Journal of Nutritional Science

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A diet-wide association study for liver cancer risk: findings from a prospective cohort study in Chinese women

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Research Article

Cite this article: Tuo J-Y, Shen Q-M, Li Z-Y, Tan J-Y, Tan Y-T, Li H-L, and Xiang Y-B (2024). A diet-wide association study for liver cancer risk: findings from a prospective cohort study in Chinese women. *Journal of Nutritional Science* **13**: e95, 1–6. doi: 10.1017/jns.2024.86

Received: 11 February 2024
Accepted: 10 October 2024

Keywords:
Cohort study; Diet; Diet-wide association study; Liver cancer; Nutrition; Women

Abbreviations:
SWHS, Shanghai women's health study; HCC, hepatocellular carcinoma; HBV, Hepatitis B virus; HCV, Hepatitis C virus; T2DM, type 2 diabetes mellitus; IL-6, interleukin-6; HPFS, Health Professionals Follow-up Study; NHS, Nurses' Health Study; DWAS, Diet-wide association study; GWAS, genome-wide association studies; FDR, false discovery rate; FFQ, food frequency questionnaire; HR, hazard ratio; CI, confidence interval; PYs, person-years; BMI, body mass index; MET, metabolic equivalent; IQR, Inter quartile range; EPIC, European Prospective Investigation into Cancer and Nutrition; NIH-AARP, National Institutes of Health-American Association of Retired Persons Diet and Health Study cohort

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Abstract

Although dietary factors have been examined as potential risk factors for liver cancer, the evidence is still inconclusive. Using a diet-wide association analysis, our research evaluated the associations of 126 foods and nutrients on the risk of liver cancer in a Chinese population. We obtained the diet consumption of 72,680 women in the Shanghai Women's Health Study using baseline dietary questionnaires. The association between each food and nutrient and liver cancer risk was quantified by Cox regression model. A false discovery rate of 0.05 was used to determine the foods and nutrients which need to be verified. Totally 256 incident liver cancer cases were identified in 1,267,391 person-years during the follow-up duration. At the statistical significance level ($P \leq 0.05$), higher intakes of cooked wheaten foods, pear, grape and copper were inversely associated with liver cancer risk, while spinach, leafy vegetables, eggplant and carrots showed the positive associations. After considering multiple comparisons, no dietary variable was associated with liver cancer risk. Similar findings were seen in the stratification, secondary and sensitivity analyses. Our findings observed no significant association between dietary factors and liver cancer risk after considering multiple comparisons in Chinese women. More evidence is needed to explore the associations between diet and female liver cancer occurrence.

Introduction

Primary liver cancer ranked as the ninth most common cancer in women worldwide and the sixth main cause of cancer death in 2020.(1) Especially for women in China, liver cancer is more severe with the sixth cancer incidence and the fourth cancer mortality.(2) As the major histological type of liver cancer, hepatocellular carcinoma (HCC) accounts for 75–80% of all liver cancer cases.(3) Hepatitis B or C virus (HBV or HCV) infection and dietary aflatoxin intake are the most important causes of HCC, leading to over 80% cases.(4,5) In addition, the incidence and mortality rates of men were two to three folds of those of women, which is a stark sex discrepancy in most countries.(3) However, the precise cause of the disparities between men and women is still unknown. According to some researchers, men are more likely than women to be exposed to high-risk behaviours such as alcohol abuse, cigarette smoking, and HBV and/or HCV infection.(6) Hormones were also considered as a cause for sex difference in cancer development,(7) which could be related to serum interleukin-6 (IL-6) and adiponectin expression.(8,9)

In recent years, dietary factors have been extensively investigated as possible risk factors for liver cancer, but the evidence for the associations is still inconclusive, especially in men and women respectively. Many studies from the two large prospective cohorts - the Nurses' Health Study (NHS) for women and the Health Professionals Follow-up Study (HPFS) for men in America have been conducted to explore the dietary factors on liver cancer risk.(10–12) For example, processed red meat intake might be associated with a higher risk of HCC (HR = 2.36, 95% CI: 1.18–4.72), and an inverse association was found between poultry and HCC risk (HR = 0.53, 95% CI: 0.28–0.98); while there was a statistically significant association between fish and decreased HCC risk among women (HR = 0.52, 95% CI: 0.30–0.91).(12) The inconsistencies in the diet research among men and women may be related to their different dietary patterns and intake amounts, or various confounding factors related to liver cancer. However, most studies combined the male and female into their analyses, which could bias the results. In addition, many studies only focus on the association between one or several foods and nutrients and liver cancer, and are mainly conducted in America and Europe. Therefore, according to the differences in dietary cultures between China and other countries, and

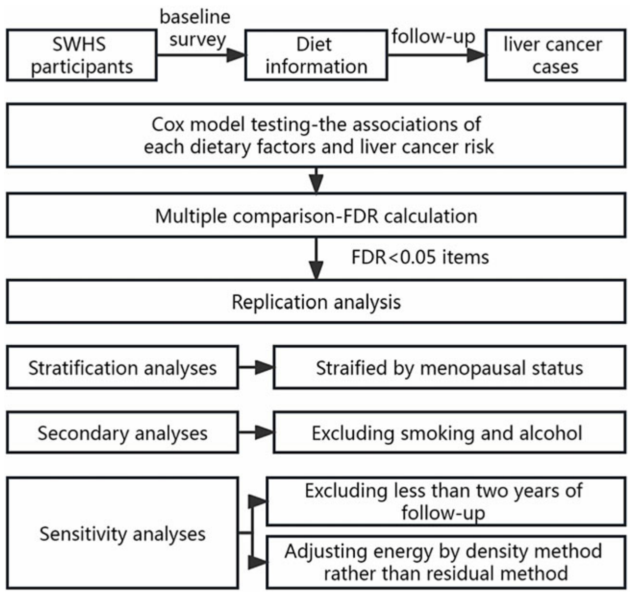

Figure 1. Study process of diet-wide association study analytical method to test associations between foods and nutrients intake and risk of liver cancer in the Shanghai Women's Health Study.

variations of eating habits for different sexes, it is worthy to conduct a high-quality epidemiologic study between diet and nutrition and liver cancer risk in Chinese women.

Similar to genome-wide association studies (GWAS), the diet-wide association study (DWAS) could estimate the association of each food and nutrient on health outcomes and perform multiple comparisons to identify the promising associations for further replication. The DWAS method has been used to find out the impact of diet on breast cancer, colorectal cancer and lung cancer,[13–15] while no studies have been conducted on liver cancer. Our study aims to provide high-quality evidence about the associations of foods and nutrients with the risk of liver cancer in Chinese female adults.

Materials and methods

Our study process of DWAS analytical method to test associations between foods and nutrients intake and risk of liver cancer, using dietary information from the cohort database of the Shanghai Women's Health Study (SWHS) has been presented (Figure 1).

Study population

From December 1996 to May 2000, the SWHS recruited 74,940 participants aged 40–70 and living in urban Shanghai. Previous reports had already revealed the study's design and rationale.[16] We have obtained written informed consent from all the participants. Each participant was questioned for a questionnaire to collect baseline demographic information, diet, lifestyle (such as smoking, alcohol drinking, and tea drinking), menstrual and reproductive condition, personal disease history and family history of cancer. Participants who met the following criteria were not allowed to participate in current study: (1) cancer in situ was discovered in follow-up (n = 135); (2) no cancer type or diagnosis date had been collected when the participant passed away from cancer (n = 244); (3) cancer at baseline (n = 1,598); (4) lost to follow-up after enrolling (n = 3); (5) diagnosis of cancer wasn't confirmed (n = 67); (5) values of total calorie intake were extremely low or high (<500 or >3,500 kcal/d) (n = 121); and (6) data of interested covariates was missing (n = 92). Finally, 72,680 participants were retained in current analysis. Informed consent has been obtained from all participants. This study was carried out in compliance with the Helsinki Declaration and was authorised by the Renji Hospital Ethics Committee of Shanghai Jiao Tong University School of Medicine (KY2019-197).

Assessment of dietary factors

At baseline of all cohort members, the dietary intake over the previous 12 months was collected using a validated semi-quantitative food frequency questionnaire (FFQ) with 71 food items, primarily about the average frequency (daily, weekly, monthly, yearly, or never) and amounts (1 liang = 50 g) per unit of time.[17] The FFQs of SWHS have fairly high validity and reproducibility as compared with multiple 24-h dietary recalls. The correlation coefficient for major food groups was 0.41–0.66 in the SWHS. The 2002 Chinese Food Composition Table [18] was used to calculate daily nutritional requirements based on the nutrient composition of each food. In the analysis, 126 dietary factors (88 foods/food groups and 38 nutrients) were included in total (Supplementary Tables S1 & S2).

Follow-up and case ascertainment

Every participant in the cohort was followed up every 3–4 years.[16] The data was synchronised yearly with the databases of the Shanghai Vital Statistics Registry, and the Shanghai Cancer Registry. In the past two decades, five follow-up surveys were done, with response rates of 99.7% from 2000 to 2002, 98.7% from 2002 to 2004, 94.9% from 2004 to 2006, 92.3% from 2007 to 2010, and 91.1% from 2012 to 2017. Each liver cancer diagnosis was supported by home visits, hospital medical reports, and reviews of medical records by clinical and pathological specialists.[18] The International Classification of Disease, the Ninth Revision was also used to code diseases, and liver cancer was defined as a primary malignant tumour with the code 155.[19] In this study, we censored the follow-up information on 31 December 2016.

Statistical analyses

In order to describe baseline characteristics, the entire cohort was separated into cases and non-cases of liver cancer. Continuous variables were reported as medians with interquartile ranges (IQRs), while categorical variables were described as counts with proportions. The Wilcoxon-Mann-Whitney test was used to compare continuous variables because of the skewed distribution, and the Chi-square test to compare categorical ones.

Cox proportional hazard regression models were used to estimate the association between each of the dietary factor and the risk of liver cancer in the cohort. The follow-up time (years) was chosen as the underlying time metric. And we determined the period from baseline to an event (i.e. liver cancer occurrence) or right-censoring (i.e. death, loss to follow-up, or Dec. 31, 2016), whichever came first, as person-years (PYs) to the event. The Schoenfeld residual method was used to validate the proportional hazards assumptions in Cox regression models,[20] and no indication of a violation of these assumptions was found. Foods and nutrients were adjusted for energy intake using the residual approach[21] and Z was transformed to reflect the associations per one standard deviation (SD) increase in dietary consumption. Hazard ratios (HRs) and 95% confidence intervals (CIs) were presented by both quartile categories and per 1-SD increment of the dietary items. All models were adjusted for age at entry (years, continuous), calorie intake (kcal/d, continuous), body mass index (BMI) (4 categories: <18.5 kg/m^2, 18.5–23.9 kg/m^2, 24–27.9 kg/m^2, ≥28 kg/m^2),[22] physical activity (multiplying the weekly hours spent on specific physical activities (including stairs climbing, housework, walking, cycling to and from work, and walking and cycling except for transportation for work) in the past 1 year by their corresponding metabolic equivalent (MET) values and cumulating the total weekly MET (MET hour/week, continuous), education (4 categories: elementary school and below, middle school, high school, and college and above), menopause status (yes/no), smoking status (defined as 'ever smoked at least 1 cigarette/day for more than 6 months continuously', yes/no), alcohol drinking status (defined as 'ever drank alcohol at least 3 times/week for more than 6 months continuously', yes/no), family history of liver cancer (yes/no), medical history of chronic hepatitis (yes/no) and type 2 diabetes mellitus (T2DM) (yes/no).

We calculated the false discovery rate (FDR) for each food and nutrient to account for multiple comparisons in the analysis, which is the proportion of expected false positives to all positive associations, or the percentage of findings drawn from the null distribution at a specific significance level.[23] To compute the value of FDR, we generated a 'null distribution' of regression test statistics through random assignment of the case status and both the case status and time to event, run the Cox proportional hazards model, and gathered the corresponding P value over 1,000 permutations.[24] Dietary factors with an FDR < 0.05 would be retained for republication.[25]

Priori exploratory stratification analyses were conducted to evaluate the associations of dietary factors on liver cancer risk among participants with different menopausal statuses (yes vs. no). The secondary analyses comprised the participants without smoking and alcohol drinking, in order to eliminate the potential role of smoking and alcohol drinking. In addition, sensitivity analyses were also conducted, such as (1) all participants with a follow-up time of less than two years were excluded; (2) dietary nutrients were adjusted by the energy density method rather than the residual method to examine the robustness of the results.[21]

The results were deemed statistically significant when the two-sided P values were less than 0.05. All of the analyses were conducted using the R software (version 4.0.5).

Data availability statement

The data will be available on request pending approval by the scientific committee of the relevant institutes.

Results

From the baseline survey to the end of 2016, there was a total of 1,267,391 person-years (average of 17.44 years) of follow-up in which 256 female participants were newly diagnosed with liver cancer. The incidence density of liver cancer was 20.20 cases per 100,000 PYs, and the cumulative incidence proportion was 0.35% during the follow-up period. Compared with non-cases, liver cancer cases were more likely to be older, overweight, lower-educated, postmenopausal, and had a family history of liver cancer, self-reported medical histories of chronic hepatitis and T2DM (Table 1). The baseline dietary items intake levels in the cohort have also been presented (Supplementary Table S3).

Of the 126 dietary factors that were evaluated in SWHS, higher intakes of four factors (cooked wheaten foods, pear, grape, copper) were associated with a low risk of liver cancer at the statistical significance level ($P < 0.05$), while the other four factors (spinach, leafy vegetables, eggplant, carrots) were positively associated with liver cancer risk. Cooked wheaten foods and pear also had inverse linear trends with the risk of liver cancer ($P_{trend} < 0.05$). However, no dietary factor retained an association with liver cancer risk after correcting for multiple comparisons (minimum FDR = 0.252) (Figure 2 & Supplementary Table S4).

Exploratory stratification analyses according to menopausal status have been conducted in the recruited women. For the 8 statistically significant dietary variables in the main analyses, only carrots among premenopausal women, and cooked wheaten foods, pear, copper and spinach among postmenopausal women still had the HRs at the statistical significance level ($P < 0.05$). While no associations were observed after correcting for multiple comparisons in both the premenopausal and postmenopausal women (Supplementary Table S5).

In the secondary analyses, after excluding the participants with smoking or alcohol drinking, we observed that all the statistically significant HRs of the 8 factors in the main analysis still existed ($P < 0.05$). After correcting for multiple comparisons, the dietary factors still had no associations with liver cancer risk (minimum FDR = 0.252) (Supplementary Table S6).

As for the sensitivity analyses, we excluded participants with less than 2 years of follow-up, and found that the statistically significant HRs of the factors in main analyses still existed except for carrots, while disappeared after correcting for multiple comparisons (minimum FDR = 0.084) (Supplementary Table S7). The results of dietary nutrients using the energy density method were similar to the energy residual method except for copper (Supplementary Table S8).

Discussion

In this study, we used the DWAS approach to systematically evaluate the association between dietary intakes of 126 foods and nutrients and the risk of liver cancer in Chinese women. At the statistical significance level ($P < 0.05$), cooked wheaten foods, pear, grape and copper were inversely associated with liver cancer risk, while spinach, leafy vegetables, eggplant and carrots showed the positive associations. After considering multiple comparisons, no dietary variable was associated with liver cancer risk (FDR > 0.05). Similar results were observed in the stratification, secondary and sensitivity analyses.

The cooked wheaten foods always include noodles and steamed bread, which are rich in starch. However, the literature evidence for the association between cooked wheaten foods and risk of liver cancer is sparse, and only a few focused on starch and liver cancer risk. Polesel J and his colleagues recruited 185 HCC patients and 412 controls and found that the highest vs. lowest tertile of OR was 1.70 for starch on HCC development ($P_{trend} > 0.05$).[26] In contrast, a prospective study from the European Prospective Investigation into Cancer and Nutrition (EPIC) found that 50g/d increment of total starch intake was associated with a lower risk of HCC (RR = 0.70, 95% CI: 0.55–0.90) among European people.[27] However, these studies didn't figure out the dietary associations among female participants. Our study showed the inverse association between cooked wheaten foods and female liver cancer risk, which disappeared after correcting for multiple comparisons. More

Table 1. Baseline demographic and lifestyle characteristics between liver cancer cases and non-cases (Shanghai Women's Health Study, 1996–2016)

	Overall (n = 72,680)	Non-cases (n = 72,424)	Liver cancer cases (n = 256)	*P* value
Age at entry (years, IQR)	50.27 (16.44)	50.24 (16.42)	60.83 (14.56)	<0.001
BMI (kg/m^2, IQR)	23.73 (4.44)	23.72 (4.43)	24.70 (5.26)	<0.001
Physical activity (MET hours/week, IQR)	100.45 (57.10)	100.45 (57.10)	102.39 (54.90)	0.918
Calorie intake (kcal/d, IQR)	1,634.10 (494.46)	1,634.32 (494.35)	1,583.62 (469.37)	0.071
Education (*n*, %)				<0.001
Elementary school and below	15,489 (21.31)	15,381 (21.24)	108 (42.19)	
Middle school	27,060 (37.23)	26,996 (37.27)	64 (25.00)	
High school	20,315 (27.95)	20,255 (27.97)	60 (23.44)	
College and above	9,816 (13.51)	9,792 (13.52)	24 (9.38)	
Menopausal status (*n*, %)				<0.001
Yes	35,496 (48.84)	35,299 (48.74)	197 (76.95)	
No	37,184 (51.16)	37,125 (51.26)	59 (23.05)	
Smoking status (*n*, %)				0.189
Yes	2,006 (2.76)	1,995 (2.75)	11 (4.30)	
No	70,674 (97.24)	70,429 (97.25)	245 (95.70)	
Alcohol drinking status (*n*, %)				0.602
Yes	1,628 (2.24)	1,624 (2.24)	4 (1.56)	
No	71,052 (97.76)	70,800 (97.76)	252 (98.44)	
Family history of liver cancer (*n*, %)				<0.001
Yes	2,383 (3.28)	2,357 (3.25)	26 (10.16)	
No	70,297 (96.72)	70,067 (96.75)	230 (89.84)	
Medical history of chronic hepatitis (*n*, %)				<0.001
Yes	1,865 (2.57)	1,829 (2.53)	36 (14.06)	
No	70,815 (97.43)	70,595 (97.47)	220 (85.94)	
Medical history of T2DM (*n*, %)				<0.001
Yes	3,122 (4.30)	3,097 (4.28)	25 (9.77)	
No	69,558 (95.70)	69,327 (95.72)	231 (90.23)	

Note: Values were medians (IQR) for continuous variables and count (proportion) for categorical variables. Compare continuous variables using the Wilcoxon-Mann-Whitney test because of the skewed distribution, and compare categorical ones using χ2 test.
Abbreviations: IQR, inter quartile range; BMI, body mass index; MET, metabolic equivalent; SWHS, Shanghai Women's Health Study; T2DM, type 2 diabetes mellitus.

