals of the diet are to promote weight management and to achieve and maintain optimal blood glucose levels. |
| Renal diet: predialysis | Predialysis meal patterns aim to reduce the workload on compromised kidneys to prevent acceleration of nephron damage. Also, they reduce the incidence of renal osteodystrophy, control dietary phosphorus intake, and maintain a normal serum calcium–phosphorous product. Can help maintain lean body mass, normal extracellular fluid volume, pH, and osmolality; postpone initial dialysis; control blood pressure; and manage glucose intolerance. | Intended for individuals with chronic renal insufficiency prior to implementation of dialysis. | The goals of this diet are to meet nutritional requirements and prevent malnutrition in patients with impaired renal function; to maintain acceptable blood chemistries, blood pressure, and fluid status; and to control edema and electrolyte balance. Basic dietary guidelines: *Energy,* 30–40 kcal/kg; *Protein,* 0.6–0.8 g/kg; *Fluid,* 500 mL + previous days urinary and other losses; *Sodium,* 1–3 g/day; *Potassium,* unrestricted unless serum K > 5.5 mEq/L; *Phosphorus,* 600–800 mg/day (Ca/K product no higher than 70); *Calcium,* 1,200–1,600 mg/day. |

(*continued*)

| Diet | Description | Indications | Diet Principles/Comments |
|---|---|---|---|
| **Modified Diets** | | | |
| Renal diet: hemodialysis | A hemodialysis diet strives to reduce the workload of diseased kidneys, replace substances lost during dialysis, prevent acceleration of nephron damage produced by excessive protein intake, prevent renal osteodystrophy, control dietary phosphorus intake and maintain a normal serum calcium–phosphorous product, maintain lean body mass and optimal nutritional status, maintain normal extracellular fluid volume, maintain pH, maintain osmolality, control blood pressure, and manage glucose intolerance. | Recommended for individuals with end-stage renal disease requiring hemodialysis. | The goals of this diet are to meet nutritional requirements and prevent malnutrition in patients with impaired renal function, to maintain acceptable blood chemistries, blood pressure, and fluid status, and to control edema and electrolyte balance. Nutrients of top nutritional concern include protein (higher intakes are recommended once hemodialysis is initiated), phosphorus, potassium, sodium, and fluids. Basic dietary guidelines: *Energy,* 30–35 kcal/kg if 60+ years and 35 kcal/kg if younger than 60 years; *Protein,* 1.0–1.2 g/kg at least 50% biological value; *Fluid,* 750–1,500 mL/day or 500 mL and urinary losses; *Sodium,* 1–3 g/day; *Potassium,* 1,500–3,000 mg/day; *Phosphorus,* 600–1,200 mg/day (Ca/P product no higher than 70); *Calcium,* 1–2 g/day. |
| High fiber | A general diet with emphasis on fiber-rich foods, which include fruits, vegetables, legumes, and whole grain rice, breads, and cereals. Intake of 20–35 g of fiber is recommended each day. When increasing fiber intake, it is recommended that it is done gradually, and plenty of fluids are emphasized. | Intended for prevention or treatment of various gastrointestinal, cardiovascular, and metabolic conditions, including diverticular disease, colon cancer, diabetes, constipation, irritable bowel syndrome, Crohn's disease, hypercholesterolemia, and obesity. | To increase fecal bulk, promote regularity, normalize serum lipid level, and blunt the postprandial blood glucose response. |
| Low fiber/low residue | To prevent the formation of an obstructing bolus from high-fiber foods in patients with narrowed intestinal or esophageal lumens or to reduce the frequency of painful stools in acute phases of diverticulitis or inflammatory bowel disease. Also, used as a postoperative diet as a step toward a regular/house diet. | Low-fiber diets are indicated during acute phases of diverticulosis, infectious enterocolitis, ulcerative colitis, or Crohn's disease when the bowel is markedly inflamed, fixed radiologic strictures are present, or the intestinal lumen is narrowed. The diet may also be useful for a short period in the transition between a completely liquid diet with patients convalescing from surgery, trauma, or other illnesses. | Fiber-rich foods are avoided in this diet, including fruits, vegetables, legumes, and whole grain rice, breads, and cereals. If a low-residue diet is indicated, all fruits and vegetables, including prune juice, should be eliminated except white potatoes without skin and strained fruit and vegetable juices. Because milk may contribute to fecal residue, certain patients may benefit from restricting their milk intake to 2 cups per day. |
| Fat and cholesterol controlled (step 1 and step 2 diets) | A diet that limits total fat intake to less than 30% of total calories and that limits saturated fat and cholesterol to varying amounts depending on the severity of lipid dysfunction. There are two phases, known as step 1 and step 2 diets. | Intended for individuals with coronary heart disease, hypercholesterolemia, hyperlipidemia, dyslipidemia, dysbetalipoproteinemia, gallbladder disease, abnormalities in fat digestion and absorption, and/or obesity in an attempt to normalize blood lipid levels, reduce gallbladder exacerbations, and manage excess body weight. | *Step 1:* Less than 30% of kcal from fat, 8–10% from saturated fatty acids (SFAs), and 300 mg/day from cholesterol. *Step 2:* Same as step 1, but 7% from SFAs and 200 mg/day from cholesterol. Emphasis on grains, cereals, legumes, vegetables, fruits, lean meats, poultry, fish, and nonfat dairy products. Restriction of animal fats recommended, meat is limited to 5–6 oz/day and eggs 4×/week. Sodium is restricted to 2,400 mg/day. |

| Diet | Description | Indications | Diet Principles/Comments |
|---|---|---|---|
| **Modified Diets** | | | |
| Lactose restricted/ lactose free | A diet limited in its content of the simple sugar (disaccharide) called lactose. It provides an amount of lactose small enough to avoid recurrence of symptoms in mild forms of lactose intolerance, usually less than 8 to 12 g of lactose daily. | Intended for lactose-deficient individuals to prevent excess gas, bloating, cramping, and diarrhea caused by foods containing the simple sugar lactose. A patient with proven lactose intolerance who does not experience relief of abdominal pain and diarrhea may have lactose intolerance secondary to another disorder that requires treatment, such as celiac disease, regional enteritis, or postgastrectomy dumping syndrome. | Small amounts of dairy foods in divided doses are recommended because individuals vary in their tolerance of lactose-containing foods. Tolerance for lactose may decrease with age and degree of gastrointestinal disease. Nutritionally adequate meal pattern as long as dairy foods are not avoided or lactose-free or lactase-containing products are used. |
| High calorie, high protein | To provide energy, protein, and additional nutrients in excess of usual requirements to improve overall nutritional status, prevent malnutrition, promote weight gain, meet need for increased nutrients, and optimize the ability to respond to medical treatment. | Recommended for persons with acute or chronic wasting conditions such as cancer, HIV infection/AIDS, chronic gastrointestinal problems, burns, wounds, trauma, renal disease (dialysis), protein-calorie malnutrition, cystic fibrosis, failure to thrive, and in preparation for and recovery from surgery. | Dietary patterns focus on calorie-dense and protein-dense foods. The diet often exceeds 30% of total calories from fat to increase calorie density of the total diet. Generally, the diet should provide at least 120–150% of Recommended Dietary Allowances for energy and protein. Small frequent feedings of calorie- and nutrient-dense foods are recommended. |
| Phenylalanine restricted | A diet in which the intake of phenylalanine is limited to a prescribed level governed by individual tolerance. Blood levels of phenylalanine rise because of a defect in or absence of the enzyme phenylalanine hydroxylase. | For individuals with phenylketonuria (PKU) in an attempt to control blood phenylalanine levels to allow the greatest development of intellectual potential. | The goal of this diet is to provide enough protein, tyrosine, and energy for the promotion of growth and development and to meet vitamin, mineral, and fluid needs of the individual. |
| Gluten free | The diet is intended to eliminate proteins referred to as gluten found in wheat, rye, oats, and barley to ameliorate symptoms such as diarrhea, abdominal distention, flatulence, steatorrhea, failure to thrive, chronic pain, and anemia associated with gluten/gliadin intolerance. | For individuals with celiac disease, nontropical sprue, or gluten-sensitive enteropathy. | A diet free of gliadin or glutens, such as those in wheat, rye, oats, and barley protein and malt. Gliadin and glutens are toxic to individuals with gluten-sensitive enteropathy. Constipation may be encountered on this diet because wheat fiber is excluded from the diet. Alternative sources of insoluble fiber and roughage from fruits and vegetables are needed to prevent this problem. |
| **Transitional Diets** | | | |
| Clear liquid | A diet that includes only foods that are clear and liquid at body temperature, such as fat-free broth, bouillon, coffee, tea, decaffeinated coffee, strained fruit juices, flavored gelatin, carbonated beverages, popsicles, fruit ices, and hard candies. | The diet is intended to provide an oral source of fluids and a small amount of energy and electrolytes. It is used to prevent dehydration and to keep colonic residue to a minimum. It is often administered in preparation for bowel surgery or before colonoscopic examination or as a transitional diet from intravenous feeding or acute gastrointestinal disturbances. | This diet should not be used for more than 24 hours because it is inadequate in calories and most nutrients even when supplemented with low-residue, liquid-protein products. |

(*continued*)

| Diet | Description | Indications | Diet Principles/Comments |
|---|---|---|---|
| **Transitional Diets** | | | |
| Full liquid | A diet consisting of foods that are liquid at body temperature, supplemented with commercial liquid supplements. | A full-liquid diet is indicated after oral or plastic surgery of the face and neck or in other postoperative states such as in esophageal surgery. It may also be used as a transition between a clear liquid diet and a fiber-restricted or regular diet. It is used in conjunction with dilatation procedures in the management of esophageal stricture, after mandibular fractures, or with any patient who cannot chew properly or who has an esophageal or pharyngeal disorder that interferes with the normal handling of solid foods. | If planned properly, the diet can be nutritionally adequate. A variety of foods may be included, such as fruit and vegetable juices, strained hot cereals, broths, milk, eggs, egg substitutes, commercial liquid formulas, and high-protein broths, cereals, puddings, and gelatins. |
| **Texture-Modified Diets** | | | |
| Mechanical soft/ground | To provide texture-modified food that requires minimal chewing. | This texture-modified diet is indicated for individuals with compromised chewing and/or swallowing ability, dental problems, and/or an edentulous status. This diet is often well tolerated by individuals after head and neck surgery as they transition back to a regular diet. | The MS/ground diet includes foods modified in texture, such as chopped, ground, mashed, and puréed foods, that allow for ease of mastication. Certain raw and hard foods are eliminated from this diet. Gravies and sauces are recommended to moisten the foods. |
| Dysphagia (dysphagia regular, dysphagia ground, dysphagia purée) | A diet intended to provide nutrition in a form that fits the specific anatomic and functional needs of the patient, to maintain or improve nutritional status, to avoid or limit possible adverse reactions such as aspiration that result from attempts to feed the dysphagic patient, to provide adequate hydration in the patient who cannot handle thin liquids, and to help the patient achieve the highest level of consistency tolerance possible. | Recommended for individuals with dysphagia, swallowing impairment, neurologic illness, and those receiving surgical procedures and anticancer therapy that may cause dysphagia. | The diet includes semisolid foods that form a cohesive bolus. Spoon-thick liquids and medium-thick liquids are most likely to be tolerated. The best consistency depends on the individual patient. Thin liquids, foods that fall apart, and sticky or bulky foods are poorly tolerated. Generally, a speech-language pathologist performs a speech evaluation and uses a test tray containing foods of varying consistency to determine which foods and textures are best tolerated by the individual. |
| Puréed | Same as above. Thick, smooth, homogenous, semiliquid textures. | Same as above. Severely reduced oral preparatory stage abilities, impaired lip and tongue control, delayed swallowing reflex triggering, oral hypersensitivity, reduced pharyngeal peristalsis, and/or cricopharyngeal dysfunction. | Same as above. Spoon-like or pudding-like consistency. No coarse textures, nuts, or raw fruits and vegetables. |

### Analysis of Protein and/or Calorie Supplements

| Product* | Indications for Use | Caloric Density (kcal/cc)† | Carbohydrate (g/1,000 cc) | Protein (g/1,000 cc) | Fat (g/1,000 cc) |
|---|---|---|---|---|---|
| Ensure | Complete balanced nutrition | 1.06 kcal/mL | 169 g/L | 37.2 g/L | 25.9 g/L |
| Ensure Plus | High-calorie liquid nutrition | 1.5 kcal/mL | 200 g/L | 54.9 g/L | 53.3 g/L |
| Carnation Instant Breakfast | Supplement | 0.93 kcal/mL | 167 g/L | 51 g/L | 21 g/L |
| Diet Carnation Instant Breakfast | General nutrition | 0.7 kcal/mL | 136 g/L | 68 g/L | 28 g/L |
| Glucerna | Glucose intolerance | 1.0 kcal/mL | 100 g/L | 45 g/L | 47.5 g/L |
| Healthshakes | Supplement | 280 (6 oz) | | 9 (6 oz) | |
| Jevity | Isotonic with fiber | 1.06 kcal/mL | 154.4 g/L | 44.3 g/L | 34.7 g/L |
| Nepro | Dialysis patient, chronic/ acute renal failure | 2 kcal/mL | 215.2 g/L | 69.9 g/L | 95.6 g/L |
| Osmolite | Isotonic | 1.06 kcal/mL | 151 g/L | 37.1 g/L | 34.7 g/L |
| Osmolite HN | High nitrogen, isotonic | 1.06 kcal/mL | 143.9 g/L | 44.3 g/L | 34.7 g/L |
| PediaSure | Children | 1.0 kcal/mL | 109 g/L | 30 g/L | 49.7 g/L |
| Promote | High protein | 1.0 kcal/mL | 130 g/L | 62.5g/L | 26 g/L |
| Pulmocare | Cardiac patient | 1.5 kcal/mL | 105 g/L | 62.6 g/L | 93.3 g/L |
| 2 Cal HN | High calorie, high protein | 2.0 kcal/mL | 217.3 g/L | 83.7 g/L | 90.9 g/L |
| Suplena | Low protein, complete, for dialyzed patient with renal failure | 2.0 kcal/mL | 256 g/L | 30 g/L | 96 g/L |

*Most of these products are manufactured by Abbott. Information about these products can be obtained by visiting the manufacturer's website: http://abbottnutrition.com.
†Remember when calculating grams per cc that 1,000 cc = 1,000 mL = 1 liter; 1 oz = 30 cc or 30 mL; 120 cc = 4 oz; 240 cc = 8 oz.

## Review of Common Laboratory Values

The following chart presents common laboratory values that you will encounter during your career as a dietitian. It provides the significance of the particular laboratory measurement, the normal laboratory value range, and possible explanations for laboratory values above or below the expected values.

| Laboratory Measurement | Significance of the Measurement | Normal Laboratory Value Range | Conditions Related to Elevated Readings | Conditions Related to Low Readings |
|---|---|---|---|---|
| Albumin, serum | Chief blood protein used to assess protein and nutritional status. Also used as a measure of hepatic function. | Adult: 3.5–5 g/ dL *Or* 35–50 g/L | Dehydration; albumin in urine signals kidney disease. | Malnutrition, pregnancy, liver disease, protein-losing enteropathy, protein-losing nephropathy, overhydration, increased capillary permeability, inflammatory disease, $B_{12}$ and familial idiopathic dysproteinemia. |

(*continued*)

| Laboratory Measurement | Significance of the Measurement | Normal Laboratory Value Range | Conditions Related to Elevated Readings | Conditions Related to Low Readings |
|---|---|---|---|---|
| Vitamin $B_{12}$, serum | To assess vitamin $B_{12}$ status. | 100–700 pg/mL *Or* 74–517 pmol/L | Leukemia, polycythemia vera, severe liver dysfunction, myeloproliferative disease. | Pernicious anemia, malabsorption syndrome, inflammatory bowel disease, intestinal worm infestation, atopic gastritis, Zollinger-Ellison syndrome, large proximal gastrectomy, resection of terminal ileus, achlorhydria, pregnancy, folic acid deficiency, and vitamin C deficiency. |
| Blood urea nitrogen (BUN) | Measures the amount of urea nitrogen. Directly related to the metabolic function of the liver and the excretory function of the kidney. | Adult: 10–20 mg/dL *Or* 3.6–7.1 mmol/L<br>Child: 5–18 mg/dL<br>Elderly: may be slightly higher than adult. | Possible renal failure/disease, gastrointestinal bleeding, congestive heart failure, increased protein intake, sepsis, insufficient renal blood supply, blocked urinary tract, or dehydration. | Possible liver disease, malnutrition, anabolic steroids, overhydration, negative N balance, pregnancy, or nephrotic syndrome. |
| Chloride, serum | Performed as part of multiphasic testing of electrolyte status. Provides feedback on acid–base balance and hydration status. | Adult/elderly: 90–110 mEq/L *Or* 98–106 mmol/L<br>Children: 90–110 mEq/L | Dehydration, renal tubular acidosis, Cushing syndrome, eclampsia, multiple myeloma, kidney dysfunction, metabolic acidosis, anemia, hyperventilation, or hyperparathyroidism. | Overhydration, congestive heart failure, vomiting, chronic respiratory acidosis, Addison disease, burns, metabolic alkalosis, diuretic therapy, hypokalemia, aldosteronism, respiratory acidosis, or salt-losing nephritis. |
| Cholesterol, serum | To help assess the risk of arteriosclerotic heart disease. Done as part of lipid profile testing. | <200 mg/dL *Or* < 5.2 mmol/L<br>Younger than age 20 years: <180 mg/dL | Hypercholesterolemia, hyperlipidemia, hypothyroidism, uncontrolled diabetes, pregnancy, nephrotic syndrome, high-cholesterol diet, stress, hypertension, or myocardial infarction. | Heredity, malnutrition. |
| Creatinine, serum | Allows for assessment of the amount of lean body mass (fat-free mass) and can identify impaired renal function. | Female: 0.5–1.1 mg/dL *Or* 44–97 mmol/L<br>Male: 0.6–1.2 mg/dL | Glomerulonephritis, acute tubular necrosis, urinary tract obstruction, reduced renal blood flow, shock, dehydration, atherosclerotic, diabetic nephropathy, nephritis, or acromegaly. | Debilitation, decreased muscle mass, muscular dystrophy, or myasthenia gravis. |
| Ferritin, serum | A test for iron status, considered the best test for detecting the early stages of anemia because it is an indicator of an individual's iron stores. | Male: 12–300 µg/mL<br>Female: 10–150 µg/mL | Hemochromatosis, hemosiderosis, megaloblastic anemia, hemolytic anemia, alcoholic/inflammatory hepatocellular disease, or advanced cancer. | Severe protein deficiency, iron-deficiency anemia, or hemodialysis. |
| Folic acid, serum | To identify hemolytic disorders and detect anemia caused by folic acid deficiency. | 5–20 µg/mL | Pernicious anemia, vegetarianism, or recent massive blood transfusion. | Folic acid deficiency anemia, hemolytic anemia, malnutrition, malabsorption syndrome, malignancy, pregnancy, alcoholism, liver disease, anorexia, or chronic renal disease. |

| Laboratory Measurement | Significance of the Measurement | Normal Laboratory Value Range | Conditions Related to Elevated Readings | Conditions Related to Low Readings |
|---|---|---|---|---|
| Glucose, serum | Measurement of the amount of glucose in the blood. A true elevation indicates diabetes. | 2 years to adult: 70–105 mg/dL *Or* 3.9–5.8 mmol/L | Diabetes mellitus, acute stress response, Cushing syndrome, chronic renal failure, glucagonoma, acute pancreatitis, diuretic therapy, or corticosteroid therapy. | Insulinoma, hypothyroidism, hypopituitarism, Addison disease, extensive liver disease, insulin overdose, or starvation. |
| Glycosylated hemoglobin (HbA1c) | Test used to monitor diabetes treatment and dietary compliance over the previous 3 months. Provides an accurate long-term index of the patient's average blood glucose levels. | Good: 7%<br>Fair: 10%<br>Poor: 13–20% | Newly diagnosed diabetes, poorly controlled diabetes, acute stress response, Cushing syndrome, pregnancy, glucagonoma, corticosteroid therapy, or splenectomized patient. | Hemolytic anemia, chronic blood loss, or chronic renal failure. |
| Hematocrit (Hct) | Measure of the percentage of the total blood volume that is made by the red blood cell. | Male: 42–52%<br>Female: 37–47%<br>Pregnant women: > 33% | Congenital heart disease, polycythemia vera, severe dehydration, erythrocytosis, severe diarrhea, eclampsia, burns, dehydration, or chronic obstructive pulmonary disease. | Anemia, hyperthyroidism, cirrhosis, hemolytic reaction, hemorrhage, dietary deficiency, bone marrow failure, normal pregnancy, rheumatoid arthritis, multiple myeloma, malnutrition, leukemia, or hemoglobinopathy. |
| Hemoglobin (Hb) | Measure of total amount of hemoglobin in the peripheral blood, which reflects the number of red blood cells. | Male: 14–18 g/dL<br>Female: 12–16 g/ dL | Congenital heart disease, polycythemia vera, hemoconcentration of the blood, chronic obstructive pulmonary disease, congestive heart failure, high altitudes, severe burns, or dehydration. | Anemia, severe hemorrhage, hemolysis, cancer, nutritional deficiencies, lymphomas, systemic lupus erythematosus, sarcoidosis, kidney disease, sickle cell anemia, or neoplasia. |
| High-density lipoprotein (HDL) ("healthy") cholesterol | Measurement identifying persons at risk for developing heart disease and to monitor therapy effectiveness if abnormalities are found. | Male: > 45 mg/dL<br>Female: > 55 mg/dL | Familial HDL, lipoproteinemia, or excessive exercise. | Familial low HDL, hepatocellular disease, hepatitis, cirrhosis, hypoproteinemia, nephrotic syndrome, or malnutrition. |
| Low-density lipoprotein (LDL) ("lethal") cholesterol | Same as above. | 60–80 mg/dL | Familial LDL, lipoproteinemia, nephrotic syndrome, glycogen storage disease, hypothyroidism, alcohol consumption, or chronic liver disease. | Familial hypolipoproteinemia, hypoproteinemia, malabsorption, severe burns, malnutrition, or hyperthyroidism. |
| Mean corpuscular volume (MCV) | A test routinely performed as part of complete blood cell count (CBC). Detects anemia and provides information about red blood cell size. | Adult/elderly/child: 80–90 $\mu m^3$ | Liver disease, antimetabolic therapy, alcoholism, pernicious anemia, or folic acid deficiency. | Iron-deficiency anemia or thalassemia. |
| Mean corpuscular hemoglobin (MCH) | Routinely performed as part of complete blood count. Provides information about the weight of the red blood cell. | Adult/elderly/child: 27–31 pg | Macrocytic anemia. | Microcytic anemia or hypochromic anemia. |
| Mean corpuscular hemoglobin concentration (MCHC) | Routinely performed as part of complete blood cell count. Provides information on the hemoglobin concentration of red blood cells. | Adult/elderly/child: 32–36 g/dL *or* 32–36% | Spherocytosis, intravascular hemolysis, or cold agglutinins. | Iron-deficiency anemia or thalassemia. |

(*continued*)

| Laboratory Measurement | Significance of the Measurement | Normal Laboratory Value Range | Conditions Related to Elevated Readings | Conditions Related to Low Readings |
|---|---|---|---|---|
| Potassium, serum | Indicator of hyperkalemia and hypokalemia. | Adult/elderly: 3.5–5 mEq/L<br>Child: 3.4–7 mEq/L | Excessive dietary intake or intravenous intake, acute or chronic renal failure, hypoaldosteronism, aldostero–inhibiting diuretics, crush injuries, hemolysis, transfusion of hemolyzed blood, infection, acidosis, or dehydration. | Inadequate dietary intake, suboptimal intravenous intake, burns, diuretics, gastrointestinal disorders, diarrhea, vomiting, hyperaldosteronism, Cushing syndrome, renal tubular acidosis, licorice ingestion, insulin administration, glucose administration, ascites, renal artery stenosis, or cystic fibrosis. |
| Sodium, serum | A measurement used to determine extracellular osmolality. | Adult/elderly: 136–145 mEq/L<br>Child: 136–145 mEq/L | Excessive dietary intake, excessive sodium in intravenous fluids, Cushing syndrome, hyperaldosteronism, excessive sweating, extensive thermal burns, diabetes insipidus, or osmotic diuresis. | Inadequate dietary intake, suboptimal intravenous intake, Addison disease, diarrhea, vomiting, diuretic administration, chronic renal insufficiency, excessive oral water intake, or congestive heart failure. |
| Thyroid-stimulating hormone (TSH) | A measurement used to differentiate primary hypothyroidism from secondary hypothyroidism. | Increased thyroid function with administration of exogenous TSH.<br>Adult: 0.4–5 mIU/L | Abnormal findings are primary (thyroidal) hypothyroidism and secondary (hypothalamic-pituitary) hypothyroidism. | Hyperthyroidism caused by Graves' disease, a type of goiter, or a noncancerous tumor or by damage to the pituitary gland, and in individuals with an underactive thyroid gland but receiving too much thyroid hormone medication. |
| Thyroxine ($T_4$) | A direct measurement of the total amount of $T_4$ present in an individual's blood. T4 makes up nearly all of what we call thyroid, the hormone. | Adult male: 4–12 µg/dL<br>Adult female: 5–12 µg/dL<br>Adult > age 60: 5–11 µg/dL | Graves' disease, Plummer disease, acute thyroiditis, pregnancy, hepatitis, congenital hyperproteinemia, familial dysalbuminemia, or hyperthyroxinemia. | Cretinism, surgical ablation, myxedema, pituitary insufficiency, hypothalamic failure, protein malnutrition, iodine insufficiency, renal failure, Cushing syndrome, or cirrhosis. |
| Total iron, serum | Measurement of the quantity of iron bound to transferrin in the blood. | Male: 65–175 µg/dL Female: 50–170 µg/dL | Hemosiderosis, hemochromatosis, hemolytic anemia, hepatitis, hepatic necrosis, lead toxicity, iron poisoning, or massive transfusion. | Insufficient dietary iron, chronic blood loss, inadequate iron absorption, pregnancy (late), iron-deficiency anemia, neoplasia, chronic gastrointestinal blood loss, chronic hematuria, or chronic heavy physiologic or pathologic menstruation. |
| Total iron binding capacity (TIBC) | A measurement of all proteins available for binding mobile iron. | 250–420 µg/dL | Oral contraceptives, late pregnancy, polycythemia vera, or iron-deficiency anemia. | Hypoproteinemia, inflammatory diseases, cirrhosis, hemolytic anemia, pernicious anemia, or sickle cell anemia. |
| Total protein, serum | Measurement to help determine osmotic pressure within the vascular space. A measure of nutritional status. | Adult/elderly: 6.4–8.3 g/dL | Dehydration, possibly leukemia, an autoimmune disease, cirrhosis, kidney disease, chronic infection, hemolytic anemia, or Hodgkin's disease. | Malnutrition, pregnancy, liver disease, protein-losing enteropathies, protein-losing nephropathies, overhydration, or inflammatory disease. |

| Laboratory Measurement | Significance of the Measurement | Normal Laboratory Value Range | Conditions Related to Elevated Readings | Conditions Related to Low Readings |
|---|---|---|---|---|
| Transferrin, serum | Transferrin is a globulin protein bound to iron. Determines the amount of iron-binding proteins. | Adult male: 215–365 mg/dL<br>Adult female: 250–380 mg/dL | Pregnancy, estrogen therapy, inadequate iron stores, iron-deficiency anemia, acute hepatitis, polycythemia, or oral contraceptive use. | Malignancy, collagen vascular disease, liver disease, infection, malnutrition, iron-deficiency, sickle cell anemia, pernicious anemia, or hypoproteinemia. |
| Triglycerides, serum | Part of a lipid profile to assess risk of coronary and vascular disease. | Male: 40–160 mg/dL<br>Female: 35–135 mg/dL | Glycogen storage disease, hyperlipidemias, hypothyroidism, diet high in carbohydrates, poorly controlled diabetes, nephrotic syndrome, alcoholic cirrhosis, pregnancy, or cirrhosis. | Malabsorption syndrome, malnutrition, or hyperthyroidism. |
| Triiodothyronine ($T_3$) | Accurate measure of thyroid function. | Aged 20–50 years: 70–205 ng/dL<br>Older than age 50 years: 40–180 ng/dL | Graves' disease, Plummer disease, toxic thyroid adenoma, pregnancy, hepatitis, acute thyroiditis, or congenital hyperproteinemia. | Hypothyroidism, cretinism, myxedema, pituitary insufficiency, hypothalamic failure, protein malnutrition, iodine deficiency, or renal failure. |

## Review of Common Medications

| Medication | Intended Use | Common Side Effects | Drug–Nutrient Interactions or Dietary Considerations |
|---|---|---|---|
| Aldactone (spironolactone) | Diuretic, antihypertensive | Anorexia, thirst, dry mouth, nausea, vomiting, cramps, diarrhea. | Take with food, avoid excessive potassium intake, avoid natural licorice. |
| Aricept | Anti-Alzheimer's, cholinesterase inhibitor | Anorexia, decreased weight, nausea and vomiting, bloating, diarrhea, gastrointestinal bleeding, slowed heart beat or fainting. | Nausea, vomiting, gastrointestinal bleeding, stomach ulcers and weight loss were more common in individuals receiving 23 mg vs. 10 mg. |
| Atenolol | Antihypertensive, antiangina | Indigestion, constipation, dizziness, depression, fatigue, and dry mouth. Can mask hypoglycemia, so often contraindicated in individuals with diabetes. | Avoid natural licorice. Take with food. Avoid orange juice for 4 hours after taking this medication. |
| AZT Retrovir (zidovudine) | Anti-HIV medications that stop HIV from infecting uninfected cells but does not help infected cells | Long-term use associated with muscle loss. Other side effects include anemia; white blood cell depression; lip, mouth, and tongue sores; bone marrow damage; headaches; skin rash; itching; weakness; nervousness; dizziness; nausea; stomach pain; confusion; loss of speech or appetite; muscle aches; fever or sweating; sore throat; or abnormal bruising or bleeding. | Best taken on an empty stomach, but may be taken with food. Recommended that users take manganese, B vitamins, and vitamin E with Retrovir. |
| BuSpar | Antianxiety | Nausea, diarrhea, fainting, unsteady heart rate, depression, and lack of balance. | Caution with grapefruit juice. Avoid alcohol. |
| Calcijex/Calcitriol | Calcium regulator | Increased calcium absorption, anorexia, decreased weight, increased thirst, dry mouth, metallic taste, nausea and vomiting, constipation, diarrhea. | Used in individuals on dialysis. A low-phosphorus diet is indicated. |

(*continued*)

## Review of Common Medications *(continued)*

| Medication | Intended Use | Common Side Effects | Drug–Nutrient Interactions or Dietary Considerations |
|---|---|---|---|
| Cholestyramine | Antihyperlipidemic, antidiarrheal | May decrease absorption of fat, Ca, Fe, Zn, Mg, vitamins A, D, E, and K, medium-chain triglycerides, and folate. Anorexia, increased or decreased weight, belching, nausea and vomiting, dyspepsia, constipation, and diarrhea. | Decrease dietary fat and cholesterol intake, increase fluids, increase fiber, decrease calcium, if needed. Take before meals. Never take powder dry. |
| Cimetidine | Antisecretory, antiulcer, anti-GERD | Decreased Fe and vitamin $B_{12}$ absorption. Decreased gastric acid secretions, increased gastric pH, nausea and vomiting, diarrhea. | May need bland diet, limit caffeine, caution with alcohol. |
| Cipro | Antibiotic | Nausea and vomiting, abdominal pain, diarrhea. | Milk or yogurt decreases absorption and availability. Take with meals. Avoid milk, yogurt, and caffeine. |
| Colchicine | Used to prevent or treat attacks of gout (also called gouty arthritis) | *Common:* Diarrhea, nausea, vomiting, stomach pain. *Rare:* Black tarry stools; blood in urine or stools; difficulty in breathing when exercising; fever with or without chills; headache; large hive-like swellings on the face, eyelids, mouth, lips, and/or tongue; pinpoint red spots on skin; sores, ulcers, or white spots on lips or in mouth; sore throat; unusual bleeding or bruising; unusual tiredness or weakness. | Limit alcohol intake. Monitor appetite. |
| Cozaar | Antihypertensive | N/A | Decreased sodium intake and decreased calcium intake may be recommended. Avoid natural licorice. |
| Corticosteroids (Prednisone) | Anti-inflammatory, immunosuppressant | Increased appetite, increased weight, anorexia, calcium wasting osteoporosis/ necrosis, edema, increased blood pressure. | Take with food, decrease sodium intake and increase protein intake, avoid alcohol. |
| Coumadin (warfarin) | Anticoagulant | Nausea and vomiting, cramps, diarrhea. | Consistent intake of potassium essential. Caution with vitamin C, caution with > 60 g raw or boiled onions. Avoid/limit garlic, ginger, Ginkgo, and avocado. |
| Digoxin | Cardiotonic, antiarrhythmic, anti–congestive heart failure | Anorexia, decreased weight, nausea and vomiting, diarrhea. | Increase potassium intake, decrease sodium intake, and ensure adequate magnesium and calcium intake. Take separately from high-fiber foods or foods high in pectin. |
| DiaBeta | Oral hypoglycemic | Increased or decreased appetite, increased weight, dyspepsia, nausea, diarrhea, constipation. | Avoid alcohol. |
| Elavil | Antidepressant | Dry mouth, nausea, vomiting, anorexia, taste changes, epigastric distress, diarrhea, constipation, paralytic ileus. | Take with food, high fiber intake may decrease the drug's effectiveness. Limit caffeine, avoid alcohol, St. John's wort, SAM-e, and yohimbe. |
| Eldepryl | Anti-Parkinson's, MAO inhibitor | Dry mouth, dysphagia, nausea, abdominal pain. | Take with breakfast and lunch, avoid foods high in tyramine. |
| Erythromycin | Antibiotic | Anorexia, epigastric distress, nausea and vomiting, abdominal cramps, diarrhea. | Avoid concurrent alcohol use. |

| Medication | Intended Use | Common Side Effects | Drug–Nutrient Interactions or Dietary Considerations |
|---|---|---|---|
| Ferrous sulfate, Fumerin and gluconate | Hematinic, antianemic | Anorexia, nausea and vomiting, dyspepsia, bloating, constipation, diarrhea, dark stools. | Take with 8 oz water or juice on an empty stomach. Limit alcohol. |
| Flagyl | Antibiotic, amebicide, antitrichomonal | Anorexia, dry mouth, stomatitis, metallic taste, nausea and vomiting, epigastric distress, diarrhea, constipation. | Take with food. Avoid alcohol. |
| Glipizide | Oral hypoglycemic | Increased or decreased appetite, increased weight, dyspepsia, nausea, diarrhea, constipation. | Take 30 minutes before first meal of the day. Limit alcohol. |
| Glucophage | Oral hypoglycemic | Anorexia. | Take with food. Avoid alcohol. |
| Glyburide | Oral hypoglycemic | Increased or decreased appetite, increased weight, dyspepsia, nausea, diarrhea, constipation. | If once a day, take with first meal of the day. Avoid alcohol. |
| Haldol | Antipsychotic | Increased appetite, weight gain, anorexia, dry mouth, increased salivation, dyspepsia, nausea, vomiting, constipation, diarrhea. | Take with food. Avoid alcohol. |
| Hydrochlorothiazide (HCTZ) | Antihypertensive, diuretic | Anorexia, increased thirst, dry mouth, nausea and vomiting, gastrointestinal irritation, diarrhea, constipation. | Take in the morning with food or milk. Limit alcohol. |
| Insulin (Novolin, Humulin) | Antidiabetic, hypoglycemic | Increased weight. | Limit alcohol. Follow a diabetic meal plan to balance food with insulin. |
| Isoniazid | Antituberculosis | Dry mouth, nausea and vomiting, epigastric distress, constipation, diarrhea. | Take 1 hour before or 2 hours after meals. Avoid foods high in tyramine or histamine. |
| Klonopin | Anticonvulsant | Dry/sore mouth, constipation, abdominal cramps, gastritis, changes in appetite, nausea, anorexia, diarrhea, increased salivation. | Take with food, limit caffeine, avoid alcohol, caution with some herbal products. |
| Lasix (furosemide) | Diuretic, antihypertensive | Anorexia, increased thirst, oral irritation, stomach cramps, nausea and vomiting, diarrhea, constipation. | Take with food. Increase potassium and magnesium. Decrease caloric and sodium intake. Avoid natural licorice. |
| Levodopa | Anti-Parkinson's | Dry mouth, bitter taste, nausea, vomiting, anorexia, constipation, diarrhea, abdominal pain, excessive salivation, increased or decreased weight, epigastric distress. | May take with low-protein foods or juices but not with high-protein foods. |
| Lipitor | Antihyperlipidemia | Nausea, dyspepsia, abdominal pain, constipation. | Avoid grapefruit juice and alcohol. Decrease fat and cholesterol intake. |
| Methotrexate | Antineoplastic, antipsoriatic, antiarthritic | Stomatitis, altered taste, nausea and vomiting, diarrhea, anorexia, decreased weight, dehydration. | Encourage fluid intake. Avoid alcohol. |
| Micronase | Oral hypoglycemic | Increased or decreased appetite, increased weight, dyspepsia, nausea, diarrhea, constipation. | If once a day, take with first meal of the day. Avoid alcohol. |
| Monoamine oxidase (MAO) inhibitors (isocarboxazid) | Antidepressant, MAO inhibitor | Possible vitamin $B_6$ deficiency, increased appetite, increased weight. | Avoid foods high in tyramine and tryptophan such as cheese, yogurt, and pickled, fermented, and smoked foods. Limit caffeine, avoid tryptophan supplements, may need vitamin $B_6$ supplement, avoid St. John's wort and alcohol. Caution with diabetes—may decrease serum glucose. |