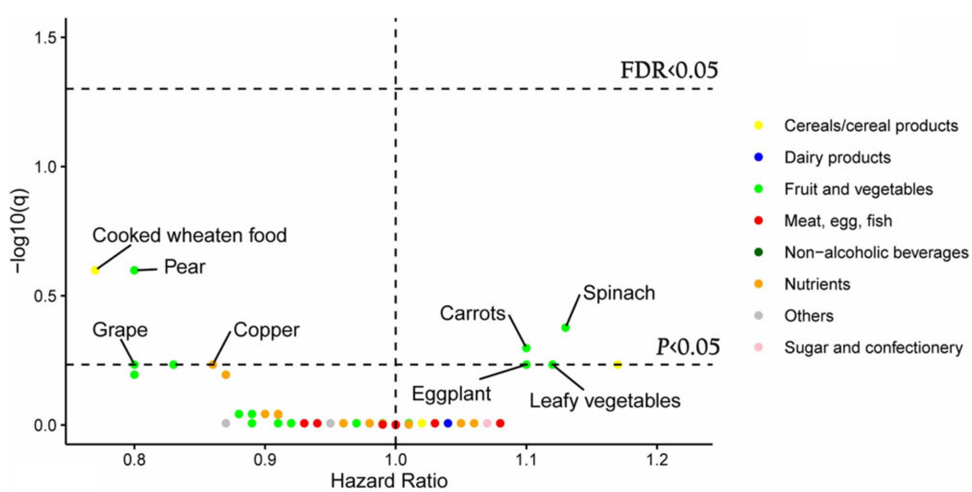

Figure 2. Volcano plot showing results from the diet-wide association study regarding the association between 126 dietary factors and liver cancer risk in the Shanghai Women's Health Study. The y-axis shows the false discovery rate (FDR) adjusted *P* values in –log10 scale from the Cox proportional hazards regression models for each dietary factor. The x-axis shows the estimated HR for each dietary factor per 1-SD increase in daily consumption. The dashed horizontal line represents the level of significance corresponding to FDR of 0.05 and *P* value of 0.05. The models were adjusted for age, BMI, calorie intake, physical activity, education, menopause status, smoking, alcohol drinking, family history of liver cancer, medical history of chronic hepatitis and type 2 diabetes mellitus.

epidemiologic studies are needed to evaluate the potential role of cooked wheaten foods in female liver cancer occurrence.

In general, vegetables and fruits are low in fat and calories, and rich in vitamins, minerals, and dietary fibre, which have been found to be inversely associated with cardiovascular disease, diabetes, and stroke.[28–30] However, a recent meta-analysis including 12 studies reported that vegetables were associated with low risk of liver cancer (Summary RR = 0.70, 95% CI: 0.56–0.87; $I^2 = 79.1\%$), while fruits had no association with liver cancer risk (Summary RR = 0.93, 95% CI: 0.80–1.09; $I^2 = 46.8\%$).[31] The inverse associations between fruits and liver cancer risk could be observed in some case-control studies.[32,33] In our study, we found the positive associations between vegetables (spinach, leafy vegetables, eggplant and carrots) and female liver cancer risk, and fruits (pear and grape) had the statistically significant HRs on decreasing the risk. However, no factors retained the associations after correcting for multiple comparisons. Longer follow-up times and more cases of liver cancer are warranted to affirm the possible effect of vegetables and fruits on the development of female liver cancer.

Copper is an essential micronutrient for human life, and shellfish and animal liver are good food sources for dietary copper.[34] Many reviews indicated that copper has a redox property, and could protect the basic elements of the cell, including lipids, protein, and DNA, from in situ produced reactive oxygen species and oxygen free radicals, which could be useful for people's physical health.[35] However, few studies observed the associations between dietary copper and liver cancer risk. Our study found an inverse association between consumption of dietary copper and liver cancer risk while disappeared after correcting for multiple comparisons. The association of copper on liver cancer is still required more reliable epidemiological evidence.

In stratification analyses, only carrots among premenopausal women, and cooked wheaten foods, pear, copper and spinach among postmenopausal women still had the HRs at the statistical significance level, while all disappeared after correction. The reason might be related to female sex hormones. Premenopausal women have higher levels of oestrogen, and animal studies have found that oestrogen could combine with oestrogen receptor α and suppress the production of IL-6 to decrease liver carcinogenesis.[8] And compared with postmenopausal women, the potential role of diet on liver cancer could be reduced among premenopausal women. More studies are needed to verify the condition.

Most cohort studies like the EPIC and the National Institutes of Health-American Association of Retired Persons Diet and Health Study cohort (NIH-AARP) evaluated dietary factors on liver cancer risk among all the participants rather than only women. Fortunately, the NHS recruited 138,483 women in America and showed that white meat, fish, n-3 PUFA were all associated with the low risk of female liver cancer,[11,12] which were not observed in our study. Except for the sample size of cases, the reason could be attributed to the different dietary patterns between the Chinese and American people - in the NHS, white meat, plant, olive oil and nuts were the main food sources of fat[11]; whereas in the participants of the SWHS, the main food source of fat were red meat and meat products, and people always considered cooked wheaten foods and rice as their staple food.[36]

Our study is the first to systematically estimate the association between a wide set of dietary factors with female liver cancer risk in Asia. The cohort additionally had a large scale of population with liver cancer cases. Moreover, a number of known risk variables for liver cancer from our earlier research have been included into our multivariable analysis, including BMI, physical activity, education, menopause status, smoking status, alcohol drinking status, family history of liver cancer, medical history of chronic hepatitis and T2DM, to reduce the effect of potential confounders. However, there are still several limitations. Firstly, the data on diet were only obtained from the baseline, and the changes over time are needed to take into account. Secondly, the items in the multivariable model weren't adjusted for the other relative dietary components, which still needs careful conclusion. Thirdly, the data of HBV or HCV infection was lacked in the participants, which could limits the interpretation and understanding of the associations and even pathogenesis of dietary factors on liver cancer. Although, the medical history of hepatitis or chronic liver diseases was used to adjust in the multivariable models instead which could reduce the potential effect of HBV or HCV infection. We still need to collect the complete information of HBV or HCV infection for further analysis. Fourthly, diet aflatoxin exposure, the significant cause of liver cancer, wasn't considered in our investigation. Moreover, the number of cases of several reproductive factors were small in our study, so the findings need to be interpreted with caution. And because our prospective cohort primarily consisted of urban Chinese adult women, it should be cautious to generalise the findings to a larger population, such as those living in rural areas. In addition, external verification and high-quality epidemiologic studies are needed to assess the reliability and stability of our findings. Finally, we lacked pertinent information regarding specific cases of hepatocellular carcinoma and other sites, and around 25% of primary liver cancer patients had unclear sites,[37] so we used the combined cases in our analysis. For additional investigation, the specific histological subtype of liver cancer would be needed.

Conclusions

In summary, no significant association was observed between dietary factors and female liver cancer risk after considering multiple comparisons. The possible associations of cooked wheaten foods, pear, grape, copper, spinach, leafy vegetables, eggplant and carrots on female liver cancer before correction warrant further investigations based on longer follow-up times, large number of cases and more high-quality research.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/jns.2024.86

Acknowledgements. We thank all participants and staff from the Shanghai Women's Health Studies for their contribution to this research.

Authorship. Y-BX designed research and obtained funding; J-YT, Q-MS, Z-YL, J-YT, Y-TT, H-LL, and Y-BX conducted the study; J-YT, H-LL, and Y-BX analysed the data and interpreted the results; J-YT and Y-BX prepared and wrote the first draft; All authors reviewed and approved the final version of the paper; and Y-BX has primary responsibility for final content.

Financial support. This work was supported by a grant from the National Key Project of Research and Development Program of China [2021YFC2500404 & 2021YFC2500405]; and the parent cohort was supported by a subcontract from grant of the US National Institutes of Health [UM1 CA182910]. All funders had no role in the design, analysis and writing of this article.

Competing of interests. The authors have declared no potential conflicts of interest.

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Journal of Nutritional Science

cambridge.org/jns

Research Article

Cite this article: Bennett G and Gibney ER (2024). An investigation of diet quality across racial groups in the United Kingdom and United States considering nutritional adequacy, disease risk, and environmental sustainability: a secondary analysis of NDNS and NHANES datasets. *Journal of Nutritional Science* **13**: e93, 1–15. doi: 10.1017/jns.2024.64

Received: 12 January 2024
Revised: 1 August 2024
Accepted: 30 August 2024

Keywords:
Diet quality; Population health; Racial groups; Sustainability

Abbreviations:
AHEI-2010, Alternative Healthy Eating Index 2010; DQI-I, Diet Quality Index International; HEI, Healthy Eating Index; NDNS, National Diet and Nutrition Survey; NHANES, National Health and Nutrition Examination Survey; SSB, Sugar sweetened beverages; U.K., United Kingdom; U.S., United States; USDA, United States Department of Agriculture

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An investigation of diet quality across racial groups in the United Kingdom and United States considering nutritional adequacy, disease risk, and environmental sustainability: a secondary analysis of NDNS and NHANES datasets

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Abstract

Diet indices are quantitative assessments of the quality of population intake. Understanding diet quality is crucial to support health and well-being; however, knowledge of diet quality across racial groups is limited. To examine diet quality of acial groups 'White', 'Black', 'Asian', and 'Other' in the United Kingdom (U.K.) and United States (U.S.), U.K. and U.S. national survey data were used to calculate Alternative Healthy Eating Index (AHEI-2010), Diet Quality Index-International (DQI-I), and EAT-Lancet scores. ANCOVA tests compared median total quality scores across racial groups adjusting for covariates. Kruskal–Wallis tests examined differences in individual component scores. Spearman correlations identified association of diet quality scores across indices. Highest diet quality scores were reported for U.K. and U.S. Asian groups. Most noticeable differences were apparent between U.S. Asian and White/Black groups (62% Asians within highest tertile of AHEI-2010 score vs. 29% Whites; $P < 0.001$). All racial groups demonstrated poor diet quality in terms of sustainability; EAT-Lancet scores were <40% of maximum total score for U.S. White, Black, and Other groups. AHEI-2010 diet quality scores were moderately associated with EAT-Lancet scores, evident across all groups ($r = 0.53–0.65$; $P < 0.001$). There is a need for all groups to increase intake of wholegrains, especially Black groups (mean Wholegrain score for U.S. Black group within DQI-I was 0.60 (maximum score of 5)) as demonstrated within AHEI-2010, DQI-I, and EAT-Lancet component scores. Additionally, increased intake of vegetables and legumes and decreased intake of processed and red meat would improve the adequacy, healthiness, and sustainability of U.K. and U.S. racial diets.

Introduction

Surveillance of dietary intake is essential to understand current consumption trends and indicate aspects of the diet which require more targeted guidance or intervention to maximise population nutrition and health.[1–3] The diet can be assessed by examining individual food group or nutrient intake, or by considering the totality of intake, implementing diet quality indices to assess adequate consumption of dietary components critical to health and well-being.[1,4,5]

A consensus for what constitutes total diet quality is lacking and while it is deemed to be multifaceted, as far as the authors are aware, little research to date has assessed population diet quality considering multiple aspects collectively.[6–8] Globally numerous diet quality indices have been developed to assess public adherence to a specific aspect of healthy eating recommendations, such as adequate nutrient intake, prevention of disease or sustainability of intake.[2,9,10] The Alternative Healthy Eating Index (AHEI-2010), Diet Quality Index-International (DQI-I), and the EAT-Lancet Index are examples of dietary indices developed to examine the quality of these aspects of habitual diet individually (disease risk, adequacy, and sustainability, respectively).[11–13] While each index has been developed for a specific purpose, there is scope to assess population intake using these indices in combination to identify total diet quality under the terms aforementioned. Detailing how such elements of diet quality rate and compare will inform how future dietary guidelines can support improved population consumption, encapsulating both adequate nutrition and optimum planetary health.

In addition to investigating diet quality in terms of the total diet, diet quality of the total population must also be understood. While many studies examine quality across age, sex or social groups, to date research has largely failed to analyse across racial or ethnic groups specifically.[14–16] A recent systematic review identified just six studies globally that examined diet quality across racial/ethnic groups in one country (namely the United States (U.S.), The Netherlands, and Australia).[14] In 2006, Nicolaou *et al.*, reported significantly poorer diet quality among Dutch respondents when compared to Surinamese ethnic groups, results supported by Yau *et al.*, in 2020.[17,18] As well as identifying differences in diet quality across racial and ethnic groups this review also highlighted that many racial and ethnic groups are neglected in nutrition research, even within literature claiming to be *'racially diverse'* (the U.S. studies identified only considered White and Black groups).[14,19–21] As the developed world becomes increasingly diverse, it is essential current diet quality of racial groups within the population is identified to determine where targeted improvements to cultural diets are needed, informing public health policy inclusive of diverse groups within one locality.

To provide a comprehensive insight into total diet quality of diverse populations, the aim of this research was to determine multidimensional aspects of diet quality across racial groups within a country, considering if diet quality was comparable to other racial groups within that country and to the same racial groups living elsewhere. Two of the most diverse countries globally include the U.S. and the United Kingdom (U.K.), and as such, this paper utilised existing national survey data from the U.K. (the National Diet and Nutrition Survey (NDNS)) and the U.S. (National Health and Nutrition Examination Survey (NHANES)) to determine diet quality across racial groups.[22,23] Although distinct aspects of the diet will be assessed independently using DQI-I, AHEI-I, and the EAT-Lancet index, the association between diet quality scores across indices will be examined, indicating whether a sustainable diet also adheres to dietary recommendations and is preventative of disease. Outcomes from this paper will identify intake of specific dietary components, that if changed, may improve the adequacy, healthiness, and sustainability of current diets across U.K. and U.S. racial groups.

Methods

Data collection

National diet and nutrition survey (U.K.)

Demographic and dietary intake data of adult participants (aged 18 years and above) from NDNS years 9–11 (2016–2019) were included in this secondary analysis.[22] Within the NDNS study, demographic data were collected using structured questionnaires and 4-day food diaries were used to record dietary intake.[22] Only participants who completed all four food diaries were included in this analysis. As part of the NDNS demographic questionnaire, participants classified their race as 'White', 'Black', 'Asian', and 'Other, including mixed race'. The NDNS dataset provides intake of individual food and drink items and aggregates this data into food group intakes; these existing aggregated food groups were used to calculate food group components of the three diet quality indices (AHEI-2010, DQI-I, and EAT-Lancet). More detailed information about the NDNS can be found on the Public Health England website.[22] All NDNS data included in this secondary analysis were freely available and downloaded directly from the U.K. data service website.[22]

National health and nutrition examination survey (U.S.)

Demographic and dietary intake data of adult participants (aged 18 years and above) from NHANES 2017–2018 were also included in this secondary analysis. Structured interviews were conducted to collect demographic data and dietary intake was assessed using 2-day 24-hour dietary recalls following the United States Department of Agriculture (USDA) Automated Multiple-Pass Method.[23,24] Only participants who recorded 24-hour dietary recalls on both days were included in this analysis. During interviews participants classified themselves as 'White Non-Hispanic', 'Black Non-Hispanic', 'Asian Non-Hispanic', 'Mexican American', and 'Other Hispanic' and 'Other including multi-racial'. In this secondary analysis, the groups 'Mexican American', 'Other Hispanic', and 'Other including multi-racial' were merged into a single racial group 'Other' to facilitate cross-country comparisons with the NDNS data.[23] The USDA has converted intake of individual food and drink items reported in NHANES into food pattern components (Food Patterns Equivalent Intakes Database), and this database was used to calculate scores of food group components of each diet quality index.[25] All data used in this secondary analysis were open access, and more information on NHANES is available on the National Center for Health Statistics website.[23]

This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects/patients were approved by the National Health Service Health Research Authority Research Ethics Committee East of England Cambridgeshire South; approval number 13/EE/0016 (NDNS data) and the National Center for Health Statistics Research Ethics Committee; approval numbers #2011-17 and #2018-01 (NHANES data). Written informed consent was obtained from all subjects/patients.[23,26] Ethical approval for this secondary analysis of NDNS and NHANES data was granted by the UCD Research Ethics Committee in May 2023 (S-LRSD-23-98-Bennett-Gibney).

Selection of appropriate diet quality indices

Alternate healthy eating index-2010

The AHEI-2010 was developed as a proxy measure of disease risk attributable to habitual diet examining intake of food groups and nutrients particularly important to chronic disease aetiology.[27–29] Although food-based dietary guidelines are designed to protect health, previous work has demonstrated associations between AHEI-2010 score and cardiovascular disease or cancer mortality, validating its use over other indices to assess diet-related disease risk.[12,29] Inequalities in rates of modifiable diseases including cardiovascular disease are prevalent across population subgroups, where minority racial and ethnic groups report higher disease incidence and mortality.[30–33] While changes to habitual dietary intake would likely mitigate these inequalities, cohort-specific dietary challenges need to be identified, and so the AHEI-2010 was implemented to assess dietary related disease risk among racial groups in the U.K. and U.S.

Diet quality index-international

To determine adherence to U.K. and U.S. dietary guidelines, which differ in their recommendations and targets, the Diet Quality Index-International was implemented. The DQI-I was developed to facilitate cross-country comparisons when assessing population adherence to dietary guidelines and nutritional adequacy of population intake.[11] Different from many existing dietary indices, the DQI-I is flexible in terms of the conditions for component minimum-maximum scores allowing these scores to be based on country-specific guidelines, providing a cross-country comparison of adherence to relevant population food-based dietary guidelines and a standardised quantitative scoring approach (i.e. using the one diet quality index for U.K. and U.S. racial groups).

EAT-Lancet index

Assessment of dietary sustainability is critical to understand how food habits could be altered to mitigate the impact of food production, consumption, and waste on the environment.[34–37] However not only should sustainable diets be *'low in environmental impact . . . contributing to food and nutrition security and to healthy life for present and future generations'*, they need also be culturally acceptable, affordable, and accessible to all within the population.[37] To safely improve the sustainability of food consumption, ensuring micronutrient sufficiency is not jeopardised with the transition to plant-rich diets, careful consideration across population subgroups is required.[38] As such, the EAT-Lancet index created by Stubbendorf *et al.*,2022, which measures adherence to optimum sustainable diets based on key food components of EAT-Lancet recommendations, was applied to identify environmental sustainability of intake across U.K. and U.S. racial groups.[13]

Calculating diet quality scores

Diet quality scores were considered in terms of disease risk (AHEI-2010), dietary adequacy (DQI-I) and environmental sustainability (EAT-Lancet). AHEI-2010, DQI, and EAT-Lancet scores were calculated for U.K. and U.S. cohorts separately. Based on original scoring criteria of AHEI-2010 and DQI-I, which accounted for different nutritional requirements by sex, AHEI-2010 and DQI-I component scores for the U.K. and U.S. were calculated for males and females separately, before combining male and female specific scores to calculate total population scores. EAT-Lancet did not provide sex-specific scoring criteria, and so EAT-Lancet scores were calculated for male and female participants jointly. A gradual scoring approach was applied to each component of the AHEI-2010, DQI-I, and EAT-Lancet index, to apply suitable scores to a tighter range of intakes; the scoring approach implemented for each diet quality index is available in Supplementary Table 1a–c. For example, when calculating AHEI-2010 component score, participants who consumed 4 or more servings of fruit daily (100% of recommendation) achieved the maximum score of 10, those who consumed 2 servings of fruit daily (50% of recommendation) achieved a score of 5, and those that had no servings of fruit scored 0; rather than participants receiving a score of 10 for meeting the recommendation and a score of 0 for consuming under 4 servings. For the purpose of this analysis, minor amendments to original AHEI-2010, DQI-I, and EAT-Lancet indices were made due to the availability of specific variables in NDNS and NHANES datasets, more detail of these amendments are described below and in Supplementary Table 2.

Alternate healthy eating index-2010

The original AHEI-2010 consists of 11 components (Fruit, Vegetables, Wholegrains, Sugar Sweetened Beverages & Fruit Juices, Nuts & Legumes, Red & Processed Meat, Trans-Fat, DHA & EPA, Polyunsaturated Fatty Acids, Alcohol, Sodium), all equally weighted at 10 points, meaning that total AHEI-2010 is scored out of 110 (favourable dietary pattern equal to higher AHEI-2010 score).[12,39] However, due to the data variables available in NDNS and NHANES datasets some amendments were made to original AHEI-2010 scoring. These amendments are described in detail in Supplementary Table 2. Briefly, the variable 'trans-fat' (one of the 11 components of original AHEI-2010 scoring) was not available in NHANES and so this variable was not included, meaning that the AHEI-2010 scores in this analysis are out of a total of 100 (10 components scored at 10 points each). This approach has been adopted by others using NHANES data to calculate AHEI-2010 scores.[28] When calculating AHEI-2010 scores for NDNS participants 'trans-fat' was also not used to ensure that AHEI-2010 scores for U.K. and U.S. populations were based on the same number of components. Additionally, within the NDNS cohort, some estimations were made to determine AHEI-2010 component scores of 'DHA & EPA', 'wholegrain', and 'alcohol' as this data was not readily available from the NDNS dataset (see Supplementary Table 2 for full explanation).

Diet quality index-international

The DQI-I is categorised into four sections; variety of food groups and protein sources (scored 0–20); adequacy of fruit, vegetables, wholegrains, fibre, protein, vitamin C, iron, and calcium intake based on national recommendations (scored 0–40); moderation of empty calorie foods, total fat, saturated fat, and cholesterol (scored 0–30); and balance of macronutrient and fatty acid intake (scored 0–10), equalling a total DQI-I score of 100, with a higher score indicating higher diet quality.

Interval cut-offs as previously described by Kim *et al.*, 2003 were applied to each component of the DQI-I for both NDNS and NHANES data.[11] Within the DQI-I, adequacy of food group and nutrient intakes are based on country-specific guidelines and so *'Government Dietary Recommendations'* for U.K. adults aged 19 years and above, published by Public Health England, were applied to NDNS data and USDA *'Dietary Guidelines for Americans 2020–2025'* recommendations for adults aged 19–51 years were applied to NHANES data.[40,41] Iron was the only nutrient to be calculated separately by age for both NDNS and NHANES data (already calculated separately by sex), allowing for age-specific iron recommendations for pre- and postmenstrual women. Intake of certain DQI-I components needed to be estimated as they did not explicitly exist in NDNS/NHANES datasets, these included 'empty calorie foods' (estimated for both NDNS and NHANES cohorts) and 'wholegrain' (estimated for NDNS cohort only). A detailed explanation into how this was done for each variable is available in Supplementary Table 2.

EAT-Lancet index

The EAT-Lancet index developed by Stubbendorf *et al.*, 2022 measures adherence of 14 dietary components to EAT-Lancet recommendations (Fruit, Vegetables, Wholegrains, Unsaturated oils, Legumes, Nuts, Fish, Beef and lamb, Pork, Poultry, Eggs, Dairy, Potatoes, and Added sugar), which are key for environmentally sustainable dietary intake.[13,36] The EAT-Lancet index is scored 0–42, where all 14 components are equally weighted: a score of 3 (i.e. maximum score) indicates optimal adherence to EAT-Lancet recommendations.[13,36] In this paper, all components were calculated as Stubbendorf *et al.*, 2022 describe except for the component 'Beef and lamb' where intake of all red meat including processed red meat were included in this component for both NDNS and NHANES datasets. In the NHANES dataset, all 14 components listed in the EAT-Lancet index were available, however food intake was reported as servings or ounces per day in the Food Patterns Equivalent Intakes database and so these were transformed into grams per day when calculating EAT-Lancet scores for NHANES participants.[25] For NDNS data, intake of wholegrain, added sugar, and unsaturated oils was estimated to determine scores of 'wholegrain', 'added sugar', and 'unsaturated oils' components (see Supplementary Table 2 for more details).

Analysing diet quality scores

AHEI-2010, DQI-I, and EAT-Lancet index scores were calculated for NDNS and NHANES cohorts on an individual level (as per approaches described above), and scores were then analysed using SPSS version 27 at a total population and racial group level by country.

Kruskal Wallis tests were performed to compare demographic characteristics across diet quality tertiles. To determine overall diet quality across U.K. and U.S. racial groups in terms of dietary adequacy; healthiness; and environmental sustainability, median total scores and interquartile ranges of AHEI-2010, DQI-I, and EAT-Lancet were calculated. Analysis of covariance (ANCOVA) examined the differences of total diet quality scores across racial groups (both within and across countries), controlling for covariates sex (NDNS)/gender (NHANES), age, education, and income levels, applying Bonferroni correction method. These covariates are widely considered in nutritional research as being influential to dietary behaviour and food intake and in this case allow examination of association between diet and race while controlling for the impact of these confounding factors.[42–44] To understand what aspects of dietary intake account for variances in diet quality index scores across racial groups, scores of individual index component scores are presented across racial groups to permit more detailed investigations of dietary habits. Spearman correlations were performed to identify the relationship of scores across diet quality indices, determining if there is potential synergy among the aspects of diet quality considered. Statistical significance was considered at a 95% confidence interval.

Results

Participant demographics

An examination of demographics and socio-economic status across racial groups within the U.K. (NDNS dataset) and the U.S. (NHANES dataset) is available in Table 1. A total of n = 1780 U.K. and n = 4339 U.S. participants were included in this secondary analysis of diet quality: 90% of NDNS participants reported to be White while the NHANES cohort was more evenly distributed across racial groups (White = 36%, Black = 25%, Asian = 12%, Other = 27%, Table 1). In the U.K., the racial group Other consisted of *'Mixed'*; n = 12 and *'Any other group'*; n = 19. In the U.S., the racial group Other consisted of *'Mexican American'*; n = 558, *'Other Hispanic'*; n = 389 as racial categorisation of Hispanic/Latino participants were not asked in NHANES, and *'Other Race – including multi-racial'*; n = 222. An even distribution across sex/gender groups was reported for all racial groups in both the U.K. and U.S. except for U.K. Black, where participants were mostly female (78%). In both countries, a BMI of ≥30 kg/m^2 was most prevalent among White and Black groups and the U.S. Asian group had the highest proportion of respondents with a BMI between 18.5 and 24.9 kg/m^2 (45% *vs.* ≤25% among non-Asian U.S. racial groups). A high percentage of non-White U.K. racial groups were educated to university level (47–60% *vs.* 32% of U.K. White) and there was an even proportion of U.K. racial groups across income levels. In the U.S., one third of the racial group Other reported having no qualification with <13% having a university degree,

Table 1. Participant demographics of NDNS and NHANES cohorts (U.K. vs. U.S.)

Variable		United Kingdom (NDNS, n = 1780)				United States (NHANES, n = 4339)			
		White (n = 1614)	*Black (n = 36)*	*Asian (n = 96)*	*Other (n = 31)*	*White (n = 1570)*	*Black (n = 1070)*	*Asian (n = 530)*	*Other (n = 1169)*
Age (yrs)	Mean (SD)	49.8 (17.9)	41.7 (13.6)	39.9 (13.5)	38.5 (13.0)	53.2 (19.5)	49.8 (17.4)	46.4 (16.4)	46.9 (30.4)
Sex; n (%)	Female	945 (58.6)	28 (77.8)	50 (52.1)	20 (64.5)	806 (51.3)	568 (53.1)	278 (52.5)	615 (52.6)
BMI; n (%)	18.5–24.9	555 (34.4)	10 (27.8)	32 (33.3)	13 (41.9)	394 (25.6)	222 (20.7)	236 (44.5)	204 (17.5)
	25.0–29.9	584 (36.2)	7 (19.4)	46 (47.9)	12 (38.7)	446 (28.4)	265 (24.8)	196 (37.0)	401 (34.3)
	≥30	429 (26.6)	13 (36.1)	13 (13.5)	6 (19.4)	685 (44.5)	544 (50.8)	83 (15.7)	529 (45.3)
Education; n (%)	No Qual*	632 (39.2)	14 (38.9)	35 (36.5)	6 (19.3)	179 (11.4)	151 (14.2)	46 (8.7)	389 (33.3)
	High School	367 (22.8)	5 (13.9)	11 (11.5)	7 (22.6)	1026 (65.4)	708 (65.7)	158 (29.8)	628 (53.8)
	Bachelors+	591 (31.9)	17 (47.3)	46 (48.0)	18 (60.2)	365 (23.2)	209 (19.5)	326 (61.5)	148 (12.7)
Income Level; n (%)	Low^	415 (25.7)	10 (27.8)	31 (32.3)	8 (25.8)	262 (17.4)	217 (20.3)	34 (6.4)	221 (18.9)
	Middle^	483 (29.9)	12 (33.3)	23 (24.0)	4 (12.9)	669 (44.4)	414 (38.7)	142 (26.8)	488 (41.7)
	High^	510 (31.6)	8 (22.2)	20 (20.8)	14 (45.2)	575 (38.2)	307 (28.7)	315 (59.4)	358 (30.6)

U.K., United Kingdom; U.S., United States of America; U.K. 'Other' group includes mixed race, U.S. 'Other" group includes Hispanic, Mexican American and multiracial groups, age measured in years, SD, standard deviation, BMI categories based on WHO classification; the proportion of participants with a BMI of <18.5 were not reported in this demographics table, No Qual* = no qualification, + = Bachelors degree and above, ^ = NDNS categorised income into three tertiles, NHANES reported incomes were categorised into these tertiles based on distribution (low <$20,000, middle =$20,000–$64,999, high = $65,000+).

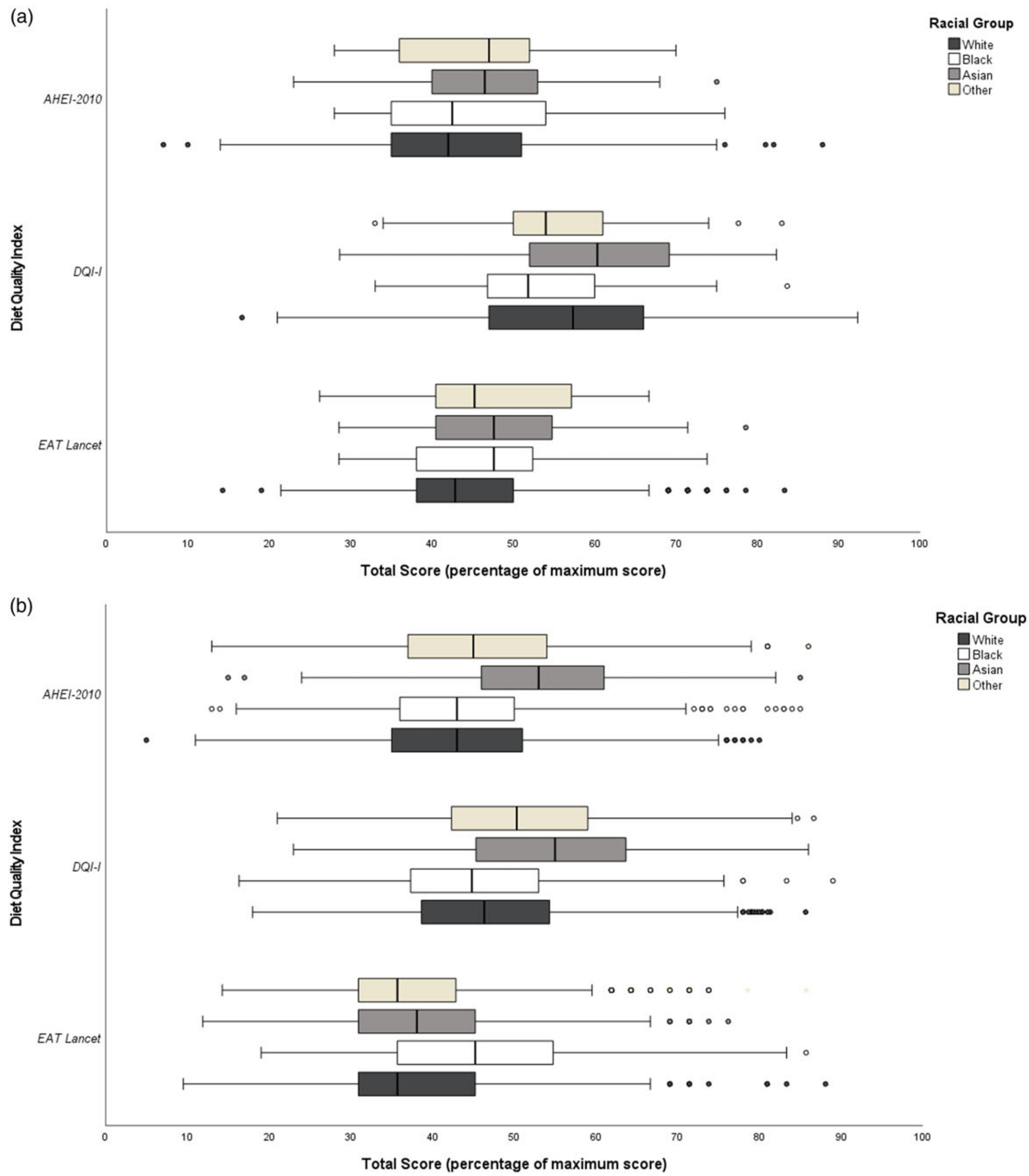

Fig. 1. Median diet quality scores (as percentage of maximum score) and ANCOVA across U.K. racial groups. AHEI-2010 = Alternative Healthy Eating Index (2010), DQI-I = Diet Quality Index-International, * = P < 0.05, ** = P < 0.001. (a) adjusted for age, sex, income, and education with Bonferroni pairwise comparisons corrected P value shown. (b) Only adjusted for age, sex, income, and education with Bonferroni pairwise comparisons corrected P value shown.

compared to 62% of the Asian group reporting the highest education level. The U.S. Asian group had the highest proportion (59%) of respondents with a household income of >$65,000 (highest income bracket). Demographic characteristics did differ across tertiles of diet quality scores including age, sex, education and income. In both U.K. and U.S. populations, tertile of AHEI-2010, DQI-I and EAT-Lancet scores differed significantly across racial groups ($P \leq 0.015$); U.K. and U.S. Asian groups had the highest proportion of respondents whose scores fell within the highest tertile of AHEI-2010 and EAT-Lancet score (Supplementary Tables 3a–c).

Diet quality across racial groups (within countries)

Median total scores of AHEI-2010, DQI-I, and EAT Lancet indices of U.K. and U.S. racial groups are illustrated in Fig. 1a and b respectively, presented as a percentage of the maximum total score of each index due to differing index scoring. In all indices a higher percentage indicates better adherence to recommendations and higher diet quality.

Among U.K. racial groups, median AHEI-2010 scores (as a percentage of maximum total) ranged from 42 to 47%, where White AHEI-2010 scores were significantly lower than Asian AHEI-2010 (median scores of 42 and 47 respectively), $P < 0.001$

https://doi.org/10.1017/jns.2024.64 Published online by Cambridge University Press

once adjusted for demographics and Bonferroni pairwise comparisons (Fig. 1a). Significantly lower EAT-Lancet median scores were found for the U.K. White group compared to the U.K. Asian group (43% *vs.* 48% of maximum total score). The U.K. White group had higher DQI-I median scores than Black and Other cohorts, with significant differences observed across White and Black groups in the fully adjusted model ($P < 0.001$). Asian groups achieved the highest median total scores for the AHEI-2010, DQI-I, and EAT-Lancet indices, however scores were not significantly higher than Black or Other cohorts in fully adjusted models. In the U.S., median total index scores were highest amongst the Asian cohort who achieved 53%, 55% and 45% of maximum total scores for AHEI-2010, DQI-I and EAT Lancet index respectively (Fig. 1b). Larger differences across racial groups were observed in the U.S. than the U.K. U.S. Asian and Other groups achieved significantly higher AHEI-2010, DQI-I, and EAT-Lancet index total scores ($P < 0.001$ once adjusted for demographics and Bonferroni pairwise comparisons) than White and Black groups, while the Other group achieved significantly lower median total AHEI-2010, DQI-I, and EAT-Lancet index scores than the Asian group in the U.S.

Diet quality across racial groups (across countries)

Median total diet quality scores obtained by racial groups in the U.K. were compared to the those of the same racial group in the U.S. (Fig. 2a–c).

Similar trends were noted across countries, where Asian participants from the U.K. and U.S. had highest median total index scores across all indices examined. When racial groups were considered across location individually, diet quality of U.S. racial groups, as estimated by DQI-I and EAT-Lancet, was lower than the same racial group in the U.K. The U.K. White group reported significantly higher median total scores for dietary adequacy (57 vs. 47; Fig. 2b) and sustainability (19 vs. 16; Fig. 2c) than their U.S. counterparts. U.K. Black and Other groups also achieved significantly higher median EAT-Lancet scores than their U.S. counterparts (Fig. 2c). U.K. Black median DQI-I total scores were also significantly higher than U.S. Black DQI-I scores (51 vs. 45; $P < 0.001$). Significant differences across U.K. and U.S. Asian median diet quality scores were noted for DQI-I (60 vs. 55; $P < 0.001$). Diet quality as calculated by AHEI-2010 was higher in U.S. White, Black and Asian racial groups than their U.K. counterparts (Fig. 2a).

Index component scores

Many dietary components of each index (AHEI-2010, DQI-I, and EAT-Lancet; Tables 2a–2c respectively) followed the same trend across racial groups, with highest individual component scores often achieved by Asian groups, while lowest scores were most often reported within White and Black groups. Larger significant differences were reported across U.S. racial groups than U.K. groups. Despite significant differences in component scores across racial groups, low component scores of similar food groups were consistently observed across racial group and diet quality index, namely wholegrain, fruit, vegetable (U.K. only), and a variety of protein sources. Components which largely differed across racial groups are outlined in greater detail for each diet quality index below.

AHEI-2010

In the U.K. only the components ‘Red and Processed Meat’ and ‘Polyunsaturated fat as total energy’ differed significantly across groups and scores were lowest in White (6.92/10) and Asian (5.00/10) respectively (Table 2a). Among the U.S. cohort, significant differences were observed across all AHEI-2010 components except ‘Alcohol’ and ‘Sodium’. Asian mean scores were highest for all remaining components except for ‘Polyunsaturated fat as total energy’ which was highest amongst the Black group, as found within the U.K. dataset (U.S. Black mean score = 8.39; $P < 0.001$). Mean scores obtained by U.S. Asians for ‘Wholegrain’ and ‘Sugar Sweetened Beverages and Fruit Juice’ (SSB) were most notably higher than remaining racial groups (wholegrain mean score: Asian = 3.45 *vs.* White = 2.34, Black = 1.84 and Other = 1.88, $P < 0.001$; SSB mean score: Asian = 9.03 *vs.* White = 7.09, Black = 7.27 and Other = 7.24, $P < 0.001$) (Table 2a).

DQI-I

In total, 10 of the 17 DQI-I components differed significantly across U.K. racial groups and in 8 of these 10 components, lowest scores were reported by the Black group (Table 2b). Despite high protein scores across all racial groups (>90% obtained maximum protein score) protein source variety was lowest among Black, followed by White and Asian groups (mean scores of 0.95, 1.28 and 1.34, scored out of 5, respectively). U.K. Black groups obtained significantly lower nutrient scores than remaining U.K. racial groups for ‘sodium’, ‘calcium’, and fat (‘total fat’, ‘saturated fat’ and ‘cholesterol’) achieving ≥9% less of the total maximum score for each component than their U.K. counterparts. In the U.S., the Black group achieved the lowest mean score for 10 out of the 16 DQI-I components where significant differences were reported across racial groups. Similar to the U.K., the Black group in the U.S. achieved lowest protein source variety (1.50/5) and calcium (2.41/5) and iron (3.84/5) scores ($P < 0.001$) (Table 2b). However, the U.S. Black group obtained slightly higher scores (i.e. lower intake) for ‘total fat’, ‘saturated fat’ and ‘sodium’ than their White counterparts, but scores remained significantly lower than Asian and Other groups.