(*continued*)

## Review of Common Medications *(continued)*

| Medication | Intended Use | Common Side Effects | Drug–Nutrient Interactions or Dietary Considerations |
|---|---|---|---|
| Monopril | Antihypertensive, angiotensin-converting enzyme inhibitor | N/A | Decreased calcium and sodium intake may be recommended. Avoid salt substitutes. Caution with potassium supplements, Ensure adequate fluid intake. Avoid natural licorice and alcohol. |
| Oral contraceptives | Used to prevent pregnancy and regulate the menstrual cycle | Increased or decreased appetite, decrease in loss of calcium from the bones, increased absorption of calcium into the bones, nausea and vomiting, bloating, cramps, diarrhea, edema, increased blood pressure. | Take with food the same time each day. Limit alcohol. Increase foods high in Mg, folate, Pyr, and vitamin $B_{12}$. |
| Ofloxacin | Antibiotic | Dry mouth, taste loss, nausea and vomiting, abdominal pain, diarrhea, decreased appetite, constipation, headache, insomnia, dizziness, vaginitis. | Take with 8 oz water. Ensure adequate fluid intake. |
| Paxil | Antidepressant, anti-obsessive-compulsive disorder, anti–panic disorder | Decreased appetite, increased or decreased weight, dry mouth, taste changes, nausea, dyspepsia, constipation, diarrhea. | Avoid St. John's wort, SAM-e, and yohimbe. |
| Phenobarbital | Sedative, hypnotic, anticonvulsant | Nausea and vomiting, constipation, increased rate of metabolism of vitamins D and K. | Avoid alcohol and caffeine. Increase consumption of vitamin C and calcium. |
| Phenytoin (Dilantin) | Anticonvulsant | Taste changes, dysphagia, nausea, vomiting, constipation. | Take with food or milk, avoid alcohol, caution with diabetes—may increase serum glucose. Folate supplement needed. May need vitamin D supplement. |
| Phos-Lo | Phosphate binder | Decreased iron absorption, anorexia, nausea and vomiting, constipation. | Take with meals. Avoid calcium supplements. |
| Prilosec | Antiulcer, antisecretory, anti-GERD | May decrease iron and vitamin $B_{12}$ absorption, decreased gastric acid secretion, increased gastric pH, abdominal pain, constipation, diarrhea. | Take just before a meal, preferably in the morning. Swallow whole, do not crush. |
| Probenecid | Used in the treatment of chronic gout or gouty arthritis | Fast or irregular breathing; puffiness or swellings of the eyelids or around the eyes; shortness of breath, troubled breathing, tightness in chest, or wheezing; changes in the skin color of the face occurring together with any of the other side effects listed here; or skin rash, hives, or itching occurring together with any of the other side effects listed here. | Do not take aspirin or other salicylates or drink alcoholic beverages while taking this medicine, unless you have first checked with your doctor. |
| Prozac | Antidepressant, selective serotonin reuptake inhibitor | Anorexia. | Take in morning with meals. No tryptophan supplements. Avoid alcohol and St. John's wort. Caution with diabetes—may cause hypoglycemia. |
| Remeron | Antidepressant | Increased appetite. | Avoid St. John's wort and alcohol. |
| Synthroid | Thyroid preparation | Appetite changes. | Take on an empty stomach before breakfast to increase absorption. |

| Medication | Intended Use | Common Side Effects | Drug–Nutrient Interactions or Dietary Considerations |
|---|---|---|---|
| Tegretol | Anticonvulsant | Anorexia. | Take with food, avoid alcohol and psyllium seed. Caution with grapefruit juice. |
| Tetracycline (doxycycline minocycline) | Antibiotic | Anorexia. | Take with 8 oz water 1 hour before or 2 hours after food or milk. |
| Thiazide diuretics | Diuretic used to treat high blood pressure and to decrease the amount of water held within the body | *Rare:* Black tarry stools. | Weight control and a low-sodium diet are recommended. Consume foods high in potassium. *Signs and symptoms of too much potassium loss:* Dry mouth, increased thirst, irregular heartbeat, mood or mental changes, muscle cramps or pain, nausea or vomiting, unusual tiredness or weakness, weak pulse. *Signs and symptoms of too much sodium loss:* Confusion, convulsions, decreased mental activity, irritability, muscle cramps, unusual tiredness or weakness. |
| Vasotec | Antihypertensive, angiotensin-converting enzyme inhibitor, anti–congestive heart failure | Stomatitis. | Decreased sodium and calorie intake may be recommended. Avoid salt substitutes, natural licorice, and alcohol. Caution with potassium supplements. |
| Wellbutrin | Antidepressant | Anorexia. | Take with food; avoid alcohol and St. John's wort. May lead to anemia. |
| Zoloft | Antidepressant, selective serotonin reuptake inhibitor | Increased or decreased appetite. | Take consistently with or without food, avoid alcohol, anemia. |

## Life Cycle Nutrition: Pregnancy and Lactation

As discussed earlier in this text, nutrient needs of the pregnant and lactating woman differ from a woman not involved in these activities. The following tables touch on the major nutritional requirements of pregnant and lactating women, environmental and lifestyle contraindications during pregnancy and lactation, recommended weight gain during pregnancy, nutritional remedies for common conditions experienced during pregnancy, and common tests performed during pregnancy.

### Special Macro- and Micronutrient Needs of Pregnant and Lactating Women

| Nutrient | Dietary Reference Intake—Pregnancy | Dietary Reference Intake—Lactation | Comments |
|---|---|---|---|
| Energy | 1st trimester: No calorie increase above prepregnancy needs<br>2nd trimester: +340 kcal above prepregnancy needs<br>3rd trimester: +450 kcal above prepregnancy needs | 1–6 months: 500 kcal<br>6–12 months: 400 kcal | Pregnancy: Adequate energy intake is necessary to satisfy the pregnant mother's nutritional needs and to allow for about a 0.4-kg weight gain per week during the final weeks of pregnancy.<br>Lactation: Increased energy needs are for women breastfeeding exclusively. |

(*continued*)

## Special Macro- and Micronutrient Needs of Pregnant and Lactating Women *(continued)*

| Nutrient | Dietary Reference Intake—Pregnancy | Dietary Reference Intake—Lactation | Comments |
|---|---|---|---|
| Protein | 25+ g/day above prepregnancy needs | | Extra protein is needed to create fetal and maternal tissues during pregnancy, and milk during lactation. |
| Carbohydrate | 175 g/day (no less than 135 g/day) | 210 g/day | Carbohydrate is needed to provide adequate immediate energy, to prevent ketosis, and to maintain normal blood glucose levels. Adequate carbohydrate intake also ensures that the protein needed for growth will not be broken down and used to make glucose. |
| Fat | 45–65% of total calories<br>Linoleic acid: 1.3 g/day<br>Linolenic acid: 1.4 g/day | 45–65% of total calories<br>Linoleic acid: 1.3 g/day<br>Linolenic acid: 1.3 g/day | These specific fatty acids are essential for fetal growth and development, especially of the brain. Beneficial to the fetus in utero and to breastfed infants of mothers consuming a diet rich in these essential fatty acids. |
| Folic acid (see the section titled "Anemia: A Common Deficiency Disease in the United States" later in this appendix for more details on the importance of folic acid in pregnant women) | 600 µg<br>No more than 1,000 µg/day | 500 µg | Folic acid helps prevent neural tube defects—serious birth defects of the spine and the brain. The requirements for folic acid during lactation are less researched. |
| Iron (see the section titled "Anemia: A Common Deficiency Disease in the United States" later in this appendix for more details on the importance of iron in pregnant women) | 27 mg/day | 9 mg/day | Iron is needed for the formation of the hemoglobin required for the expansion of the blood volume and to compensate for the loss of the blood during delivery. Adequate iron intake is also essential for preventing the depletion of the maternal stores and the development of iron-deficiency anemia. Iron deficiency in the mother does not lead to a deficiency in the infant but may increase the risk of a low-birth-weight infant, premature delivery, or prenatal mortality. |
| Zinc | 12 mg/day (≤18 years)<br>11 mg/day (19–50 years) | 13 mg/day (≤18 years)<br>12 mg/day (19–50 years) | Zinc is required for DNA and RNA synthesis, which translates to protein synthesis and cell development. |
| Vitamin $B_{12}$ (see the section titled "Anemia: A Common Deficiency Disease in the United States" later in this appendix for more details on the importance of vitamin $B_{12}$ in pregnant women) | 2.6 mcg | 2.8 mcg | Vitamin $B_{12}$ is required for red blood cell and DNA production, both of which are essential in the growth and development of the fetus. In the infant, vitamin $B_{12}$ deficiency can lead to failure to thrive, delays in reaching developmental milestones, and megaloblastic anemia. |
| Calcium and vitamin D | Calcium: 1,000 mg/day (same as prepregnancy)<br>Vitamin D: 600 IU/15 mcg (same as prepregnancy) | Calcium: 1,000 mg/day (same as prepregnancy)<br>Vitamin D: 600 IU/15 mcg (same as prepregnancy) | Maternal physiology accommodates extra calcium and vitamin D needs by increasing absorption and decreasing losses. Although calcium and vitamin D requirements do not increase during pregnancy and lactation, pregnancy leads to an increased rate of bone turnover. Therefore, adequate calcium and vitamin D are required by pregnant and lactating women to minimize osteoporosis in later life and support fetal development of bones and teeth. |

The following table shows substances not recommended during pregnancy and lactation.

### Contraindications During Pregnancy and Lactation

| Environmental and Lifestyle | Comments |
|---|---|
| Alcohol | Exposure of the developing fetus to alcohol can trigger fetal alcohol syndrome, causing malformations such as facial malformations, low weight and height, smaller than normal head circumference, and mental and physical retardation. Excessive alcohol intake during pregnancy may also lead to a stillbirth. |
| Tobacco | Exposure to tobacco in utero has been shown to decrease infant birth weight and increase the risk of perinatal morbidity and mortality. Tobacco can cause a reduced blood flow to the uterus and often blunts appetite, potentially leading to a reduced food intake by the mother. |
| Environmental contaminants | Lead: During pregnancy, lead easily crosses into the placenta and can cause severe damage to the developing fetal nervous system. Even slight exposure to lead during gestation can lead to a low-birth-weight infant. |
| | Mercury: Although fatty fish can be a good source of omega-3 fatty acids, some of are too high in the contaminant mercury. As a result, pregnant women should avoid shark, swordfish, king mackerel, and tilefish. Weekly fish consumption should be limited to 12 oz (cooked or canned) seafood or 6 oz of white (albacore) tuna. |
| Artificial sweeteners | The use of saccharin is not recommended during pregnancy. Use of aspartame should also be restricted to less than 50 mg/kg body weight. Aspartame can lead to an increase in blood phenylalanine in women with phenylketonuria, which can lead to fetal brain damage. |
| Caffeine | Excessive caffeine consumption in pregnancy may produce problems with bone formation, causing deformed fingers, toes, and cleft palate. Also, excessive caffeine intake can lead to overstimulation of the mother and fetus. Less than 200 mg/day is recommended (about 2 cups coffee/day). |

The following table shows recommended weight gain during pregnancy:

| Prepregnancy Weight | Recommended Weight Gain (lb) |
|---|---|
| Underweight (BMI < 18) | 28–40 |
| Normal body weight (BMI 18.5–24.9) | 25–35 |
| Overweight (BMI 25–29.9) | 15–25 |
| Obese (BMI ≥ 30) | 11–20 |

The following table shows nutritional remedies for conditions common to pregnancy.

### Common Complications and Conditions of Pregnancy

| Symptom/Condition | Description/Cause | Nutritional Intervention |
|---|---|---|
| Constipation | Usually occurs during the latter part of pregnancy as a result of reduced gut motility, physical inactivity, and pressure exerted on the bowel by the enlarged uterus. | Increase fluids and fiber. If necessary, add stool softeners. |
| Fluid retention/edema | Usually present in the third trimester. May be caused by the pressure of the enlarging uterus on the veins returning fluid from the legs. | Sodium restriction, adequate fluid intake, and exercise can all help reduce fluid retention. |

(*continued*)

### Common Complications and Conditions of Pregnancy *(continued)*

| Symptom/Condition | Description/Cause | Nutritional Intervention |
|---|---|---|
| Gestational diabetes | Insulin used to shuttle glucose from the blood into the cells can become less effective because of weight gain and hormonal changes during pregnancy. This results in glucose buildup in the blood. Diagnosis is generally made during the second or third trimester. | Limit intake of high-carbohydrate foods. Eat small, frequent meals. Perform self-monitoring of blood glucose levels, ketones, appetite, and weight gain. Ensure adequate calories and nutrition. Exercise and avoid excessive weight gain. |
| Heartburn | Usually occurs during latter part of pregnancy as a result of pressure of the enlarged uterus on the stomach in combination with the relaxation of the esophageal sphincter. | Limit the amount of food consumed at one time. Drink fluids between meals. Wear loose clothing around waist, eat slowly, and remain upright for at least 30 minutes to 1 hour after meals. |
| Hyperemesis | Severe and continued nausea and vomiting | Hospitalization to control dehydration, correct electrolyte imbalances, and correct excessive weight loss. Encourage small, frequent meals and lots of fluids. Eat foods with a high fluid content, as tolerated. Electrolyte replacement fluids will help with fluid retention. |
| Leg cramps | Usually occurs at night. Sudden contractions of the gastrocnemius muscles. | Increased calcium intake via calcium-rich foods or supplements is often recommended. Also, ensure adequate hydration status. |
| Nausea/morning sickness | Common during the early months. May cause acute protein/energy deficit and aberrations in some vitamins, minerals, and electrolytes. | Encourage small, frequent, dry meals of easily digested carbohydrate-containing foods. Liquids are best tolerated between meals. Avoid foods/odors that trigger nausea. |
| Pica | Pica is cravings for persistent ingestion of nonfood items that have little nutritional value and is poorly understood. Certain foods may relieve nausea and vomiting or compensate for a nutritional deficiency. | Ensure adequate calories and optimal nutrition. Medications may help reduce abnormal food cravings. |
| Pregnancy-induced hypertension | Characterized by hypertension, proteinuria, and edema. Hypoalbuminemia, hypovolemia, and subsequent hemoconcentration are also present. Generally occurs in the third trimester. Associated with poverty, lack of prenatal care, and poor nutritional status (i.e., protein and calcium deficiency). | Calcium and magnesium supplementation may help. Restriction of sodium consumption is recommended. |

The following table shows common tests performed during pregnancy.

### Common Laboratory Tests Performed During Pregnancy

| Test | Description |
|---|---|
| Alpha-fetoprotein screening | Alpha-fetal protein is normally produced by the fetal liver and yolk sac. Helpful in the diagnosis of fetal body wall defects. The most notable of these are neural tube defects. |
| Amniocentesis | To determine fetal maturity status, sex of fetus, genetic and chromosomal aberrations, fetal status affected by Rh isoimmunization, hereditary metabolic disorders, anatomic abnormalities, and fetal distress. |
| Chorionic villi sampling | Performed on women whose unborn child may be at risk for life-threatening or significant genetic defects (i.e., older age at time of pregnancy, frequent spontaneous abortions, pregnancies with fetuses with genetic defects, and women identified as having a genetic defect themselves). |

| Test | Description |
|---|---|
| Fetal heart rate monitoring | Part of the fetal biophysical profile. Ultrasound, fetal activity studies, and nonstress tests (noninvasive) can monitor acceleration of the fetal heart rate. |
| Glucose tolerance test | Intended for women who have a history of delivering large infants, stillbirths, or neonatal births. Also, for women who have transient glycosuria (glucose in their urine) or hyperglycemia during pregnancy. |
| Hemoglobin (Hb) | Refer to the preceding section titled "Review of Common Laboratory Values." |
| Hematocrit (Hct) | Refer to the preceding section titled "Review of Common Laboratory Values." |
| Oral glucose test | A test in which a pregnant woman consumes a predetermined amount of carbohydrate and then has her blood sugar levels measured 1 hour after she finishes consuming the liquid to determine whether she has elevated blood sugar levels that need to be monitored in case gestational diabetes develops. |
| Ultrasound | Provides accurate visualization of the fetus's abdominal aorta, liver, gallbladder, pancreas, bile ducts, kidneys, ureters, bladder, spine, and other important organs to ensure that development of the fetus and its systems are normal. Also used to determine the age of the fetus. |

The following table provides pregnancy and lactation medical terminology.

### Common Pregnancy and Lactation Terminology

| Abbreviation or Term | Meaning |
|---|---|
| AFP | Alpha-fetoprotein screening |
| BF | Breastfeed |
| Colostrum | A milk-like secretion from the breast, present during the first day or so after delivery before milk appears; rich in protective factors |
| DOB | Date of birth |
| Eclampsia | A severe stage of preeclampsia characterized by convulsions |
| EDC | Estimated date of confinement |
| FTT | Failure to thrive |
| G | Gravida |
| Gest | Gestation |
| GYN | Gynecology |
| Hyperemesis | Severe and continued nausea and vomiting |
| LBW | Low birth weight < 5.5 lb |
| LMP | Last menstrual period |
| Mature milk | Milk that comes in after the colostrum |
| NICU | Neonatal intensive care unit |
| OB | Obstetrics |
| Parity | The number of children borne by one woman |
| PIH | Pregnancy-induced hypertension |
| Postpartum | After birth |
| Preeclampsia | A condition characterized by hypertension, fluid retention, and protein in the urine; formerly known as pregnancy-induced hypertension |
| SGA | Small for gestational age |
| SRM | Spontaneous rupture of membranes |
| Toxemia | Hypertensive disease of pregnancy |
| VLBW | Very low birth weight |

## Infant, Child, and Adolescent Nutritional Guidelines

The first year of life is an exceptional time for growth and development. Although growth and development slow after 1 year of age, the cumulative effects of changes from age 2 through puberty has a significant effect on health and well-being. The information that follows addresses common nutritional guidelines and issues present in each stage of the life cycle from infancy through adolescence. The following table addresses the nutritional needs of an infant through age 1 year.

### Infant Nutritional Guidelines Through Age 1 Year

| Nutrient | Requirements | Rationale |
|---|---|---|
| Energy | 100 kcal/kg body weight<br>Newborn: 450 kcal/day<br>After 6 months, although the infant's energy needs decline as growth rate slows, the infant's activity levels increase, making energy needs remain high. | High energy needs are required to support doubling of birth weight by age 5 months and tripling of birth weight by 1 year. |
| Protein | 0–5 months: 9.1 g/day<br>5–12 months: 11 g/day | Protein is the single most important nutrient for growth. Inadequate protein intake can negatively affect brain, immune, and digestive capabilities of the infant. *Failure to thrive* is a term used to describe protein deficiency. Too much protein can also cause problems such as acidosis, dehydration, diarrhea, elevated blood urea and ammonia, and fever. |
| Carbohydrate | 0–5 months: 60 g/day<br>5–12 months: 95 g/day | An infant's brain weight as a percentage of total body weight is six times greater than an adult's, making the need for glucose greater, at 60% of the day's total energy intake. |
| Fat | 0–5 months: 31 g/day<br>5–12 months: 30 g/day | Fat's high energy density supports the rapid growth of the infant and is essential for proper brain growth and development. |
| Water | 0–5 months: 0.7 L/day<br>5–12 months: 0.8 L/day | The younger an infant, the greater his or her percentage of body weight is water. During early infancy, breast milk or formula provide adequate fluids, but in hot environments or when an infant experiences diarrhea or vomiting, supplemental water may be required. |

The following table addresses the most common advantages of breastfeeding for the mother and infant.

### Benefits of Breastfeeding

**For Infants**

- Provides the appropriate composition and balance of nutrients with high bioavailability
- Provides hormones that promote physiologic development
- Improves cognitive development
- Protects against a variety of conditions and diseases such as diarrhea, ear infections, and pneumonia
- May protect against some chronic diseases such as diabetes, obesity, atherosclerosis, asthma, and hypertension later in life
- Protects against the risk of sudden infant death syndrome
- Reduces the risk of food allergies
- Promotes a sense of security and belonging from the warmth of the mother's body and being held

**For Mothers**

Contracts the uterus

Delays the return of regular ovulation, but this is not considered a means of contraception

Conserves iron stores by prolonging amenorrhea

May protect against breast and ovarian cancers

Increases energy expenditure, which may contribute to weight loss

**Other**

Cost and time savings from not having to purchase and prepare formula, and reduced incidence of doctors visits.

Environmental savings to society from not needing to manufacture, package, and ship formula and dispose of the packaging.

The following table lists some common barriers to breastfeeding and solutions to these barriers.

### Common Barriers and Solutions to Breastfeeding

| Common Barrier | Solutions |
|---|---|
| Lack of breastfeeding knowledge | Have written breastfeeding policies at health centers, train healthcare workers in breastfeeding, inform all pregnant women about the benefits of breastfeeding, have mother initiate breastfeeding during the first half hour of birth. |
| Fatigue | Encourage a glass of water or other fluids during every breastfeeding session to prevent dehydration; encourage mom to sleep when the baby sleeps; use a breast pump and have another individual feed the baby during the night; begin exercise as soon as mother is cleared by Ob/Gyn. |
| Lack of freedom to return to work and social life | Use a breast pump regularly so extra breast milk is available for feedings; encourage worksites to have a separate breastfeeding rooms for mothers. |
| Possibility of breast infections (mastitis) | Have nurses or lactation consultants show mothers how to breastfeed properly; breastfeed on demand; do not wear tight-fitting bras for extended periods of time; consume plenty of fluids. |
| Concerns about the infant not receiving adequate nourishment | Feed on demand; provide newborns no food or drink other than breast milk unless medically indicated; practice rooming-in; allow mothers and infants to remain together for at least 24 hours after birth; discourage use of artificial pacifiers/teats; and establish breastfeeding support groups and refer mothers upon discharge. |
| Concerns about environmental factors being passed to the infant | Limit fish and shellfish to up to 12 oz (two average meals) per week. Choose fish that are lower in mercury, including cod, haddock, pollock, salmon, sole, tilapia, canned light tuna, shrimp, and most shellfish. Do not eat shark, swordfish, king mackerel, or tilefish. |

The following table indicates supplements that are recommended for full-term infants.

### Supplement Requirements for Full-Term Infants

| Ages | Vitamin D | Iron | Fluoride |
|---|---|---|---|
| **Breastfed infants** | | | |
| Birth–6 months | ✔ | | |
| 6 months–1 year | ✔ | ✔ | ✔ |
| **Formula-fed infants** | | | |
| Birth–6 months | | | |
| 6 months–1 year | | ✔ | ✔ |

The following table compares the various infant formulas.

### A Review of Common Infant Formulas

| Formula | Indications |
|---|---|
| Alimentum | For food allergies and colic (caused by protein sensitivity):<br>• Hypoallergenic<br>• Nutritionally complete formula for infants and children with severe food allergies and colic caused by protein sensitivity<br>• Provides an easily digested and absorbed fat blend<br>• Provides predigested protein to avoid symptoms of milk allergy in most infants and children |
| Enfamil with iron | Enfamil with iron is designed to provide complete nutrition to the infant. Enfamil with iron has a protein blend that is easy to digest, has a 60:40 whey-to-casein protein ratio, is similar to breast milk, has calcium to help build strong bones as happens with breastfed infants, and approximately 50% of the calories in both breast milk and Enfamil with iron come from fat. |
| Enfamil Premature | Enfamil Premature LIPIL is designed for use in the hospital as a sole source of nutrition for premature infants who are not breastfed. It is enriched to provide added nutrition that premature infants need. Enfamil Premature LIPIL also has a unique blend of DHA and ARA, ingredients that are important building blocks for an infant's brain and eyes. |
| Isomil Advance | This formula is used when DHA and ARA are recommended. It is milk-free and lactose-free. DHA and ARA are special nutrients found in breast milk that are important for mental and visual development. |
| Isomil 2 | For infants 6–18 months of age who are eating cereal and baby foods. Milk-free and lactose-free. As infants grow, their nutritional needs change. For example, the amount of calcium infants need doubles by the time they reach age 1 year. Cereals and other foods alone may not provide all the calcium an infant needs during this time of rapid growth. Isomil 2 has been specially designed to help meet the nutritional requirements of older infants and toddlers. Isomil 2 provides calcium to help support developing bones and is fortified with iron, which is important for brain development. |
| Nutramigen | This formula is used for those infants with colic, often indicated by an infant who screams and cries. Even the most experienced parents do not always know what to do. Calling the infant's doctor is always the best place to start to determine whether medical attention is required. When colic is the reason for the infant's distress, his or her doctor may suspect protein allergy, a reaction that can occur to the protein in either milk-based or soy-based formulas. Protein allergy is serious. In infants it can cause a variety of problems, such as diarrhea, difficulty breathing, hives, a kind of rash known as atopic eczema, and the uncontrollable screaming and crying that is often termed "colic." If a protein-related problem is suspected, a special formula, such as Nutramigen, is prescribed. This member of the Enfamil family of formulas is the only infant formula that clinical studies have proven to be effective for managing colic resulting from cow's milk protein allergy. Nutramigen has been trusted for more than 50 years to help manage infants with protein allergy. |
| PediaSure | Milk-based complete balanced nutrition: 1.0 Cal per mL, 237 Cal per 8 fl oz, from a balanced distribution of protein, fat, and carbohydrate (caloric distribution: protein, 12%; fat, 44%; carbohydrate, 44%).<br>Comes in four "kid-approved" flavors: Chocolate, strawberry, vanilla, and banana cream.<br>Potential renal solute load: Close to that of infant formulas.<br>High-quality protein: 82% casein, 18% whey. Amino acid profile meets NAS-NRC standards for high-quality proteins.<br>Gluten-free and lactose-free.<br>Fat content, 50 g/L: 50% high-oleic safflower oil, 30% soy oil, and 20% medium-chain triglycerides.<br>Contains selenium, chromium, molybdenum, inositol, taurine, and carnitine.<br>Calcium-to-phosphorus ratio of 1.2:1. Conforms to AAPCON recommendations for growing children.<br>Iron concentration of 1.4 mg/100 Cal. Meets NAS-NRC recommendations for children 1–10 years old in 1,000 Cal.<br>Appropriate vitamin D content: 51 IU/100 Cal meets children's needs better than most adult enteral products. |
| PediaSure with Fiber | PediaSure with Fiber enteral formula is designed to provide a source of complete balanced nutrition for children aged 1–10 years who may benefit from fiber. It is a lower osmolality formula than PediaSure. PediaSure with Fiber enteral formula is designed for children who are fed by tube and contains soy fiber at a level shown to be well tolerated by children. It also is appropriate for oral feeding. Not approved for children with galactosemia. |

| Formula | Indications |
|---|---|
| Peptamen | Peptamen Complete Isotonic Liquid Elemental Diet is formulated to provide complete or supplemental nutritional support in an easily absorbed form for patients with impaired gastrointestinal function. Patients benefiting from Peptamen Diet may include those with short bowel syndrome, inflammatory bowel disease, malabsorption syndromes, pancreatic insufficiency, chronic diarrhea, radiation enteritis, and delayed gastric emptying. Peptamen Diet may also be useful as a dual feeding with total parenteral nutrition or as a transition diet from total parenteral nutrition.<br>*Note:* Peptamen Diet contains ingredients (i.e., partially hydrolyzed whey protein, from cow's milk protein) that may not be appropriate for individuals with food allergies. |
| Portagen | Portagen provides complete nutrition for infants and toddlers younger than 2 years of age who do not efficiently digest or absorb conventional fat. Portagen contains appropriate levels of readily digestible protein, fat, and carbohydrate for infants and toddlers younger than 2 years of age and is iron fortified, lactose-free, and gluten-free. Available as an unflavored powder that may be flavored or sweetened to taste, Portagen provides 86% of its fat as medium-chain triglycerides (MCT oil), a type of fat that is easily digested and absorbed. Linoleic acid is provided through corn oil. Portagen should be used only under a physician's supervision. |
| Pregestimil | Pregestimil infant formula is rarely used without a doctor's recommendation. It provides the same extensively predigested protein as Nutramigen, but it differs from Nutramigen in its source of fat. The fat blend in Pregestimil consists mostly of a special type of fat, MCT oil, that is more easily digested and absorbed than the close-to-breastmilk fat blend in Enfamil, LactoFree, Nutramigen, and ProSobee. The unique formulation of Pregestimil is designed for feeding infants with diseases or disorders that make it difficult for them to absorb the fat blend in most other formulas. These conditions include short bowel syndrome, steatorrhea (fat in the stool), cystic fibrosis, or persistent diarrhea that does not respond to treatment. |
| ProSobee (Enfamil) | Iron-fortified powder specially designed for infants in the first 12 months needing a soy formula. It is milk-free and lactose-free and easy to digest. |
| Similac with Iron | Excellent nutrition for infants aged 0–12 months. When DHA and ARA, special nutrients found in breast milk, are recommended. DHA and ARA are special nutrients found in breast milk that are important for mental and visual development. Similac Advance is an excellent choice when DHA and ARA are recommended. Ideal for supplementation. Provides calcium for growing bones. Designed to be gentle on the infant's developing digestive system. |
| Similac NeoSure | NeoSure Advance contains DHA and ARA, special nutrients found in breast milk that are important for brain and eye development. DHA and ARA have been clinically shown to improve visual development in preterm infants. |

ARA, arachidonic acid; DHA, docosahexaenoic acid.

The following table outlines guidelines for introducing solid foods to infants.

### Solid Food Introduction to Infants

| Age (months) | Appropriate Food Additions |
|---|---|
| 0–4 | Breast or infant formula |
| 4–6 | Begin iron-fortified cereal (rice first, then barley or oat, and wheat last) mixed with breast milk or formula or water |
| | Begin puréed vegetables, legumes, fruits, and meats |
| 6–8 | Begin textured vegetables and fruits |
| | Begin unsweetened, diluted fruit juices from a cup |
| 8–10 | Begin breads and cereals from the table |
| | Begin yogurt |
| | Begin pieces of soft, cooked vegetables from the table |
| | Gradually begin finely cut meats, fish, casseroles, cheese, eggs, and mashed legumes |

(*continued*)

### Solid Food Introduction to Infants *(continued)*

| Age (months) | Appropriate Food Additions |
|---|---|
| 10–12 | Add variety |
| | Gradually increase portion sizes |
| **Important Notes** | To reduce the risk of an allergic response to foods as they are introduced, single-ingredient foods should be introduced one at a time, in small portions, and waiting 3 to 5 days before introducing the next new food. |
| | To prevent choking do not give infants or young children cherries, whole grapes, raw carrots or celery, whole beans, gum, hard or gel-type candies, marshmallows, nuts, popcorn, hot dog slices, peanut butter. |

The following table provides the estimated daily energy needs for children ages 2–13 years.

### Estimated Energy Needs for Children, Ages 2–13 Years

| Age | Energy (kcal) |
|---|---|
| 2–3 years | 1,000 |
| **Females** | |
| 4–8 years | 1,200–1,400 |
| 9–13 years | 1,600 |
| **Males** | |
| 4–8 years | 1,200–1,400 |
| 9–13 years | 1,800 |

The following table demonstrates meal patterns that can be followed to meet macro- and micronutrient needs of children ages 2–13 years.

### Recommended Daily Amounts from Each Food Group, 1,000–1,800 kcal/day

| Food Group | 1,000 kcal | 1,200 kcal | 1,400 kcal | 1,600 kcal | 1,800 kcal |
|---|---|---|---|---|---|
| Fruits | 1 cup | 1 cup | 1½ cups | 1½ cups | 1½ cups |
| Vegetables | 1 cup | 1½ cups | 1 ½ cups | 2 cups | 2½ cups |
| Grains | 3 oz | 4 oz | 5 oz | 5 oz | 6 oz |
| Protein foods | 2 oz | 3 oz | 4 oz | 5 oz | 5 oz |
| Milk | 2 cups | 2½ cups | 2½ cups | 3 cups | 3 cups |

The following table introduces strategies that can be implemented with children and adolescents to reduce the risk of obesity.

### Obesity Prevention Measures for Children and Adolescents

The American Medical Association recommends the following healthy habits for individuals aged 2–18 years to reduce the risk of obesity:

- Reduce consumption of sugar-sweetened beverages such as soft drinks and fruit-flavored juice drinks and sweetened beverages.
- Aim for 2–4.5 servings of fruits and vegetables every day.

- Learn to identify proper food portions, including a balance of carbohydrates, fiber, fat, and proteins.
- Consume foods with a low energy density, but a high nutrient density.
- Consume breakfast every day.
- Eat a diet rich in calcium and vitamin D.
- Limit meals at restaurants.
- Consume family meals whenever possible.
- Limit screen time, including television, computer, and video game time, to no more than 2 hours per day.
- Engage in at least 60 minutes of moderate to vigorous physical activity every day.

The following table identifies the nutrient needs of the preteen and adolescent population.

**Preteen and Adolescent Nutritional Guidelines, Ages 9–18 Years**

| Nutrient | Requirements | Rationale |
|---|---|---|
| Energy | Female (9–13 years): 2,071 kcal<br>Female (14–18 years): 2,368 kcal<br>Male (9–13 years): 2,279 kcal<br>Male (14–18 years): 3,152 kcal | Energy recommendations vary depending on the current rate of growth, gender, body composition, and physical activity level of the adolescent. |
| Protein | Female (9–13 years): 0.95 g/kg<br>Female (14–18 years): 0.85 g/kg<br>Male (9–13 years): 0.95 g/kg<br>Male (14–18 years): 0.85 g/kg | Protein is the single most important nutrient for growth. |
| Carbohydrate | 45–65% of total daily energy intake<br>RDA = 130 g/day (minimum) | Adequate carbohydrate is essential to spare protein for use in growth and development, especially during puberty. Carbohydrates are also essential for optimal brain function and athletic performance. |
| Fiber | Females: 25 g/day<br>Males: 38 g/day | To meet fiber recommendations, at least half of an adolescent's carbohydrates should consist of whole grains. Additionally, a minimum of five servings of fruits and vegetables ensures adequate fiber is consumed. |
| Fat | 20–35% of total daily energy intake<br>≤300 mg cholesterol/day<br>≤20–25 g saturated fat/day | Fat's high energy density supports the rapid growth of the child and teenager. Ideally, healthy fats, such as monounsaturated fats and omega-3 fatty acids, are consumed in place of trans fatty acids and saturated fats. |
| Water | Females: 8 cups/day<br>Males: 11 cups/day | Males tend to have more tissue than females and are more active, so their fluid needs are higher than females'. Highly active adolescents may require more fluids to prevent dehydration, especially prior to and during puberty when temperature regulation is less efficient and dehydration more common. |
| Calcium | Female (9–18 years): 1,300 mg<br>Male (9–18 years): 1,300 mg | Adolescence is a crucial time for bone development, but unfortunately many adolescents consume inadequate quantities of calcium. Inadequate calcium intake during these huge growth years can compromise the development of peak bone mass. |
| Vitamin D | Female (9–18 years): 600 IU<br>Male (9–18 years): 600 IU | Vitamin D is also essential for bone growth and development, and adolescents tend to come up short in this nutrient as well. |
| Iron | Female (9–13 years): 8 mg<br>Female (14–18 years): 15 mg<br>Male (9–13 years): 8 mg<br>Male (14–18 years): 11 mg | Iron needs increase for both males and females as they experience their growth spurt. Iron needs increase for females when they start to lose blood through menstruation. Iron needs for males increase as their lean body mass develops. |

(*continued*)

### Preteen and Adolescent Nutritional Guidelines, Ages 9–18 Years *(continued)*

| Nutrient | Requirements | Rationale |
|---|---|---|
| Zinc | Female (9–13 years): 8 mg<br>Female (14–18 years): 9 mg<br>Male (9–13 years): 8 mg<br>Male (14–18 years): 11 mg | Zinc is another crucial nutrient during stages of rapid growth and development. |
| Vitamin A | Female (9–18 years): 700 µg/kg<br>Male (9–18 years): 900 µg/kg | Vitamin A is another crucial nutrient during stages of rapid growth and development. Consuming at least five servings of fruits and vegetables each day can help children and teens meet the RDA for vitamin A. |

The following table identifies the nutrient needs of older adults.