EAT-lancet

Component scores for ‘Unsaturated Oils’, ‘Red and Processed Meat’, ‘Pork’, ‘Dairy’ and ‘Potatoes’ differed significantly across U.K. racial groups. Highest scores obtained for these EAT-Lancet components varied across races. Mean score of unsaturated oils was highest among Asian and Other groups and lowest among Black groups (Asian and Other: 0.32 *vs.* Black: 0.15; $P = 0.005$); highest mean ‘Red and Processed Meat’ and ‘Pork’ scores was obtained by the Asian group (1.79; $P < 0.001$ and 2.50; $P < 0.001$ respectively); Black groups achieved the highest mean score for ‘Dairy’ and ‘Potatoes’ but scores were similar to Asian and Other groups, the White group had the lowest mean score indicating high intake of dairy and potatoes (Table 2c). Significantly different mean scores were observed for all components except ‘Unsaturated Oils’ across U.S. racial groups. The Asian group obtained the highest scores for all EAT-Lancet components except ‘Poultry’ where the White group reported the highest score (i.e. lowest intake) (2.31 *vs.* 1.97, 2.12 and 2.13 by White, Black, Asian, and Other groups respectively; $P < 0.001$) (Table 2c). The U.S. Asian group had significantly higher scores for ‘Wholegrain’, ‘Red and Processed Meat’ and ‘Added Sugar’ than all other racial

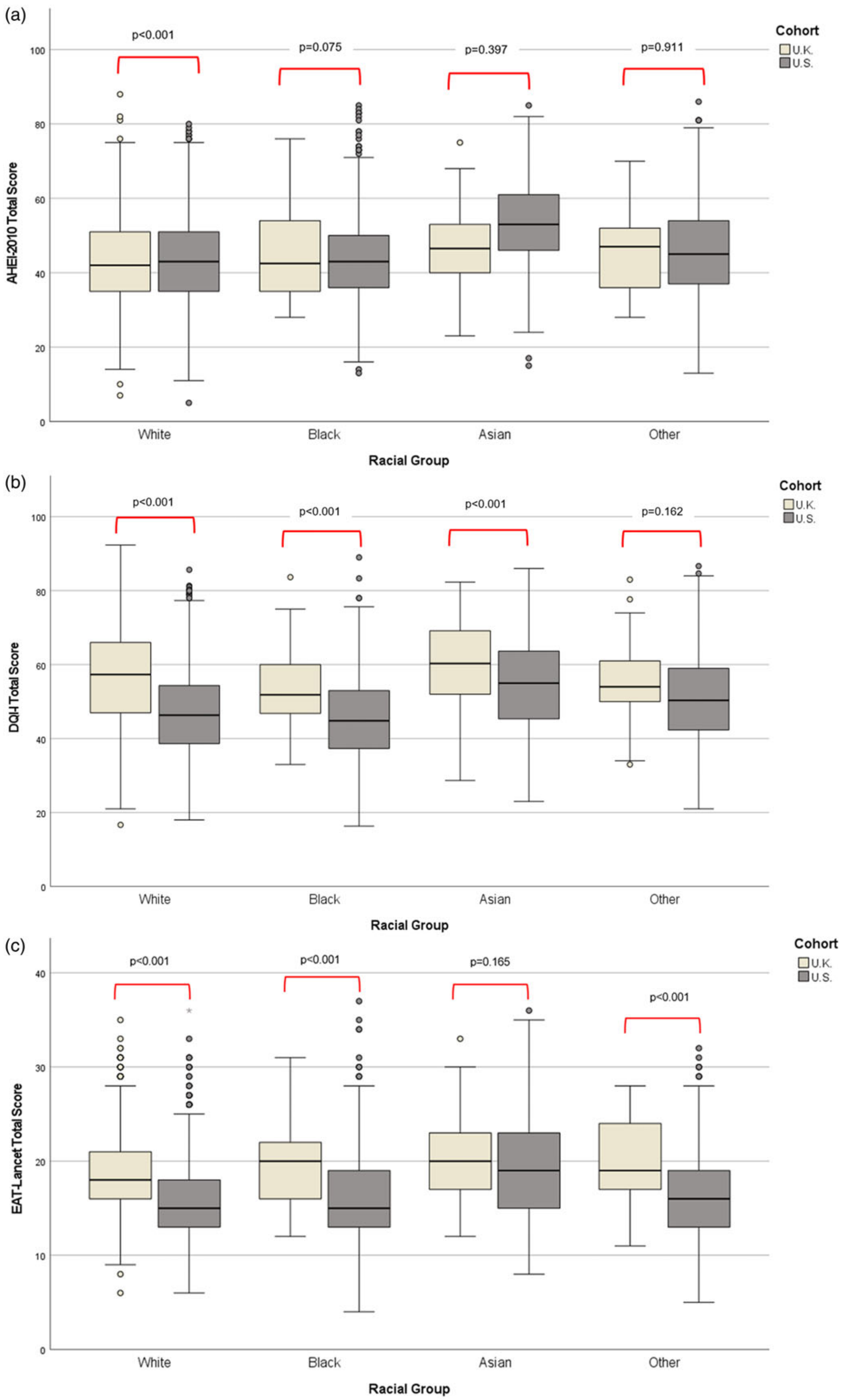

Fig. 2. (a) Median AHEI-2010 total scores and ANCOVA across racial groups in the U.K. vs. U.S. AHEI-2010 = Alternative Healthy Eating Index (2010), adjusted for age, sex, income, and education with Bonferroni pairwise comparisons corrected P value shown. (b) Median DQI-I total scores and ANCOVA across racial groups in the U.K. vs. U.S. DQI-I = Diet Quality Index International, adjusted for age, sex, income, and education with Bonferroni pairwise comparisons corrected P value shown. (c) Median EAT-Lancet total scores and ANCOVA across racial groups in the U.K. vs. U.S. Adjusted for age, sex, income, and education with Bonferroni pairwise comparisons corrected P value shown.

https://doi.org/10.1017/jns.2024.64 Published online by Cambridge University Press

Table 2a. AHEI-2010 component scores across racial groups (U.K. and U.S.)

United Kingdom (U.K.)									
AHEI-2010 Components	*White*		*Black*		*Asian*		*Other*		
Score Range; 0 (lowest)–10 (highest quality)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	P value
Fruit	2.78 (2.87)	27.75	3.20 (3.62)	32.00	2.82 (8.83)	28.20	2.86 (2.72)	28.65	0.940
Vegetables	2.33 (2.21)	23.29	2.63 (2.55)	26.25	2.54 (2.42)	25.40	2.16 (2.17)	21.62	0.765
Plant Protein	1.28 (2.02)	12.84	0.78 (1.33)	7.75	1.29 (2.19)	12.90	1.95 (2.57)	19.46	0.132
SSB and Fruit Juice	5.16 (4.32)	51.63	4.93 (4.36)	49.25	5.99 (4.20)	59.90	5.43 (4.24)	54.32	0.279
Wholegrain	2.88 (2.94)	28.76	2.15 (2.81)	21.50	3.23 (2.92)	32.30	2.27 (2.71)	22.70	0.052
Red & Processed Meat	6.92 (2.99)	69.16	7.25 (3.19)	72.50	8.30 (2.63)	83.00	7.24 (3.17)	72.43	<0.001
Total n3polyunsaturated fat as total energy (%)	5.07 (2.05)	50.67	5.60 (2.32)	56.00	5.04 (2.02)	50.40	5.62 (2.30)	56.22	0.082
Polyunsaturated fat as total energy (%)	5.45 (2.52)	54.50	5.88 (1.92)	58.75	5.00 (1.42)	50.00	5.68 (2.40)	56.76	<0.001
Alcohol	6.20 (1.60)	62.04	6.80 (1.68)	68.00	6.90 (1.62)	69.00	6.86 (1.73)	68.65	0.158
Sodium	5.21 (2.38)	52.08	5.70 (2.58)	57.00	5.32 (2.50)	53.20	4.76 (2.37)	47.57	0.472
Total Score: 0 (lowest)–100 (highest quality)	***43.33 (11.23)***	***43.33***	***44.90 (12.86)***	***44.90***	***46.43 (9.87)***	***46.43***	***44.84 (11.08)***	***44.84***	***<0.001***
United States (U.S.)									
AHEI-2010 Components	*White*		*Black*		*Asian*		*Other*		
Score Range; 0 (lowest)–10 (highest quality)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	P value
Fruit	2.03 (3.14)	20.344	1.85 (3.15)	18.51	3.61 (3.79)	36.09	2.87 (3.69)	28.70	<0.001
Vegetables	3.99 (3.50)	39.873	3.52 (3.47)	35.21	5.47 (3.62)	54.68	4.54 (3.60)	45.40	<0.001
Plant Protein	2.19 (3.40)	21.949	1.69 (3.18)	16.90	3.48 (4.10)	34.77	3.00 (3.88)	30.00	<0.001
SSB and Fruit Juice	7.09 (3.84)	70.879	7.27 (3.52)	72.75	9.03 (2.14)	90.32	7.24 (3.57)	72.40	<0.001
Wholegrain	2.34 (3.33)	23.42	1.84 (3.01)	18.42	3.45 (3.97)	34.49	1.88 (3.07)	18.80	<0.001
Red & Processed Meat	5.88 (3.90)	58.841	6.51 (3.82)	65.08	7.33 (3.57)	73.32	6.36 (3.91)	63.60	<0.001
EPA and DHA	1.86 (2.83)	18.637	2.49 (3.24)	24.86	3.00 (3.80)	30.04	2.19 (3.02)	21.90	<0.001
Polyunsaturated fat as total energy (%)	7.96 (1.71)	79.605	8.38 (1.58)	83.79	7.76 (1.66)	77.59	7.67 (1.69)	76.70	<0.001
Alcohol	4.94 (1.88)	49.363	4.93 (1.81)	49.25	4.93 (1.21)	49.34	4.84 (2.52)	48.40	0.975
Sodium	4.83 (2.29)	48.344	5.02 (2.46)	50.15	4.78 (2.34)	47.77	4.91 (1.77)	49.10	0.348
Total Score: 0 (lowest)–100 (highest quality)	***43.13 (12.22)***	***43.13***	***43.49 (11.23)***	***43.39***	***52.84 (11.34)***	***52.84***	***45.50 (12.09)***	***45.50***	***<0.001***

SSB, Sugar Sweetened Beverages; P value based on Kruskal-Wallis tests; EPA and DHA, Eicosapentaenoic acid and Docosahexaenoic acid.

https://doi.org/10.1017/jns.2024.64 Published online by Cambridge University Press

Table 2b. DQI-I component scores across racial groups (U.K. and U.S.)

United Kingdom (U.K.)									
	White		*Black*		*Asian*		*Other*		P *value*
DQI-I Components (Score Range; lowest-highest quality)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	
Food group variety (0–15)	7.51 (4.42)	50.06	5.85 (3.66)	39.00	7.89 (3.85)	52.60	7.05 (3.62)	47.03	0.049
Protein source variety (0–5)	1.28 (1.26)	25.51	0.95 (0.99)	19.00	1.34 (1.44)	26.80	1.43 (1.17)	47.75	0.336
Vegetables (0–5)	2.81 (1.94)	56.16	2.98 (2.04)	59.50	2.94 (1.87)	58.80	2.62 (2.02)	52.43	0.778
Fruit (0–5)	1.65 (1.98)	32.99	1.65 (2.14)	33.00	1.71 (1.93)	34.20	1.70 (1.98)	34.05	0.983
Wholegrains (0–5)	2.53 (2.23)	50.70	1.90 (2.23)	38.00	2.93 (2.20)	58.60	2.14 (2.03)	42.70	0.047
Fibre (0–5)	2.10 (1.61)	42.07	1.63 (1.60)	32.50	2.36 (1.49)	47.20	1.65 (1.69)	32.97	0.029
Protein (0–5)	4.71 (0.77)	94.11	4.65 (0.77)	93.00	4.53 (0.94)	90.60	4.95 (0.33)	98.92	0.022
Iron (0–5)	3.68 (1.56)	73.61	2.98 (2.04)	59.50	3.62 (1.54)	72.40	3.14 (1.78)	62.70	0.016
Calcium (0–5)	4.10 (1.28)	81.97	3.23 (1.53)	64.50	3.65 (1.40)	73.00	3.97 (1.36)	79.46	<0.001
Vitamin C (0–5)	4.40 (1.31)	87.97	4.63 (1.00)	92.50	4.52 (1.02)	90.40	4.41 (1.30)	88.11	0.784
Total Fat (0–6)	4.12 (2.29)	68.82	4.05 (2.21)	36.78	4.53 (2.02)	75.50	3.57 (2.63)	59.46	0.233
Saturated Fat (0–6)	3.43 (2.07)	57.21	4.05 (1.87)	31.11	4.47 (1.83)	74.50	3.97 (1.88)	66.22	<0.001
Cholesterol (0–6)	4.85 (2.04)	80.78	4.13 (2.51)	41.89	4.74 (2.22)	79.00	4.46 (2.41)	74.33	0.197
Sodium (0–6)	5.20 (1.58)	86.60	5.10 (1.69)	28.19	5.25 (1.67)	87.50	5.35 (1.25)	89.19	0.858
Empty Calorie Foods (0–6)	0.57 (1.47)	9.48	1.73 (2.34)	39.04	1.17 (1.70)	19.50	0.65 (1.44)	10.81	<0.001
Macronutrient ratio (0–6)	1.52 (1.36)	25.33	1.33 (1.57)	26.17	2.18 (1.85)	36.33	1.37 (1.28)	22.83	0.007
Fatty Acid ratio (0–6)	2.06 (0.98)	51.62	2.20 (1.16)	55.00	2.44 (1.03)	61.00	2.27 (0.90)	56.76	0.001
Total Score: 0 (lowest)–100 (highest quality)	***56.52 (13.10)***	***56.52***	***53.01 (11.56)***	***53.01***	***60.27 (11.92)***	***60.27***	***54.69 (11.82)***	***54.69***	***0.013***
United States (U.S.)									
DQI-I Components (Score Range; lowest-highest quality)	*White*		*Black*		*Asian*		*Other*		P *value*
	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	
Food group variety (0–15)	8.18 (3.39)	54.55	7.37 (3.34)	49.10	9.00 (3.09)	60.00	8.39 (3.39)	55.96	<0.001
Protein source variety (0–5)	1.66 (1.40)	33.15	1.50 (1.44)	29.96	1.72 (1.44)	34.30	1.95 (1.56)	38.96	<0.001
Vegetables (0–5)	0.75 (1.57)	14.92	0.72 (1.56)	14.47	1.48 (2.01)	29.58	1.12 (1.87)	22.36	<0.001
Fruit (0–5)	3.01 (2.12)	60.29	2.62 (2.18)	52.41	3.58 (1.98)	71.51	3.13 (2.10)	62.69	<0.001
Wholegrains (0–5)	0.78 (1.57)	15.67	0.60 (1.41)	12.06	1.41 (1.98)	28.11	0.62 (1.41)	12.34	<0.001
Fibre (0–5)	1.30 (1.64)	26.01	1.16 (1.61)	23.16	2.28 (1.89)	45.55	1.94 (1.78)	38.85	<0.001
Protein (0–5)	4.59 (0.94)	91.75	4.38 (1.18)	87.63	4.65 (0.85)	93.09	4.57 (0.95)	91.41	<0.001
Iron (0–5)	4.13 (1.50)	82.64	3.84 (1.69)	76.80	4.10 (1.49)	81.92	4.06 (1.52)	81.27	<0.001

(*Continued*)

Table 2b. *(Continued)*

United Kingdom (U.K.)	*White*		*Black*		*Asian*		*Other*		*P value*
DQI-I Components (Score Range; lowest-highest quality)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	
Calcium (0–5)	3.09 (1.68)	61.75	2.41 (1.83)	48.13	2.55 (1.67)	50.94	2.96 (1.72)	59.26	<0.001
Vitamin C (0–5)	2.27 (2.14)	45.38	2.57 (2.15)	51.44	3.21 (1.99)	64.19	2.77 (2.15)	55.48	<0.001
Total Fat (0–6)	1.14 (1.52)	18.92	1.21 (1.57)	20.23	1.90 (1.72)	31.70	1.65 (1.70)	27.55	<0.001
Saturated Fat (0–6)	3.04 (1.96)	50.64	3.74 (1.91)	62.34	4.32 (1.83)	72.08	3.82 (1.98)	63.69	<0.001
Cholesterol (0–6)	4.11 (2.51)	68.54	3.98 (2.55)	66.26	4.11 (2.52)	68.49	3.96 (2.55)	66.00	0.296
Sodium (0–6)	2.45 (2.45)	40.86	2.73 (2.45)	45.56	2.43 (2.41)	40.47	2.69 (2.45)	44.87	0.006
Empty Calorie Foods (0–6)	0.79 (1.76)	13.19	0.87 (1.76)	14.53	2.00 (2.28)	33.30	1.26 (2.06)	20.96	<0.001
Macronutrient ratio (0–6)	1.61 (1.38)	26.88	1.67 (1.41)	27.89	1.90 (1.77)	31.75	1.75 (1.61)	29.18	0.309
Fatty Acid ratio (0–6)	2.28 (0.94)	57.04	2.57 (1.01)	64.37	2.55 (0.99)	63.63	2.42 (0.97)	60.59	<0.001
Total Score: 0 (lowest)–100 (highest quality)	***46.84 (11.56)***	***46.84***	***45.45 (11.51)***	***45.45***	***54.89 (12.55)***	***54.89***	***51.03 (12.44)***	***54.89***	***<0.001***

P value based on Kruskal-Wallis tests. Empty calorie foods include chocolate and non-chocolate confectionary, sugar sweetened beverages, and savoury snacks.

groups and significantly higher scores 'Fish' and 'Dairy' than the White group.

Association across indices

Spearman correlations reported moderate associations between AHEI-2010 and DQI-I scores among racial groups in both the U.K. and U.S.; medium-high correlations were found for White and Asian groups in the U.K. and the U.S. ($r = 0.45$–0.65; $P < 0.001$; Table 3), however moderate-low correlations were reported for U.K. Other and U.S. Black groups. Moderate-high correlations were reported between AHEI-2010 and EAT-Lancet scores across, and correlations were similar across all racial groups in both countries ($r = 0.53$–0.65; $P < 0.001$; Table 3). Associations between DQI-I and EAT-Lancet differed across racial groups and by country. Again correlations across indices were similar within White and Asian groups in both countries (moderate-high); correlations within Black and Other differed across countries (Table 3).

Discussion

Analysing quality of habitual intake is a useful strategy to monitor population adherence to public health advice.[45–47] However insight into the quality of dietary intake across different cultural groups is limited despite the increased prevalence of diverse racial and ethnic groups living in developed regions worldwide.[14] This secondary analysis of recent NDNS and NHANES data demonstrates that while differences exist across racial groups, these differences follow similar trends by country and diet quality index. Highest quality in terms of healthiness, dietary adequacy and sustainability were reported by Asian groups in the U.K. and U.S., with more sustainable intake – as defined by EAT-Lancet - reported among U.K. racial groups. Greater adherence to recommendations for wholegrains, vegetables and legumes, red and processed meat intake would improve quality of U.K. and U.S. population diets as a whole.

To contextualise these results, this research was compared to similar work examining diet quality in U.K. and U.S. populations. A worldwide analysis of diet quality described a global mean AHEI score as *'modest'*, where Asian populations reported higher diet quality (AHEI) compared to U.S. and South American populations.[48] Similarly, a higher DQI-I score was reported among Chinese adults compared to U.S adults., with better vegetable, wholegrain and fatty acid contributing to higher diet quality among the Chinese group.[11] Previous insights into diet quality of U.S. adults using NHANES data reported a mean Healthy Eating Index (HEI) score of 58, with scores differing across age groups, lowest among ages 14–18 years.[15] A recent report by Tao *et al.,* examined diet quality trends specifically within racial groups in the U.S. In agreement with results presented here, the analysis reported highest total HEI scores among Asian adults, whose quality of intake has remained stable between 2011 and 2018.[16] Diet quality was poorest among Black adults and worryingly, HEI scores of White and Black groups are on a downward trajectory since 2011. Examining intake of individual HEI components, poor adherence to legumes, fruit and wholegrains contributed to suboptimal total scores.[16] Similarly in the U.K., low intake of wholegrains and legumes and high intake of processed meat has been noted, contributing to moderate total diet quality scores (HEI-2015 score of 60, AHEI-2010 score of 50) among U.K. adults.[49]

https://doi.org/10.1017/jns.2024.64 Published online by Cambridge University Press

Table 2c. EAT-Lancet component scores across racial groups (U.K. and U.S.)