### Older Adults Nutritional Guidelines, 65+ Years

| Nutrient | Requirements | Rationale |
|---|---|---|
| Energy | Female (65 years): 2,298 kcal<br>Male (65 years): 2,917 kcal<br>Note: Females should subtract 7 calories for every year over 19 years, and males 10 calories for every year over 19 years. | On average, energy needs decline 5% per decade as a result of declines in physical activity, basal metabolic rate, and thyroid hormone function. Although energy needs tend to decline as an individual ages, nutrient needs remain high. |
| Protein | 0.80 g/kg | Protein is essential for older adults to support their immune system, prevent muscle atrophy, and support bone mass. Because energy needs decline as an adult ages, protein must be obtained from low-calorie sources of high-quality protein, such as lean meats, poultry, fish, eggs, and low-fat dairy. |
| Carbohydrate | 45–65% of total daily energy intake<br>RDA = 130 g/day (minimum) | Adequate carbohydrate helps spare bodily protein that declines with age. |
| Fiber | Females: 21 g/day<br>Males: 30 g/day | Fiber needs are reduced after age 51 years because of lower energy requirements. |
| Fat | 20–35% of total daily energy intake<br>≤300 mg cholesterol/day<br>≤20-25 g saturated fat/day | Fat needs are the same as that of adults of all ages. |
| Water | Females: 91 oz/day<br>Males: 125 oz/day<br>Note: These fluid recommendations include fluid occurring naturally in both foods and beverages. | As individuals age, the thirst mechanism becomes delayed, which can lead to suboptimal fluid intake. Older individuals need to consume fluids proactively to prevent dehydration. Adequate fluid intake will also help alleviate constipation, which is more common in this age group. |
| Calcium | Female (50+ years): 1,200 mg<br>Male (70+ years): 1,200 mg | Adequate calcium intake is crucial for bone health maintenance during the life cycle. Calcium needs increase as a woman goes through menopause. During this time, bone mass declines rapidly. The risk of osteoporosis increases as we age in both males and females, rationalizing the increased calcium recommendations. |
| Vitamin D | 800 IU | Vitamin D requirements increase in older individuals as a result of decreased bone density, reduced ability to synthesize vitamin D in the skin, and a decline in absorption of dietary calcium. |

| Nutrient | Requirements | Rationale |
|---|---|---|
| Iron | 8 mg | Iron needs decline for women around age 51 years as a result of cessation of menstruation and some loss of muscle and lean tissue. Iron needs stay the same for males older than age 51 years. |
| Vitamin $B_{12}$ | 2.4 µg | Older individuals have similar vitamin $B_{12}$ requirements as younger adults, but can have absorption issues that interfere with proper vitamin $B_{12}$ absorption. These issues include lower levels of stomach acid and an overall decline in vitamin $B_{12}$ absorption from the gastrointestinal tract. Vitamin $B_{12}$ intake also tends to be suboptimal in this population. |

## A Review of Common Chronic Diseases and Deficiencies

### Coronary Heart Disease: The Leading Cause of Death in the United States

#### Dietary and Lifestyle Strategies for Reducing the Risk of Coronary Heart Disease

| | |
|---|---|
| Energy | Balance energy intake and physical activity to achieve a healthy body weight and to prevent age-related weight gain. |
| Fat (saturated, trans, and cholesterol) | Limit saturated fat to <7% of total daily calories, trans fats to <1% of total daily calories, and cholesterol to <300 mg/day. |
| Fish and omega-3 fatty acids | Aim to consume two servings of omega-3-rich fatty fish (salmon, tuna, mackerel) per week. Not a fish lover? Go for canola oil, soy foods, flaxseed, wheat germ, and green, leafy vegetables. |
| Added sugars | Minimize consumption of foods and beverages with added sugars to reduce the risk of weight gain, elevated triglyceride levels, and blood glucose fluctuations. |
| Soluble fibers | Soluble fiber can help lower blood cholesterol levels. Choose a diet rich in vegetables, fruits, and whole grains to maximize soluble fiber intake. |
| Soy | Consume soy foods in moderation to ensure adequate protein intake without the excessive fat or calories commonly found in high-fat animal and dairy products. |
| Potassium and sodium | Eat fresh, unprocessed foods to increase potassium intake and lower sodium intake. Choose a diet high in potassium-rich fruits and vegetables, low-fat milk products, nuts, and whole grains. Limit sodium intake to ≤1,500 mg/day. |
| Alcohol | Limit alcohol intake to 1 drink a day for women and 2 drinks a day for men. |
| Physical activity | Participate in at least 30 minutes of moderate-intensity endurance activity on most days of the week with the ultimate goal of expending 2,000 calories per week via planned physical activity. |
| Smoking cessation | Minimize exposure to any form of tobacco or tobacco smoke. |

### Cancer: The Second Leading Cause of Death in the United States

#### Dietary and Lifestyle Strategies for Reducing the Risk of Various Cancer

| | |
|---|---|
| Weight maintenance | Avoid weight gain and waist circumference increases throughout adulthood. Maintain a BMI between 18.5 and 24.9. |
| Added sugars | Minimize consumption of foods and beverages with added sugars to reduce the risk of weight gain, elevated triglyceride levels, and blood glucose fluctuations. |
| Fat (saturated, trans, and cholesterol) | Limit saturated fat to < 7% of total daily calories, trans fats to <1% of total daily calories, and cholesterol to <300 mg/day. |
| Plant foods | Aim to fill 75% of your plate with fruits, vegetables, and whole grains. Aim for at least 5 servings of nonstarchy vegetables and fruits every day. Limit refined starchy foods while focusing on unprocessed grains. Consume no more than 18 oz of red meat per week. Limit processed meats. |

(*continued*)

**Dietary and Lifestyle Strategies for Reducing the Risk of Various Cancer *(continued)***

| | |
|---|---|
| Alcohol | Limit alcohol intake to 1 drink a day for women and 2 drinks a day for men. |
| Preservation, processing, and preparation | Limit consumption of salt-preserved, salted, or salty foods. Avoid moldy grains or legumes. |
| Physical activity | Participate in at least 30 minutes of moderate-intensity endurance activity on most days of the week with the ultimate goal of expending 2,000 calories per week via planned physical activity. |

## Diabetes: The Seventh Leading Cause of Death in the United States

Diabetes mellitus is a group of genetically and clinically related disorders characterized by blood glucose levels above defined limits. Diverse etiologic and pathologic mechanisms contribute to a relative or absolute deficiency in insulin, a hormone secreted by pancreatic beta cells. Significant clinical complications are associated with the hyperglycemia of diabetes, including macrovascular disease (two to six times greater risk than in the general population), heart disease, stroke, peripheral vascular disease, microvascular disease, nephropathy, retinopathy, and neuropathy. Overall, the risk for death among people with diabetes is about two times that of people without diabetes.

Individuals with diabetes are classified into two primary categories according to the etiology and subsequent treatment needs. Nearly 90% of individuals with diabetes have non-insulin-dependent diabetes mellitus (NIDDM) and 5% to 10% have insulin-dependent diabetes mellitus (IDDM). Other classifications of glucose intolerance include gestational diabetes and impaired glucose tolerance. The following table contrasts and compares type 1 and type 2 diabetes.

**Comparison of Type 1 and Type 2 Diabetes**

| Characteristics | Type 1 Diabetes (IDDM) | Type 2 Diabetes (NIDDM) |
|---|---|---|
| Prevalence in diabetic population | 5–10% of cases | 90–95% of cases |
| Insulin production | Body's immune system destroys pancreatic beta cells, the only cells in the body that make the hormone insulin that regulates blood glucose. | Begins as insulin resistance. Insulin deficiency in relation to needs. As the need for insulin rises, the pancreas gradually loses its ability to produce insulin. |
| Age at onset | Childhood to young adult | Childhood to older adulthood |
| Etiology/metabolic abnormalities | Cell-mediated autoimmune destruction of the beta cells of the pancreas; idiopathic. | Abnormal pattern of insulin secretion and action; decreased cellular uptake of glucose and increased postprandial glucose, increased release of glucose by liver. |
| Genetic influences on disease development | Genetic predisposition | Genetic and environmental factors |
| Common disease-related symptoms | Polydipsia (excessive thirst); polyuria (excessive urination); glycosuria (glucose in the urine); unexplained increase in appetite or in later stages unexplained weight loss; fatigue; blurred vision; tingling or numbness in the hands or feet | |
| Rate at which symptoms appear | Variable: some rapidly, some slowly | Gradual onset |
| Associated conditions and complications | Skin conditions, gum disease, retinopathy, kidney disease, transplantation, neuropathy, cardiovascular disease, feet complications | |
| Risk of metabolic ketoacidosis | High | Nonketotic |
| Medications | Insulin therapy | Oral hypoglycemic agents; insulin in advanced stages |
| Primary treatment objectives | Insulin injections; insulin, food, and exercise consistency from day to day; carbohydrate counting | Hypocaloric diet and physical activity; spacing of meals; carbohydrate consistency; management of fat intake |

The following table introduces laboratory tests important to the diagnosis and management of diabetes mellitus.

### Laboratory Tests Commonly Administered to Individuals at Risk for or Diagnosed with Diabetes

| Laboratory Measurement | Description and Significance | Accepted Laboratory Value Range |
|---|---|---|
| Blood urea nitrogen (BUN) | Measures the amount of urea nitrogen in the blood, which is formed in the liver as the end product of protein metabolism. Measurements of BUN and creatinine are the two most commonly ordered tests of the kidney's ability to excrete metabolic waste. Individuals with diabetes are at a greater risk of possessing compromised renal function. | 10–12 mg/dL (adult) |
| Creatinine | Creatinine is correlated with changes in the serum urea nitrogen (BUN) for diagnostic purposes but is more accurate as an index of the glomerular filtration rate. It is therefore used to test the kidney's ability to excrete metabolic wastes or in those instances where the cause of an elevated BUN value is uncertain. | 0.5–1.1 mg/dL (female) 0.6–1.2 mg/dL (male) |
| Fasting blood glucose | Measurement of blood glucose levels after fasting for ~8 hours. In the fasting state, glucose levels tend to be at their lowest. | Normal: 70–99 mg/dL<br>Pre-diabetes: 100–125 mg/dL<br>Diabetes: 126 mg/dL+ |
| Fingerstick blood sugar evaluations | Random measurements of blood glucose levels obtained by pricking one's finger. | Goal: 70–99 mg/dL , but readings will vary depending on proximity of the individual's last meal |
| Glucose tolerance test (GTT) | GTTs are provocative, or stimulating, tests in which large doses of glucose are given, orally or by intravenous injection, and the rise and fall of the blood glucose level, and in many cases urine glucose, are checked at timed intervals. | Fasting: 70–115 mg/dL<br>30 minutes: < 200 mg/dL<br>1 hour: < 200 mg/dL<br>2 hour: < 140 mg/dL<br>3 hour: 70–115 mg/dL<br>4 hour: 70–115 mg/dL |
| Glycosylated hemoglobin (HbA1c) | Measurement of HbA1c provides a picture of the state of hyperglycemia over time in an individual (the 120-day life span of the red blood cell), and elevated levels correlate with glucose intolerance in diabetics. | Nondiabetic: 4–5.6%<br>Risk of diabetes: 5.7–6.4%<br>Diabetes: 6.5%+<br>American Diabetes Association recommends HbA1c < 7.0% for individuals diagnosed with diabetes. |
| Total cholesterol, including HDL and LDL components | Individuals with diabetes are at a greater risk for cardiovascular complications, including elevated blood lipid levels. These measurements are performed regularly to minimize diabetes-related cardiovascular complications. | Total cholesterol: <200 mg/dL *Or* <5.2 mmol/L<br>Total cholesterol (younger than age 20 years): < 180 mg/dL<br>HDL: Male: > 45 mg/dL<br>Female: > 55 mg/dL<br>LDL: < 100 mg/dL |
| Triglycerides | Generally included as part of a lipid profile along with HDL and LDL cholesterol, but has little predictive value by itself and increases after dietary fat consumption. | <150 mg/dL |
| Urine glucose tests | A measure of the amount of glucose in the urine. Urinary threshold maximums vary among individuals, and glycosuria is not a true indicator of serum glucose levels. | No glucose should be found in the urine. Urinary threshold levels vary among individuals. Changes in normal urine glucose levels are sometimes monitored. |

(*continued*)

### Laboratory Tests Commonly Administered to Individuals at Risk for or Diagnosed with Diabetes *(continued)*

| Laboratory Measurement | Description and Significance | Accepted Laboratory Value Range |
|---|---|---|
| Urine ketone tests | Ketonuria of sufficient concentration to produce a positive response in testing reflects an alteration in carbohydrate metabolism with secondary disturbances in lipid metabolism. Diabetes is the only disease in which ketonuria has a true diagnostic importance. The presence of ketonuria in diabetes is a major indicator of impending or established ketoacidosis. | *Normal:* Negative for all age groups. |

The following table reviews numerous diabetes-related complications, their symptoms, and treatment protocols.

### Diabetes-Related Complications and Common Treatment Protocols

| Diabetes-Related Complications | Description | Treatment Protocols |
|---|---|---|
| Diabetic coma | A coma caused by the buildup of ketones in the bloodstream. Ketones are a product of incomplete metabolism of fats resulting from the absence of glucose energy. | The best treatment for a diabetic coma is prevention. Careful diet, medication, and insulin dosing is needed to prevent ketone buildup. Patients with diabetes need to be aware of the early signs of ketone buildup such as weight loss, nausea, confusion, gasping for breath, confusion, convulsions, and a characteristically sweet chemical odor to one's breath similar to that of acetone or alcohol ("acetone breath"). |
| Glycosuria | Elevated blood glucose leads to spillage of glucose into the urine (glycosuria) so that the urine is sugary. (The term *diabetes mellitus* means "sweet urine.") | High blood sugar may produce few or no symptoms. When there are symptoms, they may be dry mouth, thirst, frequent urination, blurry vision, fatigue or drowsiness, weight loss or increased appetite. An individual with any of these symptoms needs to see a healthcare provider for a thorough medical assessment. |
| Hyperglycemia | High blood sugar (glucose). Hyperglycemia occurs when the body does not have enough insulin or cannot use the insulin it has to turn glucose into energy. Hyperglycemia may also occur in Cushing syndrome and other conditions. The signs of hyperglycemia are polydipsia (a great thirst), polyuria (frequent urinate), and dry mouth. | Individual meal management, physical activity, and possible drug therapy. See the following sections. |
| Hypoglycemia | Low blood sugar (glucose). When symptoms of hypoglycemia occur together with a documented blood glucose under 45 mg/dL, and the symptoms promptly resolve with the administration of glucose, the diagnosis of hypoglycemia can be made with some certainty. The causes of hypoglycemia include drugs (such as insulin), liver disease, surgical absence of the stomach, tumors that release excess amounts of insulin, and pre-diabetes. In some patients, symptoms of hypoglycemia occur during fasting (fasting hypoglycemia). In others, symptoms of hypoglycemia occur after meals (reactive hypoglycemia). | Hypoglycemia is only significant when it is associated with symptoms.<br>The symptoms may include anxiety, sweating, tremor, palpitations, nausea, pallor, headache, mild confusion, abnormal behavior, loss of consciousness, seizure, and coma. Immediate treatment of severe hypoglycemia consists of administering large amounts of glucose, and repeating this treatment at intervals if the symptoms persist. Treatment must also be directed at the underlying cause. Patients with diabetes mellitus who develop low blood glucose from their medicines require medication adjustments. Treatment of reactive hypoglycemia consists of dietary measures, including fewer concentrated sweets and the ingestion of multiple protein-rich, small meals throughout the day. |

| Diabetes-Related Complications | Description | Treatment Protocols |
|---|---|---|
| Ketoacidosis | A feature of uncontrolled diabetes mellitus characterized by a combination of ketosis and acidosis. Ketosis is the accumulation of substances called ketone bodies in the blood. Acidosis is increased acidity of the blood.<br>Symptoms of ketoacidosis include slow, deep breathing with a fruity odor to the breath, confusion, frequent urination (polyuria), poor appetite, and eventually loss of consciousness. | The treatment of ketoacidosis is a matter of urgency and is usually done in a hospital. It may require the administration of intravenous fluids, insulin, and glucose and the institution of changes in the person's diet. |
| Nephropathy | Kidney disease associated with long-standing diabetes. It affects the network of tiny blood vessels (the microvasculature) in the glomerulus, a key structure in the kidney that is composed of capillary blood vessels and that is critically necessary for the filtration of the blood. Features of diabetic nephropathy include the nephrotic syndrome with excessive filtration of protein into the urine (proteinuria), high blood pressure (hypertension), and progressively impaired kidney function. When severe, diabetic nephropathy leads to kidney failure, end-stage renal disease, and the need for chronic kidney dialysis or a kidney transplant. | 0.8 g/kg of protein, if glomerular filtration rate is normal. 0.6 g/kg of protein if glomerular filtration rate declines. |
| Polydipsia | Excessive thirst. Polydipsia occurs in untreated or poorly controlled diabetes mellitus. | Can be alleviated by treating elevated blood sugar levels. |
| Polyphagia | Frequent hunger and subsequent eating. | Management of blood sugar levels can help manage appetite. When insulin is not functioning well or is not produced in the body at all, the glucose in the blood cannot get into the body cells to feed them. As a result, the cells are starving and send a message to the brain to increase appetite. |
| Polyuria | The passage of an abnormally large amount of urine. Polyuria is a "classic textbook" sign of diabetes mellitus that is under poor control or not yet under treatment. | Treatment of hyperglycemia will alleviate polyuria. |
| Retinopathy | A common complication of diabetes affecting the blood vessels in the retina (the thin, light-sensitive membrane that covers the back of the eye). If untreated, it may lead to blindness. If diagnosed and treated promptly, blindness is usually preventable. Diabetic retinopathy begins without any noticeable change in vision. But even then there are often extensive changes in the retina visible to an ophthalmologist (eye doctor). It is therefore important for a diabetic person to have an eye examination at least once (ideally twice) a year. | Treatment is by laser surgery, usually on an outpatient basis. The nature of the laser treatment depends on the stage of the retinopathy. Laser therapy can only stop the progression of diabetic retinopathy. It cannot reverse the damage already done. The progression of the retinopathy can be slowed down by careful control of the diabetes, effective reduction of high blood pressure together with regular eye exams, and, if needed, prompt laser therapy. |

### The Role of Insulin and Oral Hypoglycemic Medications in the Treatment of Type 1 and Type 2 Diabetes

The major goal in treating diabetes mellitus is controlling elevated blood sugars (glucose) without causing abnormally low levels of blood sugar. Type 1 diabetes mellitus is treated with insulin, exercise, and management of total carbohydrate intake. Insulin is the mainstay of treatment for patients with type 1 diabetes mellitus. Insulin is also important in type 2 diabetes when blood glucose levels cannot be controlled by diet, weight loss, exercise, and oral hypoglycemic agents.

Ideally, insulin medication should be administered in a manner that mimics the natural pattern

of insulin secretion by a healthy pancreas. The complex pattern of insulin secretion by the pancreas is difficult to duplicate. Still, adequate blood glucose control can be achieved with careful attention to diet, regular exercise, home blood glucose monitoring, and multiple insulin injections throughout the day.

Type 2 diabetes mellitus is first treated with weight reduction, a diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral hypoglycemic agents are used. If oral medications are still insufficient, insulin medications are considered. Based on what is known, oral hypoglycemic agents for type 2 diabetes are designed to do the following:

- Increase the insulin output by the pancreas
- Decrease the amount of glucose released from the liver
- Increase the sensitivity (response) of cells to insulin
- Decrease the absorption of carbohydrates from the intestine

Adherence to a diabetic diet is an important aspect of controlling elevated blood sugar in patients with diabetes mellitus. The American Diabetes Association has provided guidelines for a diabetic diet that is a balanced, nutritious diet low in fat, cholesterol, and simple sugars. The total daily calories are evenly divided among three meals and snacks. The American Diabetes Association has lifted the absolute ban on simple sugars. Small amounts of simple sugars are allowed when consumed with a well-balanced meal containing carbohydrates, healthy fats, and protein.

Weight reduction and exercise are important treatments of diabetes. Weight reduction and exercise increase the body's sensitivity to insulin, thus helping to control blood sugar elevations.

The following charts introduce common characteristics of insulin and some commonly prescribed oral hypoglycemic agents. This information should be considered when planning meal and medication times.

### Characteristics of Various Insulin Preparations

| Generic Name (Brand) | Onset | Peak | Duration |
|---|---|---|---|
| Rapid acting (Lispro) | 5–15 minutes | 30–60 minutes | 2–4 hours |
| Short acting (regular) | 30 minutes | 2–5 hours | 6–8 hours |
| Intermediate acting (NPH, Lente) | 1–2½ hours | 8–14 hours | 22 hours |
| Long acting (Ultralente) | 4–6 hours | 10–18 hours | 24–36 hours |

### Characteristics of Commons Hypoglycemic Agents

| Agent | Information |
|---|---|
| Precose | The name of the alpha-glucosidase inhibitor available in the United States is Precose. Because Precose works on the intestine, its effects are additive to diabetic medications that work at other sites, such as sulfonylureas. Clinical studies have shown statistically better blood glucose control in patients treated with Precose and a sulfonylurea versus the sulfonylurea alone. Precose is currently used alone or in combination with a sulfonylurea. Precose is taken three times a day at the beginning of meals. The dosage varies from 25 to 100 mg with each meal. The maximum recommended dose is 100 mg three times a day. At doses greater than this, reversible liver abnormalities may be seen. Because of its mechanism of action, Precose has significant gastrointestinal side effects. Abdominal pain, diarrhea, and gas are common and are seen in up to 75% of patients taking Precose. For this reason, Precose is administered using a low initial dose that is increased over weeks depending on the patient's tolerance. |
| Sulfonylureas | Historically, increasing the insulin output by the pancreas has been the major area targeted by medications used to treat type 2 diabetes. These medications belong to a class of drugs called sulfonylureas. Sulfonylureas primarily lower blood glucose levels by increasing the release of insulin from the pancreas. Older generations of these drugs include chlorpropamide and tolbutamide, whereas newer drugs include glyburide (DiaBeta), glipizide (Glucotrol), and glimepiride (Amaryl). These drugs are effective in rapidly lowering blood sugars but run the risk of causing hypoglycemia. In addition, they are sulfa compounds and should be avoided in patients with sulfa allergies. |

| Agent | Information |
|---|---|
| Meglitinides | This class of drugs works on the pancreas to promote insulin secretion. Unlike sulfonylureas that bind to receptors on the insulin-producing cells, meglitinides work through a separate potassium-based channel on the cell surface. Repaglinide (Prandin) and nateglinide (Starlix) are short-acting agents that are taken 30 minutes before meals. Unlike the sulfonylureas, which last longer in the body, Prandin and Starlix are very short acting, with peak effects within 1 hour. For this reason they are given up to three times a day just before meals. Because these drugs also increase circulating insulin levels, they may also cause hypoglycemia, but the literature suggests this is less frequent than the hypoglycemia seen with sulfonylurea agents. |
| Biguanides | This class of drugs has been used for many years in Europe and Canada. In 1994, the FDA approved the use of metformin (Glucophage) for the treatment of type 2 diabetes in the United States. Glucophage is unique in its ability to decrease glucose production from the liver. Briefly, because metformin does not increase insulin levels, when used alone it does not usually cause hypoglycemia. In addition, metformin has an effect whereby it tends to suppress appetite, which may be beneficial in this population. Metformin may be used by itself or in conjunction with other oral agents or insulin. It should not be used in patients with kidney impairment and should be used with caution in those with liver impairment. The older parent compounds of metformin were associated with a serious condition called lactic acidosis with a dangerous acid buildup in the blood resulting from accumulation of the drug and its breakdown products. Although metformin is safer in this regard, it is recommended that the drug be discontinued for 24 hours before any dye-related procedure (such as intravenous pyelogram kidney study) or surgery is performed. The dyes may impair kidney function and cause a buildup of the drug in the blood. Metformin can be restarted after these procedures once the patient has urinated normally. |
| Thiazolidinediones | At present in the United States, the class of drugs known as thiazolidinediones lowers blood glucose by improving target cell response to insulin (increasing the sensitivity of the cells to insulin). Troglitazone (Rezulin) was the first of this type of compound introduced in the United States. Sister compounds are now available with a better safety profile. These drugs include pioglitazone (Actos) and rosiglitazone (Avandia).<br><br>Pioglitazone (Actos) and rosiglitazone (Avandia) are new thiazolidinediones that have been approved for use in the United States. Although they are sister compounds to Rezulin, extensive studies failed to show any liver problems associated with these particular drugs. Both Avandia and Actos act by increasing the sensitivity (responsiveness) of cells to insulin. They improve sensitivity to insulin in muscle and fat tissue. These drugs have been effective in lowering blood sugars in patients with type 2 diabetes; Actos and Avandia act within 1 hour of administration and are dosed daily. It is important to note that it takes up to 6 weeks to see a drop in blood glucose levels on these agents and up to 12 weeks to see a maximum benefit. Actos and Avandia have been approved as first-line therapy in diabetes and for use in combination. Both medications may be used in patients taking other oral agents as well as those using insulin.<br><br>As an aside, Actos and Avandia have an added benefit of changing cholesterol patterns in diabetes. HDL, or good, cholesterol increases on these medications and triglycerides often decrease, but there is some controversy regarding what happens to LDL, or bad, cholesterol. |

The following table describes the different options for insulin administration, including the rationale for use of insulin pumps, intensive insulin therapy, and multiple daily insulin injections.

### Insulin Administration Modalities

| Insulin Modality | Description |
|---|---|
| Prefilled insulin pens | In the past, insulin was available only in an injectable form. This involved carrying syringes (which a few decades ago were made of glass and required sterilization), needles, vials of insulin, and alcohol swabs. Needless to say, patients often found it difficult to take multiple shots a day, and as a result good blood sugar control was often compromised. Many pharmaceutical companies now offer discreet and convenient methods of insulin delivery. Both Novo Nordisk and Lily have an insulin pen delivery system. This system is similar to an ink cartridge in a fountain pen. A small pen-sized device holds an insulin cartridge (usually containing 300 units). |

(*continued*)

### Insulin Administration Modalities *(continued)*

| Insulin Modality | Description |
|---|---|
| | Cartridges are available in the most widely used insulin formulations, such as those listed in the preceding table. The amount of insulin to be injected is dialed in by turning the bottom of the pen until the required number of units shows in the dose-viewing window. The tip of the pen consists of a needle that is replaced with each injection. A release mechanism allows the needle to penetrate just under the skin and deliver the required amount of insulin. The cartridges and needles are disposed of when finished and new ones are simply inserted. These insulin-delivery devices are discreet and less cumbersome than traditional methods. |
| Insulin pump | The most recently available advance in insulin delivery is the insulin pump. In the United States, MiniMed, Deltec, and Disetronic market the insulin pump. An insulin pump is composed of a pump reservoir similar to that of an insulin cartridge, a battery-operated pump, and a computer chip that allows the user to control the exact amount of insulin delivered. Currently, pumps on the market are about the size of a beeper. The pump is attached to a thin plastic tube (an infusion set) that has a soft cannula (or needle) at the end through which insulin passes. This cannula is inserted under the skin, usually on the abdomen. The cannula is changed every 2 days. The tubing can be disconnected from the pump while showering or swimming. The pump is used for continuous insulin delivery, 24 hours a day. The amount of insulin is programmed and is administered at a constant rate (basal rate). Often, the amount of insulin needed over the course of 24 hours varies, depending on such factors as exercise, activity level, and sleep. The insulin pump allows for the user to program many different basal rates to allow for this variation in lifestyle. In addition, the user can program the pump to deliver a "bolus" during meals to cover the excess demands of carbohydrate ingestion. More than 50,000 people worldwide are using an insulin pump. This number is growing dramatically as these devices become smaller and more user-friendly. Insulin pumps allow for tight blood sugar control and lifestyle flexibility while minimizing the effects of low blood sugar (hypoglycemia). At present, the pump is the closest device on the market to an artificial pancreas. Naturally, the next step would be a pump that can also sense blood sugar levels and adjust the insulin delivery accordingly. Much effort is being concentrated on this area of research, and possibly, even within the next year, a prototype device may be available for trial. |
| Insulin inhalation | Another promising route of insulin administration is through inhalation. Inhaled insulin is currently being tested, but has not been approved by the FDA. Many devices are available that allow for other medications to be used in this manner, the best example of which is asthma therapy. Insulin is not absorbed through the bronchial tubes (airways) and must reach the air sacs at the end of the bronchial tubes (alveoli) to be absorbed. Once at the alveoli, insulin can be absorbed and enter the bloodstream. Currently, powdered inhalers and nebulizers are being studied to determine which delivery system is the most reliable. The safety of inhaled insulin still needs to be established before a product for consumer use can be made available. Trials are currently under way to establish the safety of inhaled insulin. |

The following table addresses issues commonly discussed as part of the dietary management of type 1 and type 2 diabetes.

### Common Considerations in the Dietary Management of Diabetes

| | |
|---|---|
| Timing and frequency of meals and snacks | This prevents spikes in blood glucose levels and maintains normal blood glucose levels. Eating meals at about the same time every day helps keep blood sugar levels in the target range. Eating snacks during the day and before, during, or after exercise is necessary as well. This prevents or delays the start of diabetes complications such as nerve, eye, kidney, and blood vessel damage. |
| Managing sick days | When sick, stress levels are increased. To deal with this stress, the body releases hormones that help it fight disease. These hormones have side effects. Blood sugar levels increase and interfere with the blood sugar–lowering effects of insulin. Usual doses of insulin should be taken when sick. Monitoring of blood glucose levels and urine testing for ketones should be done four times daily. If regular foods are not tolerated, liquid or soft cholesterol-containing foods should be eaten. At least 50 g of cholesterol should be consumed every 3 to 4 hours in small, frequent feedings. Ample amounts of liquid should be consumed every hour. The healthcare team should be called if illness continues for more than 1 day. |

| | |
|---|---|
| Necessary changes to medication administration and food choices when physical activity is added to the equation | Blood glucose should be checked twice before exercise. If insulin or diabetes pills are taken, blood glucose monitoring is important to avoid low blood glucose levels. Blood glucose should be monitored 30 minutes before and again just before exercise. Check during and after exercise also. Plan exercise and diabetes care to avoid levels that are too low or too high. It is best to exercise 1 to 3 hours after a meal. Avoid exercising when insulin is at peak. Check for ketones whenever blood glucose levels are too high. |
| Alcohol consumption and its influence on the diabetic condition | The risk of low blood sugars is greater when alcohol is consumed. Alcohol should not be consumed on an empty stomach but with a meal or after eating a snack. Drinking as little as 2 oz of alcohol on an empty stomach can lead to very low blood glucose. It is important to check blood glucose before sleep. A snack should be eaten to avoid hypoglycemia while sleeping. |
| The use of artificial sweeteners in the dietary treatment of diabetes | This can be part of a healthful eating plan, but it must be included as part of the daily carbohydrate allowance. Sucrose and sucrose-containing foods may be used in moderation as part of a balanced meal plan. Fructose appears to produce a lower glycemic response than the same amounts of sucrose or other cholesterol but should be consumed in moderation. The common sugar alcohols (polyols) sorbitol, mannitol, and xylitol produce a lower glycemic response than sucrose and other carbohydrates. |

### Carbohydrate Counting

Carbohydrate counting, or carb counting, is a meal planning system in which a person can eat a specific number of carbohydrate grams at each meal or snack. Although it is an effective plan for those tracking their diet, it is especially helpful for people with diabetes. Carb counting is easy to learn. Most people believe it gives them flexibility in food choices, making social situations and eating out easier. The most compelling reason to use carb counting is simply because it can improve blood glucose control. Before starting it is advised that individuals with diabetes consult a dietitian or healthcare provider. There is no meal planning approach that is right for everyone—each person needs an individualized plan. Carbohydrates are the basis of all the food groups, so they are found in nearly every food. With carb counting, the source of the carbohydrate is not as important as how much of it is eaten. That is good news for people with diabetes because carbohydrates are in starches, fruits, dairy products, vegetables, and sugary sweets. This plan lets individuals eat a wide variety of foods, as long as they stay within their per-meal allowances of carbohydrates.

### How Carb Counting Works

1. *Make a plan:* Determine the number of carbohydrate grams an individual should have at each meal or snack and whether to count "grams" or "choices." Grams are counted simply by adding the total carbohydrate content in each food serving. Choices, like the familiar exchanges, are selections from food groups. One choice equals 15 g of carbohydrates. Choices are less precise than grams but easier to use and accurate enough for people who do not take insulin. Each person's plan differs, and the following chart shows just one example:

| | Choices | Grams |
|---|---|---|
| Breakfast | 3–4 | 45–60 |
| Lunch | 4–5 | 60–75 |
| Dinner | 5–6 | 75–90 |
| Snacks | 0–1 | 0–15 |

2. *Learn the carbohydrate counts of foods:* Person with diabetes need their blood glucose meter, a food scale, measuring cups and spoons, carbohydrate content guidebooks, and the Nutrition Facts on food labels. They will use these essential tools at every meal while learning the system and then only at a few meals each week once they become comfortable with measuring.

   If you are counting choices and are familiar with the exchange system, you can easily memorize these converted measurements:

   1 starch exchange = 15 g = 1 choice
   1 fruit exchange = 15 g = 1 choice
   1 milk exchange = 12 g = 1 choice
   3 vegetable exchanges = 15 g = 1 choice
   1 meat exchange = 0 g = 0 choice
   1 fat exchange = 0 g = 0 choice

   In a typically healthy diet, 45% to 65% of the day's total calories come from carbohydrates.

The rest come from protein (10–35% of total daily calories) and fat (20–35% of total daily calories), which do not contain any carbohydrates and therefore are not added to the total carbohydrate gram count.

### Anemia: A Common Deficiency Disease in the United States

Anemia is a condition in which the body does not have enough healthy red blood cells. Red blood cells are crucial for oxygenating the body's millions of cells and tissues. The bone marrow, the soft tissue inside bones, is the primary producer of red blood cells. Healthy blood cells survive between 90 and 120 days in the body, after which time old red blood cells are removed from circulation. The hormone erythropoietin is made in the kidneys and signals the bone marrow to synthesize red blood cells. The body requires numerous nutrients to assist in the production of red blood cells with iron. The following questions review major concepts surrounding anemia caused by deficiencies of nutrients essential for normal red blood cell production with a primary focus on iron, vitamin $B_{12}$, and folic acid.