United Kingdom (U.K.)									
EAT-Lancet Components	*White*		*Black*		*Asian*		*Other*		
Score Range; 0 (lowest)–3 (highest)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	P *value*
Vegetables	0.65 (0.82)	21.69	0.75 (0.93)	25.00	0.69 (0.90)	23.00	0.70 (0.85)	23.23	0.957
Fruit	1.10 (1.12)	36.62	1.13 (1.20)	37.50	1.19 (1.07)	39.67	1.18 (1.13)	39.39	0.732
Unsaturated oils	0.24 (0.54)	8.00	0.15 (0.43)	5.00	0.32 (0.62)	10.67	0.32 (0.66)	10.67	0.045
Legumes	0.82 (1.06)	27.45	0.55 (0.96)	18.33	0.67 (1.00)	22.33	0.85 (1.20)	28.28	0.170
Nuts	0.21 (0.62)	6.94	0.23 (0.70)	7.50	0.24 (0.67)	8.00	0.64 (1.11)	21.21	0.104
Wholegrains	0.15 (0.41)	5.15	0.05 (0.22)	1.67	0.14 (0.38)	4.67	0.06 (0.24)	2.02	0.428
Fish	1.16 (1.35)	38.50	1.38 (1.35)	45.83	1.08 (1.34)	36.00	1.79 (1.39)	59.60	0.058
Red & Processed Meat	1.12 (1.34)	37.46	1.08 (1.31)	35.83	1.79 (1.44)	59.67	1.52 (1.44)	50.51	<0.001
Pork	1.48 (1.32)	49.18	1.95 (1.26)	65.00	2.50 (1.04)	83.33	1.48 (1.42)	49.49	<0.001
Poultry	1.99 (1.07)	66.19	1.75 (1.13)	58.33	1.74 (1.18)	58.00	1.79 (0.99)	59.60	0.099
Eggs	1.83 (1.21)	60.86	1.75 (1.21)	58.33	1.69 (1.26)	56.33	1.42 (1.32)	47.47	0.327
Dairy	2.56 (0.76)	85.23	2.98 (0.16)	99.17	2.78 (0.60)	92.67	2.76 (0.56)	91.92	<0.001
Potatoes	2.49 (0.77)	82.89	2.88 (0.40)	95.83	2.82 (0.48)	94.00	2.72 (0.58)	90.63	<0.001
Added Sugar	1.89 (1.00)	63.00	2.05 (0.93)	68.33	2.21 (0.88)	73.67	2.03 (0.90)	67.67	0.457
Total Score: 0 (lowest)–42 (highest)	***17.67 (4.42)***	***42.07***	***18.65 (4.45)***	***44.05***	***19.86 (4.31)***	***47.29***	***18.92 (4.75)***	***45.05***	***<0.001***
United States (U.S.)									
EAT-Lancet Components	*White*		*Black*		*Asian*		*Other*		
Score Range; 0 (lowest)–3 (highest)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	P value
Vegetables	1.40 (1.16)	46.67	1.22 (1.16)	40.67	1.71 (1.16)	57.11	1.43 (1.17)	47.82	<0.001
Unsaturated Oils	1.70 (1.02)	56.67	1.72 (1.07)	57.33	1.71 (0.99)	56.98	1.64 (1.03)	54.61	0.266
Legumes	0.51 (1.10)	17.00	0.42 (1.02)	14.00	1.00 (1.37)	33.33	0.99 (1.39)	33.05	<0.001
Nuts	0.67 (1.12)	37.33	0.47 (1.00)	15.67	0.84 (1.24)	27.86	0.50 (1.00)	16.57	<0.001
Wholegrains	0.19 (0.47)	6.33	0.14 (0.42)	4.67	0.37 (0.65)	12.26	0.14 (0.42)	4.82	<0.001
Fish	0.37 (0.97)	12.33	0.62 (1.19)	20.67	0.73 (1.26)	24.40	0.46 (1.06)	15.34	<0.001
Red & Processed Meat	0.15 (0.63)	5.00	0.28 (0.84)	28.00	0.50 (1.09)	16.73	0.24 (0.79)	8.07	<0.001
Poultry	2.31 (1.07)	77.00	1.97 (1.23)	65.67	2.12 (1.16)	70.63	2.13 (1.18)	70.86	<0.001
Eggs	2.25 (1.15)	75.00	2.34 (1.09)	78.00	2.20 (1.15)	73.33	2.15 (1.22)	71.74	0.003
Pork	0.40 (1.00)	13.33	0.55 (1.14)	18.33	0.64 (1.22)	21.45	0.42 (1.02)	14.06	<0.001
Dairy	1.17 (1.22)	39.00	1.65 (1.28)	55.00	1.89 (1.20)	63.02	1.37 (1.25)	45.79	<0.001

(*Continued*)

https://doi.org/10.1017/jns.2024.64 Published online by Cambridge University Press

Table 2c. (*Continued*)

United Kingdom (U.K.)									
EAT-Lancet Components	*White*		*Black*		*Asian*		*Other*		
Score Range; 0 (lowest)–3 (highest)	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	Mean (SD)	Percent	P *value*
Potatoes	2.30 (1.03)	76.67	2.35 (0.99)	78.33	2.55 (0.81)	85.16	2.49 (0.88)	83.01	<0.001
Added Sugar	1.66 (1.07)	55.33	1.54 (1.07)	51.33	2.18 (0.91)	72.64	1.73 (1.07)	57.79	<0.001
Fruit	0.52 (0.88)	17.33	0.48 (0.89)	16.00	0.96 (1.08)	32.01	0.77 (1.04)	25.58	<0.001
Total Score: 0 (lowest)–42 (highest)	***15.61 (4.13)***	***37.17***	***15.76 (4.53)***	***37.52***	***19.41 (5.11)***	***46.21***	***16.47 (4.35)***	***39.21***	***<0.001***

P value based on Kruskal-Wallis tests.

Table 3. Spearman correlations across index scores among U.K. and U.S. racial groups

	AHEI-2010 (/100)	DQI-I (/100)	EAT-Lancet (/42)	AHEI-2010 *vs.* DQI-I	AHEI-2010 *vs.* EAT Lancet	DQI-I *vs.* EAT Lancet
Racial Group	Median (IQR)	Median (IQR)	Median (IQR)	*r,* P value	*r,* P value	*r,* P value
United Kingdom (U.K.)						
White	42.00 (16.00)	57.33 (19.00)	18.00 (5.00)	0.50[a]	0.58[a]	0.35[a]
Black	42.50 (19.00)	51.83 (13.59)	20.00 (6.00)	0.51[a]	0.57[a]	0.40 (0.01)
Asian	46.50 (13.00)	60.33 (17.41)	20.00 (6.00)	0.46[a]	0.53[a]	0.43[a]
Other	47.00 (16.50)	54.00 (12.84)	19.00 (7.00)	0.28	0.65[a]	0.21
United States (U.S.)						
White	43.00 (16.00)	46.33 (15.67)	15.00 (5.00)	0.45[a]	0.58[a]	0.36[a]
Black	43.00 (14.00)	44.83 (15.67)	15.00 (6.00)	0.39[a]	0.56[a]	0.27[a]
Asian	53.00 (15.25)	55.00 (18.41)	19.00 (8.00)	0.56[a]	0.65[a]	0.53[a]
Other	45.00 (17.00)	50.32 (16.67)	16.00 (6.00)	0.56[a]	0.59[a]	0.46[a]

IQR, interquartile range.
[a]=P < 0.001.

As demonstrated by this investigation, similar improvements to dietary patterns, for example reducing red/processed meat and increasing vegetable consumption, will not only enhance adherence to dietary and healthy eating guidelines but also improve the sustainability of food intake. This research has identified that there is no specific dietary component which is of significant concern in one racial group and not another. Across the board an increase in wholegrain, fruit, vegetable, fish, and legume intake and a reduction of red and processed meat would improve adherence to current food-based dietary guidelines, which have long been cited as key dietary components whose intake does not align to recommendations, across many population groups.[18,46] However, it is evident from this and previous research that nutrition adequacy does vary across racial groups and group-specific intakes must be considered when developing and implementing dietary advice.[14] Stronger adherence to existing food-based dietary guidelines would reduce green-gas emissions caused by diet by approximately 25% without the need for total elimination of food groups, meaning inadequacies of nutrients primarily sourced from animal produce (e.g. vitamin B12 and calcium) would not be of serious public health concern moving forward.[50,51] Furthermore, promoting food behaviour change such as limiting food waste would further reduce the carbon footprint of agri-food systems without risking the dietary adequacy and health of vulnerable minority groups within a country.[52,53]

It is true that similar changes in dietary patterns are needed by U.K. and U.S. racial groups, nonetheless, some groups may need more targeted support than others to adhere to recommendations; generic messaging like 'increase consumption of plant-based food' will not provide a platform to address dietary inequalities.[14] High diet quality has been consistently reported among Asian groups relative to other racial groups, both here and elsewhere.[16,18,48,54] More research is needed to understand the key drivers of specific dietary patterns and the extent to which food choice influences vary across racial groups in order to mitigate the potential racial health disparities resulting from poor dietary intake.[14,18,55] Understanding taste and cooking preferences and incorporating more ethnic foods into sample healthy and sustainable recipes/food pyramids could undoubtedly help encourage dietary change among minority racial groups.[56–58] As highlighted by this research, the physical environment may be integral to diet quality of populations, where cultural and social factors have long been associated with dietary intake and quality of intake.[18,59–61] Deeper knowledge of how social and environmental factors impact dietary patterns across racial groups would provide greater detail of how public guidance and initiatives could encourage healthy and sustainable intake across population subgroups most in need.[62] Until social and environmental influences are fully understood across all population subgroups, improving dietary adequacy, healthiness and sustainability of diets of minority groups would be extremely challenging.

Strengths and limitations

To the researchers' knowledge, this is the first paper to investigate diet quality of racial groups, examining the dietary adequacy, disease risk and sustainability of dietary intake in combination across the U.K. and U.S. Previously validated dietary indices were applied to national dietary intake data: AHEI-2010 – previously associated with favourable health outcomes including cardiometabolic parameters and lower rates of chronic disease incidence and mortality[12,29,63]; DQI-I – shown to facilitate cross-country comparisons of diet quality, allowing flexibility to apply country-specific recommendations[11,64] and EAT-Lancet (Stubbendorf *et al.*, 2022 approach) – determining adherence to EAT-Lancet recommendations and associated with mortality risk.[13,36]

Some limitations of this work must be noted. Not all components assessed in AHEI-2010, DQI-I, and EAT-Lancet indices were available in NDNS and NHANES datasets, resulting in estimation of certain dietary component scores and alternations to AHEI-2010 were necessary. In addition, very poor distribution of participants across racial groups was achieved in the NDNS dataset (however were representative of U.K. census data) and although findings complement those reported across U.S. racial groups interpretation of results must be cautioned. In future national dietary surveys oversampling of minority groups (as done for NHANES 2017–2018 data) may be advisable to ensure accurate and meaningful analysis can be performed on data collected from population subgroups.

Conclusion

Variances in diet quality were observed across racial groups in the U.K. and U.S., White groups exhibited poorest adherence to EAT-Lancet recommendations and Asian groups demonstrated strongest adherence to healthy and sustainable diets. Unfavourable intake of wholegrain, fruit, vegetables/legumes, and processed meats was consistently reported by racial groups however better adherence to intake recommendations were found among Asian and Other groups than Black and White groups in both countries. This research highlights that to promote optimum healthy and sustainable dietary intake across all subgroups of the population equally, current dietary patterns of population subgroups need to be fully understood so that diversity in consumption across cultural groups can be adequately addressed where required. In the future, food-based dietary guidelines should not only be developed with dietary adequacy and planetary health in mind, but also consider the individual needs of population subgroups, tailoring recommendations to specific cultures where appropriate.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/jns.2024.64.

Acknowledgements. This research was supported by the Food Nutrition Security Cloud. Food Nutrition Security Cloud (FNS Cloud) has received funding from the European Union's Horizon 2020 Research and Innovation programme (H2020-EU.3.2.2.3. — A sustainable and competitive agri-food industry) under Grant Agreement No. 863059 — http://www.fns-cloud.eu.

Declarations of interests. The author(s) declare none.

Authorship. GB contributed to the conception and design of the study, analysis and interpretation of data and writing of the manuscript. ERG contributed to the conception and design of the study, interpretation of data and critically analysed the manuscript for scientific content. All authors approved the final version of the manuscript before publication.

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Journal of Nutritional Science

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Research Article

Associations of renal sinus fat with metabolic parameters, abdominal visceral adipose tissue, metabolic syndrome, fructose intake, and blood pressure control in obese individuals with hypertension: a cross-sectional study

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Cite this article: Anvarifard P, Anbari M, Naemi Kermanshahi M, Ostadrahimi A, Aliasgharzadeh S, and Ardalan M (2024). Associations of renal sinus fat with metabolic parameters, abdominal visceral adipose tissue, metabolic syndrome, fructose intake, and blood pressure control in obese individuals with hypertension: a cross-sectional study. *Journal of Nutritional Science* **13**: e94, 1–9. doi: 10.1017/jns.2024.84

Received: 11 June 2024
Revised: 3 October 2024
Accepted: 5 November 2024

Keywords:
Renal sinus fat; Hypertension; Obesity; Fructose; Visceral adipose tissue; Metabolic syndrome

Abbreviations:
CKD, chronic kidney disease; MetS, metabolic syndrome; VAT, visceral adipose tissue; SAAT, subcutaneous abdominal adipose tissue; BMI, body mass index; RAAS, renin-angiotensin-aldosterone system; RSF, renal sinus fat; PVAT, perivascular adipose tissue; FBS, fasting blood sugar; PUFT, para- and perirenal ultrasonographic fat thickness; HOMA-IR, homeostasis model assessment of insulin resistance; DBP, diastolic blood pressure; CT, computed tomography; MRI, magnetic resonance imaging; SBP, systolic blood pressure; eGFR, estimated glomerular filtration rate; BIA, bioelectrical impedance analysis; ARB, angiotensin II receptor blockers; ACE, angiotensin converting enzyme; CCB, calcium channel blockers; WHR, waist to hip ratio; IDF, international diabetes federation; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; FFQ, food frequency questionnaire; FCT, food composition table; USDA, United States Department of Agriculture; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; SD, standard deviation; IL-6, interleukin-6; TNF-α, tumour necrosis factor-alpha; ALT, alanine transaminase; AST, aspartate transaminase; VLDL, very-low-density lipoprotein; HFCS, high-fructose corn syrup

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Abstract

Renal sinus fat (RSF) crucially influences metabolic regulation, inflammation, and vascular function. We investigated the association between RSF accumulation, metabolic disorders, and nutritional status in obese individuals with hypertension. A cross-sectional study involved 51 obese hypertensive patients from Salamat Specialized Community Clinic (February–September 2022). Basic and clinical information were collected through interviews. Data included anthropometrics, blood pressure, number of antihypertensive medications, body composition (bioelectrical impedance analysis), dietary intake (semi-quantitative 147-item food frequency questionnaire), and blood samples. Renal sinus fat was measured via ultrasonography. Statistical analyses included Pearson correlation, binary logistic regression, and linear regression. RSF positively correlated with abdominal visceral adipose tissue (VAT) area ($P = 0.016$), systolic blood pressure (SBP) ($P = 0.004$), and diastolic blood pressure (DBP) ($P = 0.005$). A strong trend toward a positive association was observed between antihypertensive medications and RSF ($P = 0.062$). In linear regression, RSF was independently associated with abdominal VAT area, SBP, and DBP after adjusting for confounders. After considering other risk factors, RSF volume relates to prescribed antihypertensive medications, hypertension, and central fat accumulation in obese hypertensive subjects. These findings suggest the need for further investigations into whether RSF promotes metabolic disorders.

1. Introduction

Obesity presents a significant global health challenge, with its prevalence having tripled over the past four decades (World Health Organization, 2016). In 2016, more than 1.9 billion adults were classified as overweight, of which over 650 million were categorised as obese, accounting for 39% and 13% of the adult population, respectively (World Health Organization, 2016). Lifestyle changes towards consumption of calorie-dense food and adoption of sedentary lifestyles are two basic forces spreading this malady[(1)]. If current trends persist, the global prevalence of obesity is expected to reach 21% in women and exceed 18% in men by 2025[(2)]. The adverse effects of obesity, particularly abdominal obesity, on various cardiovascular and metabolic conditions such as dyslipidemia, hypertension, diabetes, chronic kidney disease (CKD), cardiovascular disease, and cardiovascular mortality are well-documented[(3)]. Metabolic syndrome (MetS), characterised by the co-occurrence of hyperlipidaemia, insulin resistance, hypertension, and abdominal obesity, is a significant driver of many major diseases and is highly prevalent worldwide[(4,5)]. The incidence of MetS commonly parallels the incidence of obesity and type 2 diabetes mellitus[(6)]. Early diagnosis of MetS is crucial for the identifying high-risk patients who require aggressive lifestyle modifications[(4)]. In recent years, excess abdominal visceral adipose tissue (VAT), also called visceral obesity, rather than total or subcutaneous abdominal adipose tissue (SAAT), has been acknowledged as a primary predictor of metabolic and cardiovascular disease and overall mortality independent of generalised obesity and body mass index (BMI)[(7)]. Visceral adipose tissue is regarded as a form of 'ectopic fat', contributing to systemic inflammation, dyslipidemia, insulin resistance, and subsequently, increasing the risk of developing MetS and cardiovascular diseases[(4)]. Ectopic fat can accumulate in various areas of the body, such as the liver, muscle, pericardium, and perivascular area[(8)]. The kidneys, which are surrounded by abdominal VAT, are susceptible to ectopic fat accumulation in the renal sinus[(3)].

The renal sinus is a perirenal region bounded from the hilum of the kidney to the edge of the renal parenchyma where the renal vein, the renal artery, lymphatic vessels, and the ureter enter the kidney[9]. Mechanistically, excessive accumulation of fat in the renal sinus can elevate intra-abdominal pressure, compressing low-pressure renal venous structures and leading to renal volume expansion, increased renal interstitial pressure, and activation of the renin-angiotensin-aldosterone system (RAAS)[9]. RAAS activation contributes to hypertension, atherosclerosis, insulin resistance, and other obesity-related adverse outcomes[10]. Renal sinus fat can be easily measured by computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound[4]. The renal sinus fat (RSF) is similar to perivascular adipose tissue (PVAT) in terms of its characteristics. Perivascular adipose tissue is a type of active endocrine tissue that plays a crucial role in regulating inflammation, vascular function, and metabolism[11,12]. These characteristics suggest a potential role for RSF in MetS regulation. Recent studies have linked RSF to MetS components such as central obesity, hypertension, insulin resistance, and dyslipidemia. Chughtai HL. et al. demonstrated that higher RSF volume was independently associated with stage II hypertension, an increased number of medications required for hypertension management, abdominal fat, and hyperlipidaemia in individuals at risk for cardiovascular events. However, their study found no significant difference in RSF volume between patients with and without diabetes, nor was RSF associated with fasting blood sugar (FBS) or BMI[10]. Similarly, a cross-sectional study involving individuals with normal glucose levels, prediabetes, and diabetes revealed that RSF volume increased significantly in prediabetic subjects and was strongly associated with VAT and hypertension[13]. De Pergola G. et al. found a positive association between para- and perirenal ultrasonographic fat thickness (PUFT) and waist circumference, insulin levels, homeostasis model assessment of insulin resistance (HOMA-IR), and mean 24-hour diastolic blood pressure (DBP) in overweight and obese individuals. However, no significant correlation was observed between PUFT and BMI or FBS[14]. Another cross-sectional study identified adipose tissue deposition, particularly in the left renal sinus, as being related to VAT levels; however, reductions in VAT were not mirrored by decreases in RSF accumulation[15]. Additionally, Guo XL. et al. showed that perirenal fat thickness was significantly associated with MetS[4].

Modern communities, mainly those with high obesity rates, are characterised by high intake of fructose[16]. Despite the rationale that dietary fructose and fructose-sweetened beverage consumption can disturb several functions in adipocytes and increase VAT, to date, no studies have yet explored the association between RSF and fructose intake[7,16].

Based on previous anatomical and cross-sectional studies, we hypothesised that increased RSF is associated with a higher risk of developing MetS. Additionally, high-fructose consumption is expected to contribute to RSF expansion. Therefore, we designed this cross-sectional study to examine the associations between RSF, metabolic parameters, abdominal VAT, MetS, fructose intake, and blood pressure control in obese individuals with hypertension.

2. Materials and methods

2.1. Study design

In the current cross-sectional study, obese patients with hypertension were consecutively enrolled using the convenience sampling method from Salamat Specialized Community Clinic, Tabriz, Iran, from February 2022 to September 2022. The study was approved by the Medical Ethics Committee of Tabriz University of Medical Sciences (approval number: IR.TBZMED.REC.1399.1173) and was carried out according to the latest version of the Declaration of Helsinki. All participating patients provided written consent prior to their involvement in the study.

2.2. Study population

The study included hypertensive patients aged 20-75 years with BMI over 30 kg/m^2. Hypertension was characterised by a systolic blood pressure (SBP) equal to or exceeding 130 mmHg, DBP equal to or exceeding 80 mmHg, or the utilisation of antihypertensive medication[17]. Patients with renal abnormalities (such as a difference in kidney length between the right and left side of more than 1.5 cm, solitary kidney or multiple kidneys, polycystic kidney, pelvic kidney, glomerulonephritis, hydronephrosis, renal artery stenosis, or congenital renal anomalies), renal transplant, history of renal surgery, estimated glomerular filtration rate (eGFR) < 45 ml/min/1.73 m^2, liver cirrhosis, active cancer, and those who were currently pregnant or breastfeeding were excluded from the study. Additionally, subjects with an implantable cardioverter defibrillator or pacemaker were excluded due to the conditions required for performing bioelectrical impedance analysis (BIA). The sample size was determined using PASS software (version 15.0.5) based on the results of a previous related study[14]. Pearson's correlation test was selected to calculate the appropriate sample size, considering the association between PUFT and waist circumference[14]. The minimum required sample size was determined to be 49 subjects, with an alpha level of G0.05, power level of 80%, and a Pearson's correlation coefficient of 0.39. On this basis and considering the inclusion and exclusion criteria, a total of 51 subjects (39.2% male, 60.8% female) out of the initially screened 202 subjects were enrolled in the current study.

2.3. Socio-demographic, blood pressure, anthropometric, and body composition assessments

We gathered socio-demographic data including gender, age, educational background, occupation, marital status, smoking habits, and medical history through structured interviews. Blood pressure measurements were taken using a mercury sphygmomanometer (ALPK2, Japan) twice after a 30-minute rest in a seated position. The mean of the two readings was reported as the final result. We adopted the American College of Cardiology/American Heart Association (ACC/AHA) hypertension guidelines, whereby stage I hypertension was identified as having a SBP ranging from 130 to 139 mmHg or DBP ranging from 80 to 89 mmHg, and stage II hypertension was defined as having an SBP of 140 mmHg or higher or a DBP of 90 mmHg or higher. Antihypertensive agents were classified as angiotensin II receptor blockers (ARB), angiotensin-converting enzyme (ACE) inhibitors, calcium channel blockers (CCB), beta-blockers, alpha-blockers, combined alpha and beta-blockers, *alpha-2* receptor *agonists,* diuretics, and direct vasodilators. Patients were categorised based on the number of antihypertensive agents they were receiving (1, 2, 3 or more). Body weight and height were measured with participants in a straight standing position, without shoes, and with light clothing using a digital Seca scale (Seca 22089, Hamburg, Germany) and a portable stadiometer (Seca, Hamburg, Germany) with an accuracy of approximately 100 g and 0.5 cm, respectively. The body mass index was computed by dividing the body weight by the square of the height (kg/m^2). Waist and hip circumferences were measured using a nonstretchable tape to the nearest 0.1 cm at the narrowest area of the abdomen (midpoint of the lowest rib and iliac crest) and widest area of the hips (greatest protuberance of the buttocks), respectively. Waist-to-hip ratio (WHR) was calculated by dividing the waist measurement by the hip measurement. Body composition, including VAT (cm^2), was evaluated using BIA technology (Tanita BC-420MA; Tokyo, Japan), following standard procedures[18]. Participants were instructed not to engage in strenuous physical activity and to avoid consuming alcohol or caffeine the day before the BIA. They were also asked to be well hydrated but to stop drinking water an hour before the analysis. The analysis was performed after a 12-hour fasting period and with an empty bladder. All study subjects received a low-calorie diet for weight management and were encouraged to enhance their physical activity.

2.4. Definition of MetS

Metabolic syndrome was defined according to the 2006 international diabetes federation (IDF) parameters as abdominal obesity (waist circumference ≥ 94 in men, ≥ 80 cm in women), along with any two of the following criteria: (1) SBP of at least 130 mmHg or DBP of at least 85 mmHg, or the use of antihypertensive medications; (2) FBS ≥ 100 mg/dL or previously diagnosed diabetes mellitus with treatment; (3) fasting triglycerides (TG) ≥ 150 mg/dL or ongoing treatment for elevated TG; (4) high-density lipoprotein cholesterol (HDL-C) level < 40 mg/dL in men, < 50 mg/dL in women[19]. We divided participants into the MetS- and MetS + groups.

2.5. Assessment of dietary intake

Usual dietary intakes of the study subjects were assessed using a semi-quantitative 147-item food frequency questionnaire (FFQ) that had been previously evaluated for reliability and validity[20,21]. All questionnaires were administered through individual interviews conducted by qualified dietitians. The FFQ included a list of food items with standard serving sizes mostly consumed by Iranians. Participants were asked to report the frequency and amount of consumption of each item based on serving size during the last year, on a daily, weekly, monthly, or yearly basis. The portion size of consumed foods was converted to daily intakes (grams) using household measures. Daily intake of energy and each nutrient, as well as total fructose, was determined using the Iranian food composition table (FCT)[22] and United States Department of Agriculture (USDA) food composition data[23].

2.6. Measurement of RSF

To measure RSF, we followed the same method as previously described by others[14]. We used a duplex Doppler ultrasound apparatus (Acuson Sequoia 512 ultrasound system, Siemens, USA) to conduct the ultrasound examinations. The patients were positioned supine, and the probe was placed perpendicular to the skin on the lateral side of the abdomen. Longitudinal scanning was performed, and the optimal position, where the surface of the kidney was almost parallel to the skin, was found by slowly moving the probe laterally. Minimal pressure was applied to the probe to avoid compressing the fat layers. The ultrasound volume of RSF from the inner side of the abdominal musculature to the surface of the kidney was measured. The average of the maximal volumes on both sides was taken as the RSF. The correlation between RSF values measured on both sides was 0.849 ($P < 0.001$). RSF was measured three times. The intraoperator coefficient of variation was 4.6 %. The sonographer conducting the ultrasound examinations was blinded to all other aspects of the study.

2.7. Biochemical assessments

A fasting blood sample was obtained from each participant and then centrifuged to separate serum. Serum TG, total cholesterol (TC), HDL-C, and FBS were measured using commercial kits (Mancompany, Tehran, Iran) in accordance with the manufacturer's instructions. All biochemical tests were performed on fresh blood samples. The concentration of low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula[24,25].

2.8. Statistical analysis

The data were first examined for normal distribution by using the Shapiro-Wilk test. Results were expressed as mean ± standard deviation (SD) for normally distributed continuous values, median (interquartile range 25-75 percentile) for data with skewed distribution, or frequency (percentage) for qualitative variables. We compared two groups using independent samples t-test for normally distributed continuous variables and Mann–Whitney U test for non-normally distributed variables. Pearson or Spearman correlation coefficients, as appropriate, were used to evaluate univariate correlations between RSF and all investigated parameters. Linear regression analyses were used to assess the significance of covariate-adjusted cross-sectional relation of RSF (dependent variables) with VAT, SBP, and DBP. Furthermore, to test the independent relationship between MetS (dependent variables) and RSF, we constructed binary logistic regression analysis. Data are expressed as unstandardised (B) regression coefficient. All analyses were conducted using IBM SPSS Statistics software, version 25 (SPSS Inc., and Chicago, IL, USA); $P < 0.05$ was considered statistically significant.

3. Results

The general, metabolic, anthropometric, and dietary parameters of the study participants are described in Table 1. The mean age of the 51 obese patients with hypertension was 53.39 ± 9.84, ranging from 29 to 70 years old. The mean value of RSF in the study sample was 24.24 ± 11.10. The prevalence of MetS was 84.3%. Patients with and without diabetes had similar amounts of RSF ($P = 0.14$). Table 2 displays the correlations of RSF with all investigated parameters in a sample of 51 study participants. RSF was significantly and positively associated with abdominal VAT area ($r = 0.335$, $P = 0.016$), but not associated with waist circumference ($P = 0.657$) and BMI ($P = 0.554$). Male patients had significantly greater amounts of the VAT area (167.75 cm^2 versus 121.84 cm^2; $P = 0.017$), waist circumference (112.70 cm versus 107.80 cm; $P = 0.056$), and WHR (0.97 versus 0.90; $P < 0.001$) compared with female patients. On the contrary, the BMI level was significantly higher in female than male subjects (35.67 kg/m^2 versus 32.16 kg/m^2; $P < 0.001$). As the number of antihypertensive medications taken by the participants increased, there was a strong trend toward a positive correlation ($r = 0.264$, $P = 0.062$) with RSF volume, indicating an increase in RSF volume. Additionally, the correlations of RSF with SBP ($r = 0.395$, $P = 0.004$) and DBP ($r = 0.391$, $P = 0.005$) were statistically and positively significant. However, there were no significant differences in RSF volume between participants with stage I hypertension and those with stage II hypertension ($P = 0.484$).

Table 1. Characteristics of the study population

Clinical characteristics	All ($n = 51$)
Age, years	53.39 ± 9.84
Male, *n* (%)	20 (39.2)
Diabetes, *n* (%)	13 (25.5)
MetS, *n* (%)	43 (84.3)
Weight, kg	90.08 ± 12.00
Height, cm	162.20 ± 10.44
BMI, kg/m^2	34.30 ± 4.03
Waist circumference, cm	109.72 ± 8.96
SBP, mmHg	135 (125.00–145.00)
DBP, mmHg	90 (85.00–100.00)
FBS, mg/dl	101.00 (93.75–119.25)
TG, mg/dl	155 (117.50–228.50)
TC, mg/dl	173.18 ± 47.90
HDL-C, mg/dl	42.50 ± 9.79
LDL-C, mg/dl	106.52 ± 40.44
RSF, cc	24.24 ± 11.10
VAT area, cm^2	139.84 ±67.92
Total daily energy intake, kcal	2541.71 (2030.63–3325.95)
Total daily fructose intake, g	21.32 (16.30–32.10)

The data are presented as means ± standard deviation (SD), medians (interquartile range), or frequencies. MetS, metabolic syndrome; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBS, fasting blood sugar; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; RSF, renal sinus fat; VAT, visceral adipose tissue.

Table 2. Correlations between RSF (cc) and all other investigated parameters in 51 subjects under study

Variables	r	*P*-value
BMI, kg/m^2	−0.085	0.554[b]
Waist circumference, cm	0.064	0.657[b]
VAT area, cm^2	0.335	**0.016[b]**
SBP, mmHg	0.395	**0.004**[a]
DBP, mmHg	0.391	**0.005**[a]
Number of antihypertensive agents	0.264	**0.062**[a]
FBS, mg/dl	0.104	0.491[a]
TG, mg/dl	0.078	0.592[a]
TC, mg/dl	−0.032	0.829[b]
HDL-Cholesterol, mg/dl	−0.150	0.383[b]
LDL-Cholesterol, mg/dl	−0.073	0.673[b]
Total daily energy intake, kcal	−0.048	0.737[a]
Total daily fructose intake, g	−0.024	0.869[a]
Energy-adjusted fructose intake, g	−0.042	0.769[a]

[a]Data show the Spearman correlation coefficient.
[b]Data show the Pearson correlation coefficient.
RSF, renal sinus fat; BMI, body mass index; VAT, visceral adipose tissue; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBS, fasting blood sugar; TG, triglycerides; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 3. The prediction power of MetS by RSF based on binary logistic regression analysis

Dependent variable	Unstandardised β	OR	*P*-value	95% CI
MetS	0.028	1.029	0.560	0.936–1.131

MetS, metabolic syndrome; RSF, renal sinus fat; OR, odds ratio; CI, confidence interval.

Table 4. The prediction power of RSF by VAT based on linear regression analysis

Dependent variable		Unstandardised *B* (95% CI)	*P*-value
RSF	Crude	0.055 (0.011–0.099)	0.016
	Model 1	0.061 (0.012–0.111)	0.015

Model 1: Adjusted for waist circumference.
RSF, renal sinus fat; VAT, visceral adipose tissue; CI, confidence interval.

Neither lipid profile measures, including TG, TC, HDL-C, and LDL-C nor FBS showed a significant correlation with RSF ($P = 0.592$, $P = 0.829$, $P = 0.383$, $P = 0.673$, $P = 0.491$, respectively). Moreover, there were no significant correlations found between RSF and total daily fructose intake ($P = 0.869$) or total daily energy intake ($P = 0.737$). It should be noted that there was a significant positive correlation between fructose and energy intake ($r = 0.651$, $P < 0.001$), so the daily intake of fructose was adjusted for energy intake using the residual method[26], but still, no significant association with RSF was observed ($P = 0.769$). Patients with stage II hypertension were found to have a higher level of fructose consumption compared to those with stage I hypertension ($P = 0.041$). However, this significance disappeared after adjusting for energy intake ($P = 0.297$).

The association between MetS and RSF was investigated by binary logistic regression analysis (Table 3). Metabolic syndrome (dependent variable) showed no association with RSF (OR = 1.029, 95% CI, 0.936-1.131; $P = 0.560$).

To further confirm the associations of VAT, SBP, and DBP with RSF, we performed linear regression analyses. Using the RSF as a dependent variable and waist circumference as a covariate (Table 4), the results showed that VAT was a significant and independent predictor for RSF ($B = 0.061$, 95% CI, 0.012–0.111; $P = 0.015$) after adjusting for confounding factor (Model 1) (Figure 1). Moreover, considering SBP as an outcome variable and independent variables including RSF, age, and gender, the results showed that RSF was independently correlated with SBP (Table 5) (Figure 2). As shown in Table 5, linear regression analysis also confirmed that the association of DBP (dependent variable) and RSF was independent of other variables added to the model (age and gender) (Figure 3).

4. Discussion

The aim of this study is to explore the potential link between RSF and various anthropometric and metabolic parameters, MetS, and fructose consumption among obese individuals with hypertension. The findings of this research reveal a substantial positive correlation between RSF and abdominal VAT area. Additionally, we observed a positive correlation between RSF and both SBP and DBP. However, we did not identify any significant association between RSF and waist circumference or BMI. Furthermore, there was no significant relationship between RSF and fructose intake or MetS. Although a

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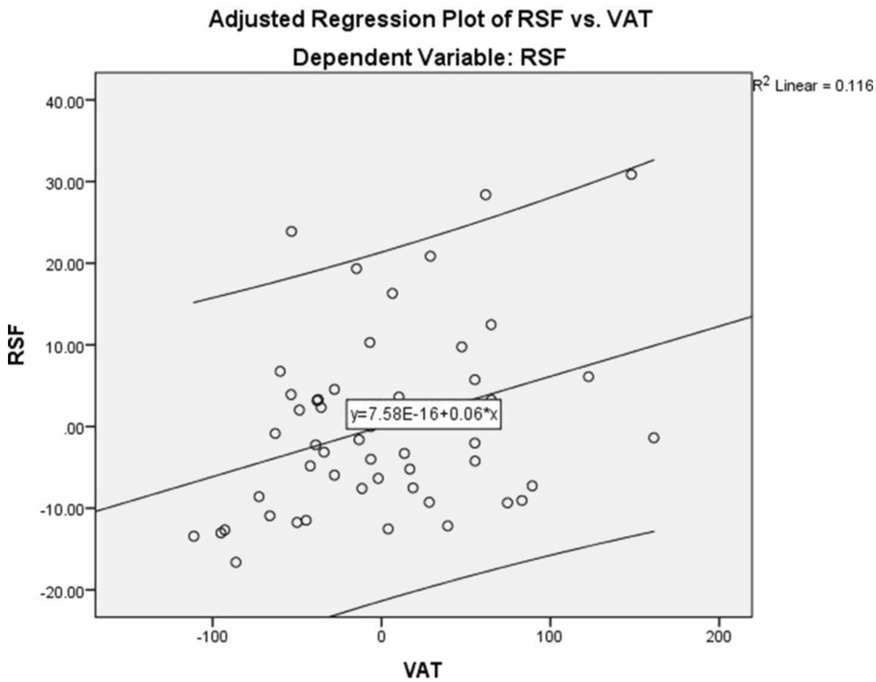

Figure 1. Adjusted regression plot showing the relationship between RSF and VAT, adjusted for waist circumference. The regression line demonstrates a significant positive association.

Table 5. The prediction power of SBP and DBP by RSF based on linear regression analyses

Dependent variable		Unstandardised *B* (95% CI)	*P*-value
SBP	Crude	0.769 (0.407–1.132)	<0.001
	Model 1	0.746 (0.387–1.104)	<0.001
DBP	Crude	0.418 (0.204–0.633)	<0.001
	Model 1	0.391 (0.169–0.614)	0.001

Model 1: Adjusted for age and gender.
SBP, systolic blood pressure; DBP, diastolic blood pressure; RSF, renal sinus fat; CI, confidence interval.

marginally significant positive correlation was found between the volume of RSF and the number of antihypertensive medications taken, no significant correlation was observed between RSF and lipid profile measures, FBS, or daily energy intake.

Obesity is a significant global public health concern and is associated with several chronic illnesses[27]. The prevalence of obesity is increasing worldwide[28], and studies have shown that obesity is directly related to increased blood pressure and the risk of MetS[29,30]. Furthermore, the intake of fructose has been linked to an increased risk of obesity and metabolic diseases[31,32]. Therefore, treatments aimed at reducing the volume of RSF as a major predictor of metabolic diseases and hypertension are crucial for the management of obesity.

4.1. Renal sinus fat and anthropometric indices

The results of this study support the hypothesis that VAT plays a role in the accumulation of RSF. Linear regression analysis showed that VAT was a significant and independent predictor for RSF, even after adjusting for confounding factor. These findings are consistent with previous research demonstrating that following bariatric surgery, RSF was reduced along with other markers of adiposity in obese patients[33]. Additionally, a cohort study found that the accumulation of central fat in healthy overweight and obese individuals is associated with an increase in pararenal, perirenal, and epicardial fat[34]. The results of another study showed that perirenal fat thickness was significantly correlated with metabolic risk factors like BMI and waist circumference[35]. Obesity is characterised by excessive accumulation of adipose tissue, including VAT, which releases various bioactive molecules called adipokines[36,37]. Pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and adiponectin play an important role in the development of chronic low-grade inflammation, which is a hallmark of obesity[38,39]. The chronic inflammatory state created by obesity can lead to the recruitment and activation of immune cells, such as macrophages, in the renal sinus. These activated immune cells release additional pro-inflammatory cytokines, perpetuating the inflammatory response in the renal sinus[40].