1. *Which nutrients are required for red blood cell formation and maturation, other than protein?* Folic acid (folate), with vitamin $B_{12}$, is necessary for the formation of normal red blood cells and the synthesis of DNA, the genetic material of cells. Iron is also essential for production of the protein molecule in RBCs called hemoglobin, the oxygen- and carbon-dioxide-carrying protein inside red blood cells. Hemoglobin imparts the red color characteristic of red blood cells. Without adequate hemoglobin red blood cells cannot carry oxygen from the lungs to the body's tissues or return carbon dioxide from the tissues to the lungs.
2. *How does a deficiency of folic acid lead to anemia?* Folic acid deficiency causes macrocytic anemia in which the red blood cells are fewer in number, larger in size, and contain less oxygen-carrying hemoglobin than normal. The symptoms of anemia are lethargy, apathy, breathlessness, poor body temperature regulation, pallor, forgetfulness, irritability, and stomach disorders.
3. *What is the name for folic acid deficiency anemia?* Megaloblastic anemia.
4. *What other nutrient deficiency can cause this type of anemia?* Vitamin $B_{12}$.
5. *What are the best sources of folic acid?* Most of the folic acid in foods (with the exception of the folic acid added to enriched flour and breakfast cereals) occurs as folate. Folate is only about half as available for the body to use as is the folic acid in pills and supplements. Folate also is easily destroyed by sunlight, overcooking, or the storing of foods at room temperature for an extended period of time.
   Good dietary sources of folate include the following:

   Leafy green vegetables
   Liver
   Mushrooms
   Oatmeal
   Peanut butter
   Red beans
   Soy
   Wheat germ
   Enriched grain products
   Citrus fruits (e.g., orange juice, grapefruit)
6. *Why is there an expanded need for folate during pregnancy?* Folic acid deficiency has been implicated as a cause of neural tube defects in the developing fetus. Recent research has shown that adequate amounts of folic acid can prevent up to half of these birth defects if women start taking folic acid supplements shortly before conception. It is important for all women attempting to become pregnant to take folic acid supplementation. It is best to begin taking supplements 3 months before attempting pregnancy; if already pregnant, begin immediately. Folic acid is contained in almost all prenatal vitamins.
   Besides helping to prevent certain birth defects, folic acid plays other important roles during pregnancy. A pregnant woman needs extra folic acid to help her to produce the additional blood cells she needs. Folic acid also is crucial to support the rapid growth of the placenta and fetus. This vitamin is needed to produce new DNA (genetic material) as cells multiply. Without adequate amounts of folic acid, cell division could be impaired, possibly leading to poor growth in the fetus or placenta.
7. *What is the Recommended Dietary Allowance for folic acid in pregnancy?* The March of Dimes recommends that all women who can become pregnant take a multivitamin that contains 400 µg of folic acid every day and eat a

healthy diet. This is the only sure way a woman can get all the folic acid and other vitamins she needs. Most women get less than half of the recommended amount of folic acid daily. The Institute of Medicine recommends that women increase their intake of synthetic folic acid to 600 µg/day once their pregnancy is confirmed. Most doctors recommend a prenatal vitamin that contains at least this amount of folic acid. However, women should not take more than 1,000 µg (or 1 mg) without their doctor's advice. The Institute of Medicine also recommends that women eat a diet rich in foods that contain folate or folic acid. Folate is the natural form of folic acid that is found in foods. Orange juice, other citrus fruits and juices, leafy green vegetables, beans, peanuts, broccoli, asparagus, peas, lentils, and whole-grain products all contain folate. Synthetic (manufactured) folic acid is added to certain grain products, including flour, rice, pasta, cornmeal, bread, and cereals. These foods are considered to be "fortified" with folic acid.

8. *Women who have prolonged use of oral contraceptives before pregnancy may have low serums levels of folate. Why?* In several cases women taking oral contraceptives developed folic acid deficiency. However, it appears that many of these women had low intake of folic acid or problems with intestinal absorption before taking birth control pills. Again, women on birth control pills should regularly eat good sources of folic acid. Good folate nutrition is especially important for women who become pregnant shortly after they stop taking oral contraceptives.
9. *Vitamin $B_{12}$ deficiency is rare. Why?* Unlike other water-soluble nutrients, vitamin $B_{12}$ is stored in the liver, kidneys, and other body tissues. It can take several years before signs of the deficiency appear, all because of poor dietary intake, so nutritional deficiency of this vitamin is extremely rare.
10. *When is a deficiency of vitamin $B_{12}$likely to occur?* Diets of most adult Americans provide recommended intakes of vitamin $B_{12}$, but a deficiency can occur in individuals with dietary patterns that exclude animal or fortified foods. As a general rule, most individuals who develop a vitamin $B_{12}$ deficiency have an underlying stomach or intestinal disorder that limits the absorption of vitamin $B_{12}$. There are two major steps required in vitamin $B_{12}$ absorption. Vitamin $B_{12}$ is combined with protein in animal foods. Vitamin $B_{12}$ must be separated from dietary protein, which occurs upon exposure to stomach acid. As an individual ages, the production of stomach acid declines so that the separation of protein and vitamin $B_{12}$ is limited. As many as 30% of older people may lack sufficient stomach acid to absorb adequate amounts of vitamin $B_{12}$ from natural sources. This can also occur in individuals who are chronic users of acid-suppressing drugs such as Prilosec, Prevacid, Nexium, Pepcid, and Tagamet. Fortunately, synthetic $B_{12}$, found in supplements and fortified foods, does not depend on stomach acid to be absorbed. There is another step required for natural and synthetic vitamin $B_{12}$ absorption. Vitamin $B_{12}$ must be combined with a substance in the stomach called intrinsic factor to be absorbed through the gut. This factor may be limited in individuals with a condition called pernicious anemia. Either a lack of stomach acid or intrinsic factor can reduce vitamin $B_{12}$ absorption and potentially lead to a $B_{12}$ deficiency.
11. *What are classic signs and symptoms of a vitamin $B_{12}$ deficiency?* Characteristic signs of $B_{12}$ deficiency include fatigue, weakness, nausea, constipation, flatulence, loss of appetite, and weight loss. Deficiency also can lead to neurologic changes such as numbness and tingling in the hands and feet. Additional symptoms of $B_{12}$ deficiency are difficulty in maintaining balance, depression, confusion, poor memory, and soreness of the mouth or tongue. Some of these symptoms can also result from a variety of medical conditions other than vitamin $B_{12}$ deficiency. It is important to have a physician evaluate these symptoms so that appropriate medical care can be given.
12. *Maternal iron deficiency does not usually result in an infant who is anemic at birth. Why?* An infant gets all the iron he or she needs from the mother's iron stores. The only way an infant may be anemic at birth is if the mother is severely anemic and has minimal iron to offer the fetus. Iron-deficiency anemia is a risk for the mother during pregnancy because the infant draws on the mother's iron stores and also the mother requires more iron to support her expanded blood volume. It is important to note that iron deficiency during pregnancy is

associated with an increased risk of preterm delivery and low-birth-weight infants.

13. *What is hematocrit?* The hematocrit is the percentage of whole blood that is composed of red blood cells. The hematocrit is a measure of both the number of red blood cells and the size of red blood cells. The hematocrit is almost always ordered as part of a complete blood count, which measures the number of red blood cells, the number of white blood cells, the total amount of hemoglobin in the blood, and the fraction of the blood composed of red blood cells (hematocrit).
14. *Around 3 to 5 months, lower levels of hemoglobin are normal in the pregnant women. Why?* Anemia in pregnancy is very common and is present in almost 80% of pregnant women. Because the volume of blood increases during pregnancy (hemodilution), a moderate decrease in the concentration of red blood cells and hemoglobin is normal. The hematocrit value (the percentage of red blood cells relative to plasma volume) in nonpregnant women ranges from 38% to 45%. However, in pregnant women, because of hemodilution normal values can be much lower, for example, 34% in single and 30% in twin or multiple pregnancies even with normal stores of iron, folic acid, and vitamin $B_{12}$. This lower range simply reflects "the physiologic hemodilution of pregnancy" and does not indicate a decrease in oxygen-carrying capacity or true anemia.
15. *What are acceptable levels of hematocrit and hemoglobin in pregnancy? In nonpregnant women?* Nonpregnant: Hb 12.0, Hct 36; pregnant first trimester: Hb 11.0, Hct 33; pregnant second trimester: Hb 10.5, Hct 32; pregnant third trimester: Hb 11.0, Hct 33. During the third trimester, it is almost impossible to get enough iron from the diet, which means that the mother's iron stores will be drawn on to meet the demand.
16. *What is the recommended level of iron supplementation in pregnancy?* 27 mg/day
17. *How much iron is typically found in breast milk?* Breast milk is a complex composition of important proteins, fatty acids, sugars, amino acids, iron, and many other nutrients that are tailored to meet an infant's specific and changing needs. By 6 months of age, though breast milk is still an excellent source of nutrition for the infant, it no longer provides the entire range of nutrients needed for continued growth. Between the 4th and the 6th month an infant's diet should begin to include solid foods that provide the extra calories and nutrients (especially iron) that breast milk alone cannot. When taken in combination with solid foods, breast milk remains an excellent source of nutrition for infants for as long as breastfeeding continues.
18. *To diagnose iron-deficiency anemia, what signs and symptoms would you look for both physically and from a laboratory standpoint?* With iron-deficiency anemia there is a history of weakness, fatigue, tachycardia, dyspnea, and pallor from anemia and tissue iron depletion. Physical findings include pallor, tachycardia, glossitis, koilonychia (spoon nails), and angular stomatitis. Signs and symptoms of an underlying disease may be present. Laboratory studies reveal (1) decreased reticulocyte count and mean corpuscular volume, (2) hypochromic microcytic cells on blood smears, (3) low serum ferritin, and (4) hypercellular marrow with absent sideroblasts and storage iron.
19. *How long does it take for a woman to replace iron stores after pregnancy?* Birth is associated with blood loss. If a woman is anemic during pregnancy, she should take iron for several months after delivery to help the body replace the lost blood cells and iron stores. Breastfeeding women may also need to take iron because iron is lost in breast milk. How long it takes a woman to replace her iron stores depends on how deficient she became during pregnancy, but generally between 3 and 6 months post pregnancy a woman's iron stores should be back to normal.
20. *What are the three most common causes of anemia?* There are a number of different causes and types of anemia. Some of the more common causes of anemia include the following:

    - Increased loss of red blood cells
      - Increased blood loss from specific diseases (ulcers, cancers, certain types of infection)
      - Menstruation, trauma, or hemorrhoids
      - Certain medications that increase or cause bleeding, such as aspirin or nonsteroidal anti-inflammatory drugs (e.g., ibuprofen, naproxen)

- Increased need for hemoglobin or red blood cells
  - Chronic diseases, such as arthritis, kidney failure, inflammatory bowel disease, and liver disease
  - Specific vitamin/mineral deficiencies, such as iron, vitamin $B_{12}$, and folic acid
  - Conditions where nutrient absorption is a problem, such as with celiac disease
  - Excessive alcohol ingestion
  - Decreased activity of the bone marrow
  - Kidney disease or kidney failure
  - Chemotherapy medications
- Increased destruction of the red blood cells in the body (hemolysis)
  - Inherited (genetic) conditions, such as sickle cell disease
  - Conditions where the body's defense (immune system) causes destruction of the red blood cells

# Glossary



**acanthosis nigricans:** A velvety skin texture often found in skinfold areas that is frequently associated with obesity and type 2 diabetes.

**achlorhydria:** Absence or decrement in the level of hydrochloric acid in the stomach that results from decreased production by the parietal cells in the fundus of the stomach.

**acrocyanosis:** Blueness of the extremities (the hands and feet), caused by narrowing (constriction) of small arterioles (tiny arteries) toward the end of the arms and legs.

**adrenergic agonists:** Bind to and activate adrenergic receptors.

**advance directive:** Information in written or oral form provided by the patient that outlines the competent adult's wishes regarding medical treatment should he or she become incompetent in the future.

**affect:** Emotion.

**allergic disease:** Sensitization to allergens manifested as urticaria, angioedema, anaphylaxis, atopic dermatitis, respiratory symptoms, or gastrointestinal disorder.

**allergic eosinophilic esophagitis:** A disease involving patchy infiltration of one or more layers of the esophagus with eosinophils.

**allergic eosinophilic gastroenteritis:** A disease involving patchy infiltration of one or more layers of the stomach and/or small intestine with eosinophils.

**alpha-linolenic acid:** An essential 18-carbon omega-3 fatty acid.

**amenorrhea:** Absence of three consecutive menstrual cycles in a menarchal female.

**anaphylaxis:** Sudden, severe, potentially fatal systemic allergic reaction that can involve various areas of the body, such as the skin, respiratory tract, gastrointestinal tract, and the cardiovascular system.

**anemia:** A condition in which blood has a lower-than-normal red blood cell (RBC) count.

**anorexia:** Diminished appetite.

**anthropometrics:** Measurements of the human body.

**apoptosis:** Preprogrammed cell death.

**Arnold-Chiari malformation of the brain:** A structural disorder affecting the cerebellum, frequently found in spina bifida; can affect swallowing and gagging.

**aspiration:** Inhalation into the airways of fluid or foreign body; when food or liquid actually enters the lungs.

**attention deficit hyperactivity disorder (ADHD):** Neurobehavioral problem associated with learning disorders, hyperactivity, attention deficit, and inappropriate degrees of impulsiveness.

**autism spectrum disorders (ASDs):** A number of disorders that involve poor social interaction, impaired communication skills, a tendency to be repetitive, and sometimes mental retardation; found more in males than in females.

**autonomy:** The paramount ethical principle that states the competent adult has the right to make choices about treatment options.

**autosomal recessive:** Both parents must be carriers of a gene on one of the autosomal (nonsex) chromosomes for a child to inherit the disease. If both parents are carriers, there is a 25% chance that a child will inherit the disease and a 50% chance the child will be a carrier.

**bacteriostatic:** The characteristic of immune cells, which can incapacitate bacterial invaders through engulfing, dissolution, or direct toxicity.

**basal metabolic rate:** The minimal amount of energy required to sustain life in the waking state.

**beneficence:** The ethical principle of doing good; for example, proposing a therapy that is worthwhile for the patient (patient centered). This agrees with the ethical principle of nonmaleficence, which is "not doing any harm."

**beriberi:** Classic thiamine deficiency disease that affects the gastrointestinal tract, cardiovascular system, and peripheral nervous system and is usually confined to alcoholics.

**binge drinking:** The consumption of five or more standardized ethanol equivalents in one sitting. A standardized ethanol equivalent is 12 oz of beer, 4 oz of wine, or 1.5 oz of distilled spirits.

**bioavailability:** A measure of how available to the body a nutrient is after it is ingested.

**body habitus:** The size and shape of the body; may also include perceptions of the size and shape of the body.

**body mass index (BMI):** Calculated by dividing weight in kilograms by height in meters squared (BMI = $kg/m^2$); shown in the research literature to be correlated with adiposity and increased risk of chronic disease.

**bone mineral density:** A measurement of bone mass after development is complete; reported in grams per centimeter squared.

**bradycardia:** Abnormally slow heart rate (< 60 beats/minute) that does not meet the body's metabolic demands.

**Care Area Assessment (CAA):** The CAA is an analysis of the MDS data and provides a framework for guiding the review/assessment of 20 care areas. When an area is "triggered" by MDS data, further assessment is required to clarify the resident's functional status and determine the root cause of impairments to guide intervention.

**care plans:** As per the Centers for Medicare and Medicaid Services regulation, a facility must use an interdisciplinary approach to develop a comprehensive plan of care for each resident that includes measurable objectives and timetables to meet a resident's medical, nursing, mental, and psychosocial needs. These care plans must be revised as the resident's status changes.

**case-control studies:** A case series of individuals who have the disease of interest and a group of similar individuals who do not have the disease—exposure comparisons are made.

**cataract:** Complete or partial opacity of the ocular lens.

**Centers for Medicare and Medicaid Services:** The federal agency responsible for nursing home regulations and their enforcement. The Centers for Medicare and Medicaid Services is part of the U.S. Department of Health and Human Services.

**cerebral palsy (CP):** A disorder of motor control or coordination resulting from injury to the brain during its early development.

**chromosomal aberration:** A change in the makeup of a chromosome, often leading to a developmental disability.

**cobalamin:** Vitamin $B_{12}$.

**cohort studies:** Research studies that classify participants based on the presence or absence of exposure and follow them over time to assess disease development. An example of this type of study is the Nurses' Health Study. In a retrospective cohort study, the disease of interest has already occurred at the time the study begins; in a prospective cohort study, the disease has not yet occurred.

**competitive foods:** Those foods that are less nutritious, such as high-fat high-sugar snacks, soda, and other sweetened beverages. Easy access to these foods competes with healthier food choices for attention and consumption.

**complementary feeding:** The period that begins with the introduction of the first nonmilk food and ends with the cessation of breast-feeding or formula feeding.

**congenital anomalies:** Malformations present at birth that can affect various organs or structures of the body, such as a cleft lip or palate.

**corpulmonale:** A heart condition characterized by enlargement of the right side of the heart (right ventricle) and failure caused by pulmonary hypertension.

**corpus luteum:** A yellow glandular mass in the ovary formed by an ovarian follicle that has matured and released its egg; secrets progesterone.

**cross-sectional surveys:** Research that looks at individuals with respect to both exposure and disease at a point in time. Because both exposure and disease are assessed at the same time, it can be difficult to determine whether exposure predated disease.

**cytokines:** A class of cellular chemical messengers often involved in cascade reactions such as with inflammatory response.

**decubitus ulcer:** Focal ischemic necrosis of skin and underlying tissue at sites of constant pressure or recurring friction in patients immobilized by illness or disability.

**dementia:** The loss, usually progressive, of cognitive and intellectual function.

**dentition:** The natural teeth, as considered collectively.

**diaphyses:** Shafts of long bones.

**dietary disinhibition:** Unrestrained eating without regard to hunger or satiety.

**dilutional hyponatremia:** Low blood sodium concentrations; in athletes, typically resulting from sodium losses in sweat and excessive consumption of plain water during exercise.

**docosahexaenoic acid (DHA):** A 22-carbon omega-3 fatty acid found in fish oil.

**double-blind placebo-controlled design:** When *neither* the individuals nor the investigator knows whether participants are assigned to a treatment or a placebo group. This study design is usually considered to provide the most compelling evidence of a cause-and-effect relationship.

**Down syndrome:** An aberration of the 21st chromosome that causes mental retardation, low muscle tone, and other physical abnormalities.

**dysphagia:** Difficulty in swallowing.

**dyspnea:** Shortness of breath.

**edentulous:** Toothless; having lost the natural teeth.

**ego-syntonic:** Consistent with self-concept.

**eicosapentaenoic acid (EPA):** A 20-carbon omega-3 fatty acid found in fish oil.

**elimination diets:** Diets in which suspected foods are eliminated in an attempt to identify and/or confirm offending allergenic foods.

**endosteal resorption:** Removal of bone from the inner surface.

**energy expenditure:** Kilocalories expended during the day. Total energy expenditure comprises basal metabolic rate, dietary-induced thermogenesis, and the thermic effect of activity. A fourth component is nonexercise activity thermogenesis (NEAT).

**enteral nutrition:** The delivery of nutrients into the digestive system.

**epiphyses:** Ends of long bones.

**exclusive breast-feeding:** Infant receiving only breast milk and no other foods or drinks for the first 6 months.

**exercise:** Planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness.

**extremely low birth weight:** Birth weight of less than 1,000 g.

**fat-soluble vitamins:** Including vitamins A, D, E, and K; soluble in polar solvents, absorbed and transported with dietary lipids, and excreted in the feces.

**federal nursing home regulation for nutrition services:** Documents published by the Centers for Medicare and Medicaid Services for use in the survey process for long-term care facilities that focus on the nutritional oversight and delivery to the residents in the facility.