4.2. Renal sinus fat and blood pressure

The results of this study suggest that RSF may be associated with blood pressure, as there was a significant correlation between RSF and SBP or DBP. However, there were no significant differences in RSF volume between participants with stage I hypertension and those with stage II hypertension. Additionally, there was a strong trend toward a positive correlation between the number of antihypertensive medications taken and RSF volume, suggesting that an increase in RSF volume may be associated with a higher number of antihypertensive medications. Several studies have demonstrated a significant association between RSF and hypertension, as well as the number of prescribed antihypertensive medications and renal size[13,33]. Furthermore, RSF has been shown to be associated with SBP, DBP, and mean arterial pressure regardless of visceral adiposity and BMI[3,41]. This may be due to

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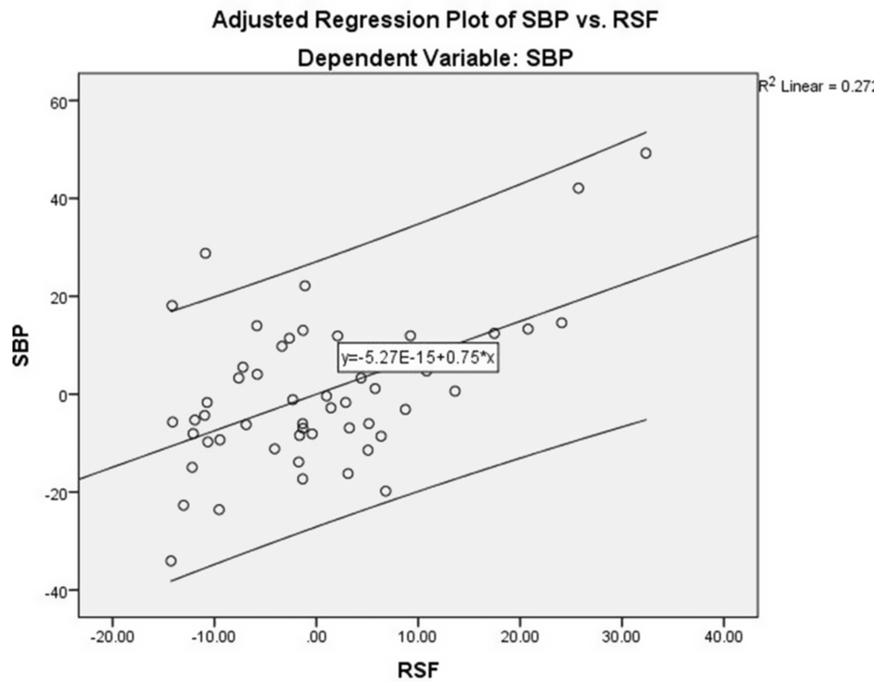

Figure 2. Adjusted regression plot illustrating the independent association between RSF and SBP after controlling for age and gender. The results indicate that RSF is a significant predictor of SBP in hypertensive obese individuals.

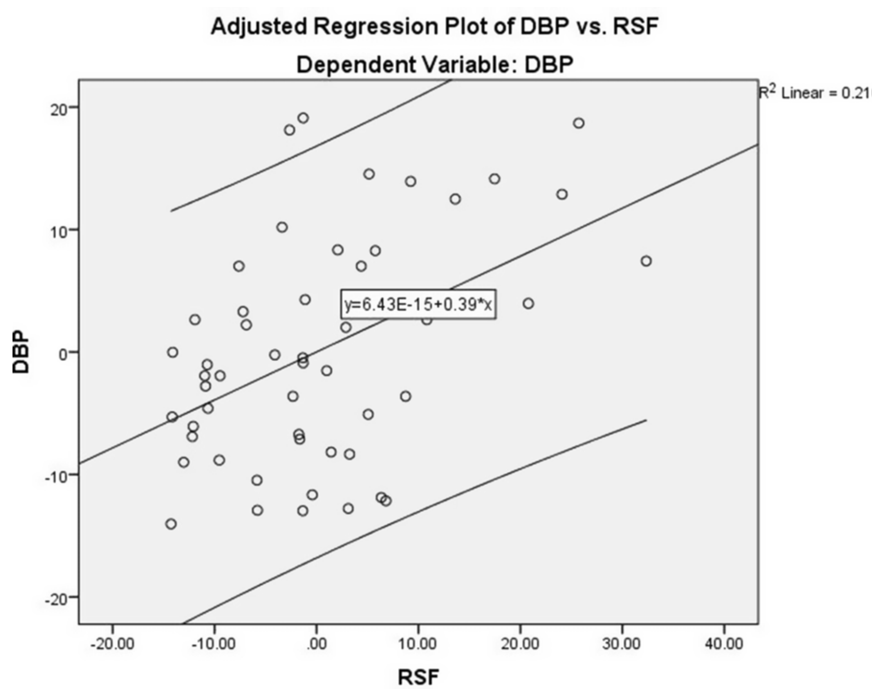

Figure 3. Adjusted regression plot depicting the correlation between RSF and DBP. The relationship is adjusted for relevant confounders, showing a positive association between RSF and DBP.

the compression of renal structures, leading to increased renal interstitial pressure, activation of the RAAS, and retention of sodium(42,43). Consistent with our study, the Framingham Heart Study found a positive association between RSF and hypertension, SBP, and DBP(3). The excessive accumulation of fat in the renal sinus may lead to an increase in intra-abdominal pressure and compression of the low-pressure renal veins, causing changes in kidney function through the activation of the RAAS. Therefore, the expansion of fat in the renal sinus may contribute to the worsening of hypertension and renal dysfunction in individuals with obesity(41,42).

4.3. Renal sinus fat and MetS

Lipid profile measures (TG, TC, HDL-C, LDL-C) and FBS did not show a significant correlation with RSF. Binary logistic regression analysis showed no association between MetS and RSF.

The significance of RSF in examining cardiovascular risk factors in MetS has gained attention. Perivascular adipose tissue plays a crucial role in linking obesity, liver function, insulin resistance, and both macro- and microangiopathy across multiple organs[44,45]. Recent studies on the relationship between RSF and MetS have been challenging and contradictory. Contrary to the findings of the present study, the results of Notohamiprodjo M. et al trial indicate a significant increase in RSF in individuals with prediabetes to healthy subjects and RSF as a PVAT acts as a potential imaging biomarker as an important predictor of metabolic diseases[13]. In another retrospective study, patients with MetS had greater perirenal fat thickness, HOMA-IR, alanine transaminase (ALT), and aspartate transaminase (AST) than those without MetS[46]. But consistent with our findings, the results of another study have shown no association between insulin sensitivity with VAT, intra-hepatic lipid, intra-pancreatic lipid, and intra-myocellular lipids in black West African men[47].

It appears that various factors such as age, sex, and ethnicity are involved in the relationship between RSF and MetS. Recent studies indicate that the quality of adipose tissue in different anatomic regions and the effect of renal sinus adipose tissue quality on renal dysfunction are effective in developing MetS[8]. Although we did not find an association between RSF and MetS, it is possible that different results would have been obtained with a larger sample size. Nevertheless, these findings should be further investigated in a larger population.

4.4. Renal sinus fat and fructose intake

Our study showed that daily fructose intake did not have a significant correlation with RSF. To the best of our knowledge, no study has examined the direct relationship between fructose intake and RSF. Recent studies have focused on the association between fructose intake and lipogenesis, as well as chronic diseases such as diabetes, hypertension, and obesity[16,48]. Fructose is primarily metabolised in the liver, where it undergoes phosphorylation by fructokinase, leading to the formation of fructose-1-phosphate. This process bypasses the main regulatory step of glycolysis, resulting in uncontrolled glycolytic flux. Excessive fructose metabolism leads to increased production of acetyl-CoA, which promotes de novo lipogenesis and TG synthesis[49,50]. Elevated TG levels can subsequently contribute to ectopic fat deposition, including the renal sinus[51].

A clinical trial showed that a 7-day high-fructose diet increased fasting very-low-density lipoprotein (VLDL) triacylglycerols and ectopic lipid deposition in the liver and muscle, and decreased hepatic insulin sensitivity in healthy subjects with a family history of type 2 diabetes[48]. However, in line with our study, the results of Bravo S. et al indicated that normal consumption of fructose as part of a typical diet in commonly consumed sweeteners, such as sucrose or high-fructose corn syrup (HFCS), does not promote ectopic fat storage in the liver or muscles[52].

Although we did not find a correlation between fructose intake and RSF, it is possible that fructose metabolism disrupts lipid homeostasis, leading to ectopic fat deposition within the renal sinus. This could be due to the misclassification of study participants resulting from the use of FFQ. Further studies are needed to explore therapeutic interventions targeting fructose metabolism and lipogenesis to mitigate the detrimental effects on RSF volume and metabolic disorders.

4.5. Strengths and limitations of this study

This study has several strengths worth highlighting. First, we had access to comprehensive information on both dietary and non-dietary factors, which allowed us to control for a broad range of potential confounders and obtain more robust independent associations. Second, the use of validated questionnaires for data collection enhances the reliability and accuracy of our findings. However, there are also several limitations to consider. Firstly, due to the cross-sectional nature of this study, causality cannot be established for the observed associations. Secondly, although we controlled for most lifestyle factors and diet quality, residual or unmeasured confounding may still influence the results. Furthermore, while the sample size was calculated using an appropriate formula, larger sample sizes are needed to confirm these findings. Additionally, as with many studies in nutritional epidemiology, there is a potential for participant misclassification due to the use of FFQ. Lastly, the lack of longitudinal follow-up limits our ability to assess the progression of RSF accumulation over time and its long-term effects on the variables measured.

4.6. Conclusion

Overall, these findings suggest that RSF is positively associated with abdominal VAT area, SBP, DBP, and antihypertensive medication use. However, no significant associations were observed between RSF and other anthropometric, metabolic, or dietary parameters, including MetS. These results highlight the potential of VAT as a contributor to RSF accumulation, emphasising the importance of managing VAT in clinical strategies aimed at reducing RSF and improving blood pressure control. Identifying individuals with excessive VAT could help tailor interventions to limit RSF accumulation and better manage hypertension. Further, longitudinal studies are needed to establish causality and elucidate the underlying mechanisms linking RSF accumulation to metabolic disorders and nutritional status, ultimately guiding more effective prevention and treatment strategies.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/jns.2024.84.

Data availability statement. Data described in the manuscript and analytic code will be made available from the corresponding author upon reasonable request.

Acknowledgements. The research was approved and supported by Kidney Research Center, Tabriz University of Medical Sciences, Tabriz, Iran (approval number: IR.TBZMED.REC.1399.1173).

Authors' contributions. Study design and development of the proposal: PA, MA, AO, and MRA. Study management: PA, MA, and MRA. Study conduct and data collection: PA, MA, and MRA. PA and MNK drafted the manuscript, and MRA and SA revised the paper critically. All authors read and approved the final manuscript.

Funding statement. The present study was funded and supported by Tabriz University of Medical Sciences. The Tabriz University of Medical Sciences had no role in designing the study and collecting, analysing, and interpreting data or in writing the manuscript.

Competing interests. The authors declare that they have no competing interests.

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Journal of Nutritional Science

cambridge.org/jns

Diet diversity score might be associated with reproductive health in women and infant outcomes: a systematic review

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Review

Cite this article: Ahmadi P, Bayat N, and Abbasi B (2025). Diet diversity score might be associated with reproductive health in women and infant outcomes: a systematic review. *Journal of Nutritional Science* **13**: e98, 1–10. doi: 10.1017/jns.2024.81

Received: 2 June 2024
Revised: 7 October 2024
Accepted: 5 November 2024

Keywords:
Anemia; Birth weight; Diet diversity; Pregnancy outcomes; Reproductive health

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Abstract

Lifestyle and diet may affect the reproductive cycle. A dietary index called Diet Diversity Score (DDS) may be related to various reproductive outcomes. The present review aims to look over and conclude the prior studies on the relationship between the diversity of food ingredients and issues related to reproductive health and pregnancy. In the case of this relationship, our findings can increase clinical knowledge and help recommend a well-balanced diet for the target group. A comprehensive search was performed in major databases such as PubMed, Google Scholar, Web of Science, Scopus, and Scientific Information Database until March 2024. This research was combined with a search of Elsevier and SpringerLink databases, which led to the inclusion of relevant articles in this review. Our study was conducted based on 27 articles from 2012 to 2023, all containing a possible link between dietary diversity and reproductive complications. The Newcastle-Ottawa Scale quality assessment was used to evaluate the quality of included studies. Due to our results, a higher score in DDS, which led to an increased intake of major nutrients and a greater variety of foods, was correlated with a lower risk of reproductive health disorders such as polycystic ovary syndrome, maternal anaemia, and maternal bone status, as well as a reduced likelihood of certain birth outcomes, including low-birth weight infants, Apgar score and congenital heart defect. These findings highlight the importance of improving the DDS for maternal and infant health.

Introduction

Reproduction is an essential life cycle phase producing new individuals with a unique genome.[1] Currently, many people are influenced by reproductive problems during their lives. According to the World Health Organization report,[2] around 17.5% of the adult population, approximately 1 in 6 worldwide, experience fertility difficulties, demonstrating that this is a major health challenge.[3] Furthermore, according to Njagi *et al.*, the incidence of infertility among couples of reproductive age ranges from 12.6% to 17.5% globally, with almost higher prevalence rates in some regions, such as the Western Pacific, the Americas, Africa, and Europe.[4] Additionally, people's pockets largely cover fertility treatment costs, and its high financial costs often prevent people from accessing the treatments. For instance, median US costs per IVF cycle or birth were predicted to be about $9,226 and $56,419, respectively, by 2011.[5]

Moreover, according to previous studies, reproduction was related to anaemia, infant birth weight, and quality of diet, such as diet diversity, lifestyle, and physical activity.[6–9]

Diet diversity is defined as the number of food group items such as cereals, vegetables, fruits, legumes, meat/fish/egg, and dairy products consumed over 24 hours.[10] Diet diversity is considered an essential element of a balanced diet and is also suggested to indicate overall diet quality. Due to most dietary guidelines in the US and globally, increasing the variety of foods across food groups is recommended as it is thought to ensure sufficient intake of key nutrients and promote health.[11] According to the Food Guide Pyramid, there are five main groups to score dietary diversity. Each of the five food sets has a maximum diversity score of two out of the ten possible score points. The total food diversity score is the sum of the scores of the five main groups.[12]

Based on the literature, several studies were conducted on the relationship between a diversified diet and reproductive problems despite conflicting results. For instance, Zerfu *et al.* reported that a score of ≥ 4 in dietary diversity food groups during pregnancy was correlated with a lower possibility of maternal anaemia, infants with low-birth weight, and preterm birth, which is explained by the consumption of more nutrient-dense foods such as animal-source foods, fruits, and vegetables, and improved antioxidant and fibre intakes in the adequate DDS group.[13] While Yang *et al.* observed maternal dietary diversity, their associations with inappropriate gestational weight gain and adverse birth outcomes.[14] This inconsistency might be due to differences in study design, participants' socio-economic status, geographic location, and eating habits.[15]

According to our findings, the dietary diversity score and reproduction may probably be related despite the conflicting results. Thus, this systematic review evaluated the articles in this field to determine whether dietary diversity is linked to poor reproductive health and infant outcomes. To the best of our knowledge, this was the first systematic review examining this relationship.

Methods

This systematic review was carried out based on PRISMA guidelines for Systematic Reviews and Meta-Analyses.[16] This protocol was registered in PROSPERO under the registration number CRD42024548428.

Our research was performed using the following major databases: PubMed, Google Scholar, Web of Science, Scopus, and ProQuest. Moreover, publisher databases, mainly Elsevier, Springer, and Wiley, were also reviewed until March 2024 for possible relevant studies. This search was supplemented by covering the references of all articles for any probably related articles.

All relevant articles were retrieved using a combination of Mesh and non-Mesh terms and keywords like: 'Pregnancy', 'Reproduction', 'Infant', 'Maternal Dietary Diversity', 'Pregnancy Complications', 'Reproductive Health', 'Diet Diversity Score', 'Low Birth Weight', and 'Small for Gestational Age', 'Polycystic Ovary Syndrome', 'Gestational Weight Gain', 'Preterm Birth', 'Birth Outcomes', and 'Apgar Score'.

Inclusion and exclusion

After screening the titles and abstracts, all related articles addressing the relationship between diet diversity score and reproductive health or different infant outcomes were included to be assessed in the current study. Furthermore, no language restrictions or restrictions regarding the quality of studies were imposed. No limitation criteria were assigned to the study's publication date or geographic region. In addition, the database search results were classified, duplicate articles were removed using EndNote software, and articles with irrelevant titles and abstracts were excluded. Overall, observational studies were covered, while review articles and experimental and animal studies were ruled out from further assessment.

Quality assessment

To evaluate the methodological quality and bias risk, each included study was checked out using The Newcastle-Ottawa Scale (NOS) quality instrument. The NOS quality assessment is based on a star rating system for each response, allowing a maximum of seven stars for cross-sectional studies and nine stars for case-control and cohort studies. This assessment consists of three main subscales: Selection, Comparability, and Outcome/Exposure.

Two authors approved the evaluation process. The total star points were then converted into percentages, as illustrated in Fig. 1.[17]

Results

The primary search retrieved 278 articles from the databases mentioned above. According to the inclusion criteria, duplication, and abstract screening, 27 articles between 2012 and 2023 were added to the final analysis (Fig. 2). They all assessed the correlation between diet diversity score and reproductive issues.

The cited articles provided evidence from eleven countries, including the United States of America, Spain, Tanzania, Sri Lanka, Iran, Ethiopia, Ghana, China, Cameroon, Pakistan, and India. Also, the population size of these studies ranged between 200 and 7553 women. Regarding the study design, 11 articles had a cohort design, five had a case-control design, and the remaining 11 were cross-sectional studies.

The articles used various methods for assessing dietary diversity scores, such as FAO MDDW (Minimum Dietary Diversity for Women),[18] Women Dietary Diversity Score (WDDS),[19] and the methodology developed by Kant *et al.*.[20] Additionally, Soodi *et al.* utilised the food guide pyramid approach for DDS measurement.[21] Rammohan *et al.* implemented the DDI method to measure DDS.[22] Furthermore, Saaka *et al.* used the Individual Diet Diversity Score (IDDS) method to measure DDS.[23] Zerfu *et al.* also adopted the Women Individual Dietary Diversity (WIDD) method in their DDS measurements.[24]

The articles included were categorised into two main sets: 'Reproductive Health' (Table 1) and 'Infant Outcomes' (Table 2).

Reproductive health

The following group assessed in this study is women's reproductive health, which includes conditions such as anaemia, nutritional and bone status, polycystic ovary syndrome, and metabolic disorders.

Maternal anaemia

Six out of 27 studies analysed the effect of a diversified diet on the prevalence of anaemia, all of which indicated a beneficial impact of higher DDS on maternal anaemia and, thus, a beneficial impact on managing this condition.[13,24,27,31–33]

Nutritional status

In sum, four out of 27 articles examined the ratio of nutrient adequacy in women's diets. Four papers found that a higher score in dietary diversity was correlated with greater nutritional status for women of reproductive age.[25,28,29,34]

Bone status

One out of 27 articles examined the effect of a varied diet on women's bone status. Zhong W. *et al.* reported that a diversified diet was positively associated with better bone status, recommending the importance of modifying diet diversity for pregnant women.[29]

Polycystic ovary syndrome

Of the 27 studies, Soodi *et al.* found a link between diet diversity score and the incidence of polycystic ovary syndrome in women of reproductive age, showing a significant association with higher scores and lower odds of PCOS.[30]

Metabolic disorders

Out of 27 studies, Gicevic S. *et al.* identified the association between diet diversity score and different metabolic disorders. Gicevic S. *et al.* stated that the food-based dietary diversity score did not

Study	NOS Quality Assessment of Observational Studies Based on percentage
Karimi et al. (2022)	55.5%
Gicevic et al. (2018)	77.7%
Jugha, V et al. (2020)	100%
Ali, F et al. (2014)	57.1%
Zerfu, T.A et al. (2016)	77.7%
Zhong, W et al. (2022)	85.7%
Soodi, S et al. (2021)	88.8%
Abriha, A et al. (2014)	100%
Ayensu, J et al. (2020)	85.7%
Cai, J et al. (2023)	55.5%
Yeneabat, T et al. (2019)	85.7%
Zerfu, T.A et al. (2019)	88.8%
Yang, J et al. (2021)	77.7%
Teng, Y et al. (2023)	71.4%
Rammohan, A. et al. (2019)	85.7%
Saaka et al. (2012)	85.7%
Quansah et al. (2019)	71.4%
Madzorera.I et al. (2021)	88.8%
Quansah et al. (2020)	100%
Berhe et al. (2021)	100%
Sharma, S et al. (2021)	66.6%
Sun, C et al. (2023)	88.8%
N. Cano-Ibanez et al. (2020)	88.8%
Wondemagegn et al. (2022)	77.7%
Ahmed et al. (2018)	88.8%
Madzorera.I et al. (2020)	100%
Yang, J et al. (2022)	100%

Figure 1. NOS Quality Assessment of 27 observational studies.

predict gestational diabetes mellitus (GDM) and hypertensive disorders of pregnancy (HDPs).[26]

Infant outcomes

Infant outcomes are the other set, containing articles on subjects such as low-birth weight and preterm birth infants (LBW), Apgar score, small for gestational age foetuses (SGA), and congenital heart defect.