**federal regulations:** Documents published by the Centers for Medicare and Medicaid Services for use in the survey process for long-term care facilities.

**Feeding Infants and Toddlers Study (FITS):** This study was conducted on a large random sample of infants and toddlers to collect data on food choices and feeding practices and their impact on growth and development.

**fluorosis:** An abnormal condition caused by excessive intake of fluorides, characterized in children by discoloration and pitting (mottling) of the teeth.

**food allergy:** An immune-mediated abnormal response to food (specifically food proteins) triggered by the body's immune system with involvement of multiple systems in the body, including the skin, respiratory system, cardiovascular system, and/or the gastrointestinal tract.

**Food Code:** Published by the U.S. Food and Drug Administration, this is a model that assists food control jurisdictions at all levels of government by providing them with a scientifically sound technical and legal basis for regulating the retail and food service segment of the industry.

**food intolerance:** Any abnormal reaction to food that does not involve the immune system and is usually limited to the gastrointestinal system, such as lactose intolerance.

**fructose intolerance:** An inborn error of metabolism caused by the lack of the enzyme fructose-1-phosphate aldolase (aldolase B).

**fruitarian:** A person following a diet based on fruits, nuts, and seeds that often includes vegetables that are botanically fruits such as avocado and tomatoes.

**gag reflex:** Reflex stimulated by contact with the tongue that gradually diminishes over the first 6 months.

**galactosemia:** An inborn error of carbohydrate metabolism caused by a deficiency of galactosyl-1-phosphate uridyl-transferase, which inhibits glycogen breakdown and glucose synthesis, causing severe hypoglycemia after the ingestion of fructose.

**glomerular filtration rate:** The quantity of glomerular filtrate formed per unit in all nephrons of both kidneys.

**glycemic control:** Maintaining blood glucose within the target range.

**glycosylated hemoglobin (HbA1c):** A blood test that can reflect blood glucose control over a 3-month period. An A1cof 6% reflects an average plasma glucose level of about 120 mg/dL. In general, each 1% increase in A1c is a reflection of an increase in average glucose levels of about 30 mg/dL.

**gonadotropin:** Hormones secreted by the pituitary gland that affect the function of the male or female gonads.

**growth charts:** Graphs used to assess nutritional status of children by plotting height and weight and comparing these to reference data.

**gynecologic age:** Years since menarche.

**Hazard Analysis Critical Control Point (HACCP):** A process used in cooking to identify steps and points in food preparation that are deemed as critical in preventing foodborne illness.

**high-density lipoproteins:** Often referred to as "good cholesterol," a lipoprotein that contains mostly protein with less cholesterol and triglyceride. High levels of high-density lipoproteins in the blood are associated with a decreased risk of coronary heart disease.

**HIV wasting syndrome:** Catabolic condition; loss of body weight (body fat and muscle stores);very similar to cancer cachexia.

**holistic approach:** Individual lifestyle factors, social and political issues, and access to quality health care.

**homeostasis:** The tendency of the body to seek and maintain a condition of balance or internal stability.

**homocysteine:** An amino acid that can be measured in the blood that has been shown to be an independent risk factor for cardiovascular disease.

**hyperhomocystinemia:** Elevated blood levels of homocysteine, the sulfur-containing amino acid metabolite.

**hyperinsulinemia:** Blood levels of insulin above the normal range.

**hypertension:** Systolic blood pressure above 140 mm Hg or diastolic blood pressure above 90 mm Hg.

**hypochlorhydria:** Presence of an abnormally small amount of hydrochloric acid in the stomach.

**hypotonia:** Low tone of the muscles frequently found in Down syndrome, Prader-Willi syndrome, and other conditions such as prematurity.

**IgE- and non-IgE-mediated food allergies:** IgE-mediated allergies are characterized by an acute onset of symptoms that typically involve the skin, respiratory system, cardiovascular system, and/or the gastrointestinal tract. Non-IgE-mediated or cell-mediated allergies are slower in onset and primarily are gastrointestinal reactions.

**inborn errors of metabolism:** Traits arising from a variation in the structure of enzymes or protein molecules.

**incidence:** Rate of occurrence.

**incontinence:** Inability to control the discharge of urine or feces.

**insulin resistance:** An impaired biological response to either exogenous or endogenous insulin; plays a role in the etiology of type 2 diabetes.

**intervention studies:** A type of prospective cohort study where the exposure is controlled by the investigator. These studies can be considered therapeutic (secondary prevention) or preventative. Dietary Approaches to Stop Hypertension (DASH) and DASH-sodium are intervention studies.

**intrauterine growth restriction:** When an infant has not achieved his or her full in utero growth potential.

**iron deficiency:** The most common nutritional disorder in the world, with approximately 25% of the world's population being deficient.

**iron-deficiency anemia:** The most common childhood deficiency resulting in motor and cognitive developmental deficits.

**isovaleric acidemia:** A disorder of leucine metabolism caused by a deficiency of isovaleryl-CoA characterized by the excessive production of isovaleric acid upon ingestion of protein or during infectious episodes and resulting in severe metabolic acidosis.

**lacto-ovo-vegetarian:** A vegetarian who eats dairy products and eggs.

**lacto-vegetarian:** A vegetarian who eats dairy products but not eggs.

**lanugo:** Downy, very fine, soft, and usually unpigmented hair.

**leucopenia:** A decrease in the number of white blood cells circulating in the blood.

**limbic system:** A group of brain structures and their connections with each other as well as their connections with the hypothalamus and other areas; largely associated with emotions.

**long-chain pzzolyunsaturated fatty acids (LCPUFAs):** A category of dietary fats important in brain and retinal growth; normally present in human milk, they are now added to infant formula.

**lordosis:** An abnormal forward curvature of the lumbar spine.

**macrobiotic:** A vegetarian or near-vegetarian diet based largely on grains, legumes, and vegetables; may include limited amounts of fish.

**macrominerals:** Minerals that are needed in large amounts (calcium, magnesium, and phosphorus).

**macular degeneration:** An eye disease that affects the macula, a part of the retina.

**maple syrup urine disease (MSUD):** An autosomal recessive disorder characterized by a defect in the metabolism of the branched-chain amino acids isoleucine, leucine, and valine caused by a deficiency of branched-chain ↑-ketoacid dehydrogenase, resulting in the accumulation of the branched-chain amino acids in the plasma.

**medroxyprogesterone acetate:** A synthetic progestin.

**menarche:** First menstrual period.

**Menkes disease:** X-linked genetic disorder that is responsible for copper deficiency. Symptoms are characterized by hypothermia; neuronal degeneration; mental retardation; abnormalities in hair, skin, and connective tissue; bone fractures; and widespread vascular abnormalities, with death typically occurring by age 4 years.

**metabolic equivalent:** The ratio of the work metabolic rate to the resting metabolic rate. One metabolic equivalent is defined as 1 kcal/kg/h or 3.5 mL $O_2$ consumed/kg/min.

**metabolizable energy:** Gross energy in a food minus the energy lost in feces, urine, and combustible gases. The metabolizable energies for protein and carbohydrate are 4 kcal/g, and the metabolizable energy for fat is 9 kcal/g.

**metastasis:** Growth of malignant tissue that spreads to surrounding tissues or organs.

**methylation reactions:** The process of attaching a methyl group to an existing compound through 1 C transfer by several vitamin/mineral cofactors.

**minerals:** Inorganic elements not produced by plants and animals and primarily stored in bone and muscle tissues.

**Minimum Data Set (MDS):** An electronic summary of the medical status of each resident mandated by the Centers for Medicare and Medicaid Services.

**myocyte:** A single muscle cell.

**myostatin:** A growth factor that limits muscle tissue growth.

**n-3 fatty acid:** A polyunsaturated fatty acid in which the first double bond is three carbons from the methyl end of the carbon chain. Important sources in the U.S. diet include certain fish tissues, canola and soybean oils, and seeds and nuts such as flax seeds and walnuts.

**necrotizing enterocolitis (NEC):** An inflammation or death of the intestinal tract.

**neural tube defects:** Defects in the structure of the embryo that give rise to the brain, spinal cord, and other parts of the central nervous system as a result of inadequate folic acid intake during pregnancy.

**neurodegenerative disease:** Diseases that involve malfunction of the central and peripheral nervous tissue. These often are diseases that progress slowly but insidiously and result in progressive loss of function and eventual mortality. Examples include Alzheimer's disease and Parkinson's disease.

**neuropeptide Y:** A 36-amino-acid peptide neurotransmitter found in the brain and autonomic nervous system; it augments the vasoconstrictor effects of noradrenergic neurons.

**nonexercise activity thermogenesis (NEAT):** The energy expenditure of all physical activities other than volitional sporting-like exercise. NEAT includes activities such as working, playing, and dancing.

**nonheme iron:** The form of iron found in plants and the portion of iron from animal foods that is not part of hemoglobin or myoglobin.

**nosocomial:** Hospital-acquired infection, usually with an antibiotic-resistant strain of organism.

**nutritional rickets:** Vitamin D deficiency that was thought to have vanished but is reappearing.

**nutrition assessment:** A thorough evaluation of the resident's food and eating preferences, medical and nutritional status, estimation of nutritional needs, and identification of medical conditions/treatments and other factors that might make the resident at risk of developing malnutrition in the future. This information is used to develop the individualized plan of nutrition care for the resident and/or to determine whether the current nutritional regimen is adequate to meet the resident's nutritional needs.

**nutrition screening:** A quick review to determine whether the resident meets any known factors for nutrition risk that would require an immediate assessment by a registered dietitian.

**obesity:** Category of body weight above overweight; for children, a body mass index for age and gender percentile is used.

**obesity:** The universal definitions of overweight and obesity have been established using body mass index (BMI; kilograms of body weight/height [$m^2$]). For adults, a BMI between 25 and 29.9 is considered overweight, whereas a BMI of 30 or greater is defined as obese. Note that this is not the best definition for individuals.

**oligomenorrhea:** Irregular or infrequent menstruation in menarchal females; cycles occur at an interval of 35 days or greater, resulting in four to nine cycles per year.

**opportunistic infection:** Infection by an organism that would not ordinarily cause disease, but because of an impaired immune response the organism becomes pathogenic.

**oral food challenges:** A procedure during which the patient eats or drinks the suspected food allergen gradually in small portions over a given period of time under a physician's supervision.

**orthostatic hypotension:** A sudden fall in blood pressure that occurs when a person assumes a standing position, causing dizziness, lightheadedness, blurred vision, and syncope.

**osteopenia:** Bone mineral density that is lower than normal peak bone mineral density but not low enough to be classified as osteoporosis; often present in preterm infants, resulting in fractures.

**osteopenia of prematurity:** Reduction in bone volume to below normal levels, often seen in infants born prematurely.

**osteoporosis:** Reduction in the quantity of bone.

**parathyroid hormone:** Peptide hormone secreted by the parathyroid gland; it increases blood calcium by acting on the bone, intestine, and kidneys.

**parenteral nutrition (PN):** The delivery of nutrients into the circulatory system.

**pellagra:** A nutritional wasting disease attributable to a combined deficiency of the essential amino acid tryptophan and niacin (nicotinic acid).

**periosteal apposition:** Deposition of bone on the outer surface.

**permissive effect:** Factors that aid in the pathologic effect of another factor or set of antecedents.

**phagocytosis:** The ability of white blood cells to engulf foreign bodies.

**phenylketonuria:** A form of hyperphenylalaninemia caused by the complete or near complete deficiency of the liver enzyme phenylalanine hydroxylase that results in an accumulation of phenylalanine in body fluids and the central nervous system.

**physical activity:** Body movement produced by the contraction of skeletal muscle and that substantially increases energy expenditure. Common categories of physical activity include occupational, household, leisure time, and transportation.

**phytate:** A phosphorus-containing compound found in whole grains and dried beans that binds with minerals, particularly iron and zinc, and interferes with their absorption.

**polycystic ovarian syndrome (PCOS):** A clinical syndrome in women consisting of two of the following three conditions: oligo- or anovulation, hyperandrogenism, and polycystic ovaries.

**Prader-Willi syndrome (PWS):** A genetic condition caused by an absence of material from the 15th chromosome. Characteristics include developmental delays, low motor tone, and an insatiable appetite.

**prematurity:** Less than 37 weeks gestation at birth.

**presbyosmia:** Loss of the sense of smell.

**primary prevention strategies:** Encourage health-enhancing behaviors by giving individuals, families, and communities ways to reduce risk factors associated with disease and injury.

**pro-oxidant:** A compound that normally has the potential to act as a free radical in its present oxidative state.

**psychiatric comorbidity:** Presence of additional mental disorders, for example, substance abuse or personality disorder.

**Quality Assurance Performance Improvement (QAPI) programs:** This is a type of quality assurance program that is mandated in nursing homes by the Affordable Care Act.

**quality indicators:** A quality indicator report is generated from a facility-wide Minimum Data Set to be used by surveyors during the survey process. The quality indicator report is used to help surveyors identify potential quality problems in the facility. The quality indicator report for nutrition/eating includes "prevalence of weight loss," "prevalence of tube feeding," and "prevalence of dehydration."

**quality measures:** Selected Minimum Data Set made available to the public by the Centers for Medicare and Medicaid Services at the Nursing Home Compare website. These data are intended to represent the quality of care provided at nursing facilities so consumers can make an informed decision. Prevalence of weight loss is one of the quality measures from each nursing home that the Centers for Medicare and Medicaid Services shares with the public.

**randomized design:** When individuals are randomly assigned to a treatment or control group.

**recumbent length:** Measuring the length of an individual lying down.

**Resident Assessment Instrument (RAI):** Part of the medical record in skilled nursing facilities and includes the Minimum Data Set, Care Area Assessment process, and the RAI guidelines.

**rickets:** Vitamin D deficiency resulting in growth deficits, developmental delay, failure to thrive, short stature, tetany, seizures, and skeletal deformities.

**risk management:** Identification of areas where harm may occur to an individual, such as in the case of a fall with injury. Taking precautions to limit the number of times this situation may occur is the responsibility of facility management.

**rooting reflex:** Reflex present from birth to age 3 months that assists the infant to locate the breast and nipple by turning the head side to side and opening the mouth wide when the mouth is stroked.

**sarcopenia:** Decrements in muscle mass that result in declining functional ability.

**sarcopenia:** Progressive reduction in muscle mass with aging.

**scurvy:** Classic vitamin C deficiency in which symptoms are often subtle and difficult to diagnose, with early signs being listlessness, weakness, irritability, vague muscle and joint pain, and weight loss.

**sea vegetables:** Wild ocean plants, including nori, kelp, hiijiki, and dulse, that often are purchased in dried form.

**secondary prevention:** Risk appraisal and screening to emphasize early detection and diagnosis of disease.

**senescence:** The process of aging taking into account cellular death and dysregulation leading to functional decline.

**short bowel syndrome (SBS):** A malabsorptive state caused by a significant bowel resection or congenital defect.

**Sjögren syndrome:** Dryness of mucous membranes.

**small for gestational age (SGA):** Birth weight plots below the 10th percentile.

**spastic quadriplegia:** Spastic paralysis of the arms and legs.

**specificity:** The ability of a measure actually to measure the component of interest.

**spina bifida:** A neurologic tube defect caused by a lesion in the spinal cord that occurs during the formation of the spinal cord.

**State Survey:** The process of the state department of health entering a facility to determine whether the Centers for Medicare and Medicaid Services and state requirements are being met.

**substituted judgment:** A legal term used to describe what a person would decide if he or she were able to decide; used when the individual is not competent to provide an autonomous decision.

**sucking reflex:** Strong reflex present in the newborn elicited by stroking the infant's lips, cheeks, or inside the mouth.

**syncope:** Temporary loss of consciousness.

**syndromes:** A term used to identify a developmental disability with a cluster of distinctive features, such as Down syndrome.

**synergistic:** The outcome of two influential factors is greater than the sum of each alone (such as 1 + 1 = 3 times the effect).

**tertiary prevention:** Treatment and rehabilitation; reduction in the amount of disability caused by a disease to achieve the highest level of function.

**therapeutic alliance:** Cooperation between the patient and the therapist to engage in treatment; dependent on good rapport between patient and therapist.

**The Special Supplemental Nutrition Program for Women, Infants, and Children (WIC):** A program that serves to safeguard the health of low-income women, infants, and children up to age 5 years who are at nutritional risk by providing nutritious foods to supplement diets, information on healthy eating, and referrals to health care.

**trace minerals:** Minerals that are needed in small amounts (iron, zinc, copper, selenium, iodine, manganese, molybdenum, chromium, and cobalt).

**tumor necrosis factor-alpha:** An indicator of inflammatory response and a mediator of several cascade mechanisms involved in immune function and homeostasis.

**type 1 diabetes:** Diabetes caused by a lack of endogenous insulin.

**type 2 diabetes:** Diabetes caused by ineffective or low levels of insulin.

**tyrosinemias:** A group of inherited inborn errors of metabolism characterized by disordered tyrosine metabolism.

**urea cycle disorders:** Inborn errors of urea synthesis caused by a deficiency of the enzymes in the urea cycle (carbonyl phosphate synthetase, n-acetylglutamate synthetase, ornithine transcarbamylase, arginosuccinic acid synthase, arginosuccinate lyase, and arginase).

**vegan:** A vegetarian who avoids all animal products, including dairy products and eggs.

**vegetarian:** A person who does not eat meat, fish, or fowl or products containing those foods.

**very low birth weight (VLBW):** Birth weight of less than 1,500 g.

**vitamins:** Organic compounds that occur naturally in plants and animals and are not synthesized by the body in adequate amounts to meet physiologic needs.

**water-soluble vitamins:** Including vitamin C, thiamine, riboflavin, niacin, vitamin $B_6$, biotin, pantothenic acid, folate, and vitamin $B_{12}$; soluble in nonpolar solvents, absorbed by passive and active processes, and excreted in the urine.

**Waterlow criteria:** A set of calculations used to classify the severity of malnutrition in children.

**Wilson disease:** An inherited disorder of copper metabolism characterized by a failure of the liver to excrete copper, leading to its accumulation in the liver, brain, corneas, and kidneys, with resulting chronic degenerative changes.

**xerostomia:** A dryness of the mouth.

**zinc deficiency:** Prevalent in undernourished children and associated with poor growth, poor developmental outcomes, and diarrheal disease.

# Index



*Note:* Page numbers followed by a "t" or a "f" indicate a table or a figure, respectively.

## N