Low birth weight and preterm birth infants

Eleven out of 27 articles expressed the correlation of diet diversity score with low-birth weight and preterm birth infants. The findings indicated that a greater maternal diet diversity was associated with

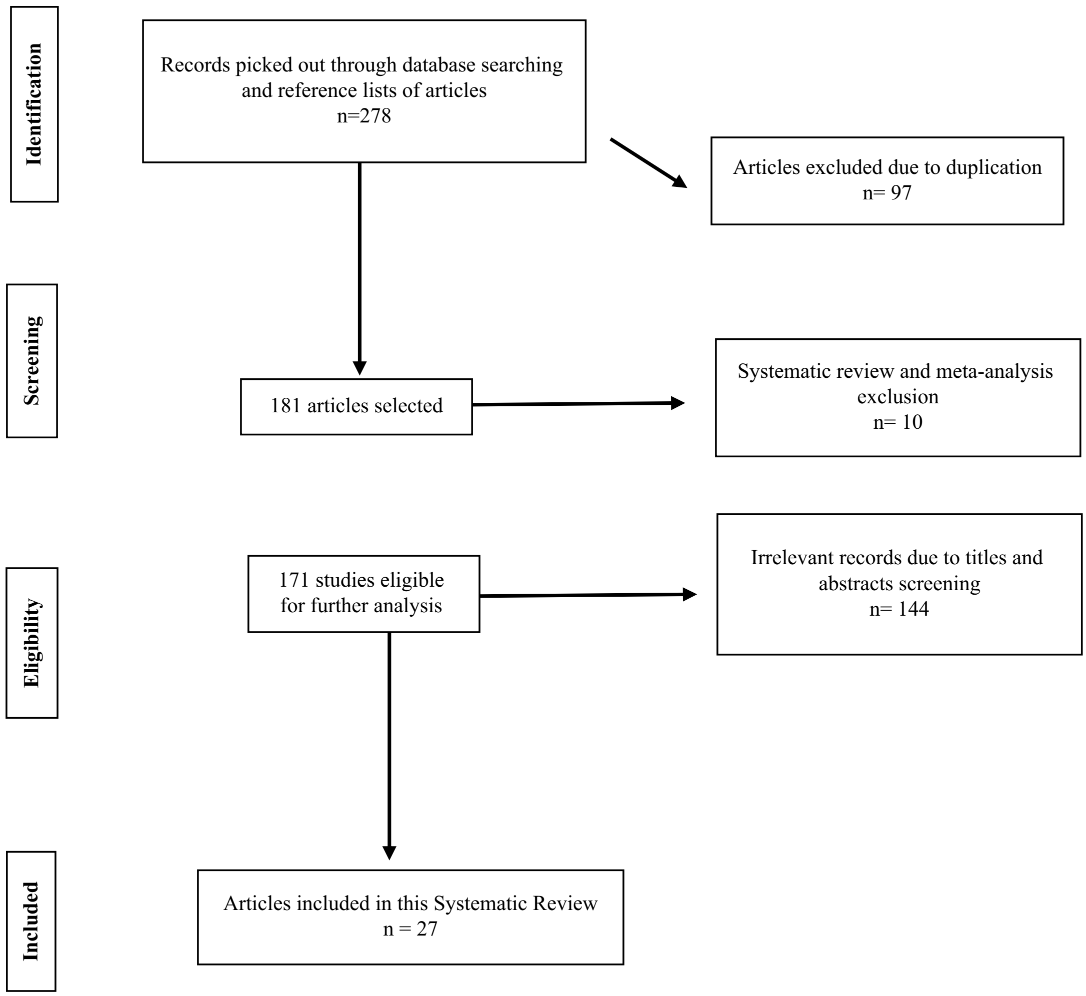

Figure 2. Flow diagram for illustrating the literature search and selection process.

lower rates of low-birth-weight infants, a significant relationship consistently observed across all eleven studies.[14,22,23,36,37,39–42,45,46]

Small for gestational age (SGA)

Two studies evaluated the relationship between small for gestational age infants and diet diversity scores from the 27 articles. These studies found that women with a higher diet diversity score presented a lower SGA risk.[43,44]

Apgar score

Among the 27 papers, Quansah D. Y. *et al.* assessed the correlation between diet diversity and infant Apgar scores. Indeed, they reported a positive impact of increasing diet diversity on the Apgar score. Most mothers who gave birth to babies with a standard Apgar score had a more diversified diet.[38]

Congenital heart defect

Of the total of 27 studies, Yang *et al.* was the only study that examined the effect of diet diversity on the neonatal heart. Thus, it was concluded that an adequate diet diversity score during pregnancy might be related to a reduced chance of congenital heart defects.[35]

Discussion

To the best of our knowledge, this is the first systematic review to investigate the correlation between diet diversity score and reproductive health among women, as well as different infant outcomes, aimed at updating and concluding the prior studies.

The diet diversity score was related to maternal nutritional status. Karimi *et al.* pointed out that women with a higher score in diet diversity had an increased mean adequacy of different food groups and higher intake in all main components of the DDS, such as cereals and bread, meat, dairy products, vegetables, and fruit groups. As a result, they received more diverse foods. These circumstances may be correlated with different birth outcomes, affecting newborns' bioavailability of nutrient resources.[25]

Overview of evidence related to women's reproductive health

In the present study, 12 articles emphasised the significant impact of dietary diversity on women's reproductive health, particularly highlighting the adverse effects of a low-diversity diet.

According to our assessment, a more diversified diet may be related to a lower incidence of maternal anaemia, which aligns with the findings from the six articles on this topic. Specifically, women

Table 1. Summary of twelve selected articles assessing the relationship between diet diversity score and Reproductive Health

Author(s)/ Year	Country	Study Design	Study population and location	DDS Measurement/ Food Groups	Max Number	Mean/ Median DDS	Findings
Karimi, T. *et al.* (2022)[25]	Iran	Cohort	585 pregnant women in Tehran	Kant *et al.* method[a]/five food groups of the pyramid	10	Before pregnancy: 5.31 ± 1.11, During: 5.23 ± 1.42	A higher dietary diversity and nutritional adequacy before and during pregnancy may cause a higher micronutrient intake, leading to better neonatal outcomes.
Gicevic, S. *et al.* (2018)[26]	United States	Cohort	41101 women US nurse without major chronic disease or GDM/ HDPs	MDDW method[b]/10 food groups	10	2	The food-based diet diversity score did not predict GDM or HDPs.
Jugha, V. *et al.* (2020)[27]	Cameroon	Cross-Sectional	1014 pregnant women in the Mount Cameroon area	MDDW method[b]/10 food groups	10	3.57 ± 0.82	A diverse diet rich in micronutrients had a positive effect on haemoglobin levels and will prevent anaemia.
Ali, F. *et al.* (2014)[28]	Pakistan	Cross-Sectional	350 pregnant women at Pakistan Institute of Medical Sciences, Islamabad	MDDW method[b]/10 food groups	10	6.17 ± 0.99	Increasing dietary diversity may lead to weight gain in pregnant women, which is associated with increased food intake and improved nutritional status for both mother and infant.
Zerfu, T. A. *et al.* (2016)[13]	Ethiopia	Cohort	374 pregnant women of Arsi zone, Oromia region, Ethiopia	WDDS method[c]/nine food groups	9	Not Reported	Pregnant women in the inadequate DDS group had a higher risk of anaemia, LBW, and PTB due to lower energy and nutrient intake.
Zhong, W. *et al.* (2022)[29]	China	Cross-Sectional	775 pregnant women aged 18 years old or older in urban China	MDDW method[b]/10 food groups	10	6.61 ± 1.53	A diversified diet, including animal foods and dairy products rich in calcium, vitamin D, and protein were correlated with better bone status.
Soodi, S. *et al.* (2021)[30]	Iran	Case-control	494 participants (203 cases and 291 controls)	Food Guide Pyramid[e]/ five food groups of the pyramid	10	5.19 ± 1.19 for the cases and 5.51 ± 1.19 for the controls	PCOS women had lower consumption of meat and vegetables and higher intake of high-glycemic index foods and sugar-rich foods. An enhanced DDS will reduce the risk of PCOS.
Abriha, A. *et al.* (2014)[31]	Ethiopia	Cross-Sectional	619 pregnant women in Mekele, Northern Ethiopia	Single 24-hour recall/Not reported	High DDS ≥ 6	4.9	Food frequency, dietary diversity, and red meat consumption due to increased haemoglobin concentrations and heme iron levels significantly affect anaemia in pregnant women.
Ayensu, J. *et al.* (2020)[32]	Ghana	Cross-Sectional	379 pregnant women in the Ashanti region	WDDS method[c]/nine food groups	10	3.81 ± 0.7	Dietary diversity positively impacted haemoglobin levels, associated with a lower odd of anaemia.
Cai, Z. *et al.* (2023)[33]	China	Cohort	969 pregnant women from rural townships in Western China	WDDS method[c]/nine food groups	9	6.2 ± 1.39	Improving dietary diversity leads to higher consumption of nutrient-dense foods incredibly bioavailable heme iron, vitamin A, and vitamin C sources, reducing maternal anaemia.
Yeneabat, T. *et al.* (2019)[34]	Ethiopia	Cross-Sectional	834 pregnant women in East Gojjam Zone, Northwest Ethiopia	MDDW method[b]/10 food groups	10	3.68 ± 2.10	The consumption of less diversified food, decreased meal frequency, and food groups consumed by non-educated pregnant women were common.
Zerfu, TA. *et al.* (2019)[24]	Ethiopia	Cohort	432 pregnant women in Arsi Zone, Central Ethiopia	WIDD method[**d**]/nine food groups	9	Not Reported	Low DDS and not consuming animal-source foods, along with pre-existing anaemia, were associated with a risk of anaemia at term.

[a]**Kant *et al.***[20] method: this method includes five food groups such as 1) dairy, 2) meat, 3) grain, 4) fruit, 5) vegetables.
[b]**MDDW**[18] method: Minimum Dietary Diversity for Women Food groups: It includes the 10 following food groups: 1) starchy staples, 2) pulses, 3) nuts and seeds, 4) dairy, 5) meat, poultry, and fish, 6) eggs, 7) dark-green leafy vegetables, 8) other vitamin-A rich fruits and vegetables, 9) other vegetables, and 10) other fruits.
[c]**WDDS**[19] method: Women Dietary Diversity Score Food groups: The foods were categorised into nine food groups: 1) cereals, roots, and tubers; 2) vitamin A– rich fruit and vegetables; 3) other fruit; 4) other vegetables; 5) legumes and nuts; 6) meat, poultry, and fish; 7) fats and oils; 8) dairy; and 9) eggs.
[d]**WIDD**[24] method: Women Individual Dietary Diversity Food groups following nine food groups to calculate the WIDD score: 1) cereals, roots, and tubers; 2) dark-green leafy vegetables; 3) vitamin A-rich fruits and vegetables; 4) other fruit and vegetables; 5) legumes and nuts; 6) meat, poultry, and fish; 7) organ meat; 8) dairy; and 9) eggs.
[e]**Food Guide Pyramid**[21]: 5 main groups, including 1) grains/bread, 2) vegetables, 3) fruits, 4) meats, and 5) dairy products, were used.

Table 2. Summary of fifteen/sixteen selected articles assessing the relationship of dietary diversity with Infant Outcomes

Author (s)/Year	Country	Study Design	Study population and location	Measurement of DDs/Food Groups	Max Number	Mean/Median DDS	Findings
(Karimi, T. *et al.*) (2022)[25]	Iran	Cohort	585 pregnant women in Tehran	Kant *et al.* method[a]/five food groups of the pyramid	10	Before pregnancy: 5.31 ± 1.11, During: 5.23 ± 1.42	A higher dietary diversity and nutritional adequacy before and during pregnancy may cause higher micronutrient intake, leading to better neonatal outcomes.
Yang. J, *et al.* (2021)[35]	China	Case-Control	474 cases and 948 control in Xi'an City, Northwest China	MDDW method[b]/10 food groups	10	5 for the cases and 6 for the controls	Adequate DDS during pregnancy might be associated with a lower risk of CHD in offspring through improved antioxidant status.
Madzorera.I., *et al.* (2020)[36]	Tanzania	Cohort	7553 HIV-negative pregnant women from Tanzania	MDDW method[b]/10 food groups	10	3	A diversified maternal diet rich in energy and protein intake leads to preventing SGA and LBW infants.
Teng. Y, *et al.* (2023)[37]	China	Cross-Sectional	6805 Chinese pregnant women	MDDW method[b]/10 food groups	10	6.11 ± 1.97	Maternal dietary diversity full of animal-based foods and high-quality proteins was positively associated with neonate birth weight.
Yang, J. *et al.* (2022)[14]	Tanzania	Cohort	1190 pregnant women in urban Tanzania	MDDW method[b]/10 food groups	10	4.2 ± 1.9	A poor maternal diet diversity would fail to meet the nutrition required for mother and foetus, thus leading to a higher risk of different birth outcomes.
Rammohan, A, et.al (2019)[22]	India	Cross-Sectional	230 women in Lucknow	DDI method[c]/eight food group	4.063	0 ± 1	Lack of access to food and low diversity in food intake leads to maternal malnutrition and LBW infants.
Saaka (2012)[23]	Ghana	Cross-Sectional	524 pregnant women in Ghana	FAO IDDS method[d]/11 food groups	11	9.1 ± 1.4	Higher maternal dietary diversity results in more significant nutrient transfer across the placenta, leading to a lower risk of LBW infants.
Quansah. *et al.* 2019)[38]	Ghana	Cross-Sectional	420 mothers	MDDW method[b]/10 food groups	10	Not Reported	A diverse diet during pregnancy provides essential nutrients for foetus growth, leading to a higher Apgar score.
Madzorera. I, *et al.* 2021)[39]	Uganda	Cohort	3291 women and infant pairs from Uganda's population	MDDW method[b]/10 food groups	10	During pregnancy: 3.4 At all other times: 3.1	Maternal diet diversity and nutrient deficiencies during pregnancy may predispose the foetus to experience nutrient deficits that can affect its growth.
Quansah. *et al.* 2020)[40]	Ghana	Cross-Sectional	420 mothers	MDDW method[b]/10 food groups	10	Not Reported	Higher dietary diversity provides adequate micronutrients that are important for foetal development and may be protective of LBW.
Berhe *et al.* 2021)[41]	Ethiopia	Cohort	500 pregnant women in Tigray regional state	MDDW method[b]/10 food groups	10	Not Reported	Poor diet diversity may cause multiple micronutrient deficiencies, negatively impacting birth weight.
Sharma S *et al.* 2021)[42]	India	Case-control	157 mothers with and 214 without an LBW child in India.	MDDW method[b]/10 food groups	10	Mothers with LBW child: 2.2 ± 1.3 Mothers without LBW child: 2.7 ± 1.7	A less diverse diet may limit nutrient availability to transfer to the foetus and affect birth outcomes.
Sun, C *et al.* (2023)[43]	China	Cohort	560 mothers in Southwestern China	MDDW method[b]/10 food groups	10	Not Reported	Inadequate diet diversity was associated with SGA infants through disruptions in the functioning of the placenta.

(*Continued*)

https://doi.org/10.1017/jns.2024.81 Published online by Cambridge University Press

Table 2. (*Continued*)

Author (s)/Year	Country	Study Design	Study population and location	Measurement of DDs/Food Groups	Max Number	Mean/Median DDS	Findings
N. Cano-Ibanez *et al.* (2020)[44]	Spain	Case-control	518 cases and controls of pregnant women in Eastern Andalusia	Kant *et al.* method[a]/five food groups of the pyramid	10	Not Reported	High total DDS and dairy products were associated with a lower risk of SGA newborn due to the inclusion of calcium and high biological value proteins.
Wondemagegn *et al.* (2022)[45]	Ethiopia	Cohort	416 pregnant mothers in East Gojjam, Ethiopia	MDDW method[b]/10 food groups	10	4.08 ± 1.64	An elevated score in diet diversity during pregnancy may serve essential nutrients necessary for a newborn's growth and lead to improved birth outcomes.
Ahmed *et al.* 2018))[46]	Ethiopia	Case-control	95 cases and 191 controls in Dessie Town, Northeast Ethiopia	MDDW method[b]/10 food groups	10	Not Reported	Inadequate maternal dietary diversity due to micronutrient deficiencies was a determinant of LBW infants.

[a]**Kant *et al.***[20] method: this method includes five food groups such as 1) dairy, 2) meat, 3) grain, 4) fruit, and 5) vegetables.
[b]**MDDW**[18] method: Minimum Dietary Diversity for Women Food groups: It includes the ten following food groups: 1) starchy staples, 2) pulses, 3) nuts and seeds, 4) dairy, 5) meat, poultry, and fish, 6) eggs, 7) dark-green leafy vegetables, 8) other vitamin-A rich fruits and vegetables, 9) other vegetables, and 10) other fruits.
[c]**DDI**[22]method: Dietary Diversity Index: including eight main groups: 1) grains, 2) pulses, 3) flour types, 4) sweet, 5) egg, 6) milk products, 7) fruits, and 8) vegetables.
[d]**IDDS**[23] method: Individual Dietary Diversity Score: including 11 food groups: flesh meats (i.e. beef, pork, lamb, goat, poultry), fish, eggs, milk and milk products, organ meat (e.g. liver, kidney), legumes, cereals, roots & tubers, dark-green leafy vegetables, vitamin A rich fruits and fats & oils.

who achieve a higher diet diversity score tend to consume more micronutrient-rich foods, such as sources of animal foods, vegetables, and fruits.[47] Conversely, inadequate dietary diversity leads to mineral and vitamin deficiencies, which may affect iron levels and bioavailability.[48] Moreover, meat consumption due to increased haemoglobin concentration and as an essential source of heme iron was another factor that showed a significant relationship with anaemia in pregnant women.[49] Organ meats are an excellent source of iron and vitamin A, while milk and its products also provide substantial amounts of vitamin A. These nutrients are crucial in synthesising red blood cells and haemoglobin.[50] Moreover, meat contains protein with a high biological value, and fruits abundant in ascorbic acid can enhance iron absorption. Likewise, eating less or not eating meat and fruits also leads to inadequate intake of iron, which ultimately causes anaemia.

Furthermore, vegetables provide a valuable source of folic acid, and a lack of folic acid is related to anaemia during pregnancy.[47] Overall, a diverse diet consisting of different food groups rich in micronutrients, particularly iron, vitamin A, vitamin B12, and folic acid, promotes the production of red blood cells and thus prevents anaemia in women of reproductive age.[27] However, dietary practices and cultural taboos may also restrict mothers from consuming available iron-rich foods.[51]

In this review, we investigated the associations between diet diversity score and bone status in women. Our results were aligned with Zhong *et al.*,[29] which confirmed that women with a higher diet diversity score had a higher intake of animal-source foods, such as dairy products, eggs, and flesh foods. These are rich sources of nutrients such as calcium, vitamin D, and protein, which are beneficial for peak bone mass, reduced bone resorption, and enhanced bone mineral density.[52] In addition, as stated by Weaver *et al.*, women with a diversified diet had a higher consumption of fruits, vegetables, pulses, and nuts or seeds, which were rich in unsaturated fatty acids and bioactive complexes such as flavonoids that protect in case of oxidative stress and inflammation.[53]

In agreement with our assessments, Soodi *et al.* also reported that an elevated diet diversity score was significantly related to a lower incidence of polycystic ovary syndrome. Women with PCOS had more consumption of high-glycemic index foods and a lower intake of vegetables and legumes. As Azadbakht *et al.* and Shishehgar *et al.* claimed, vegetable consumption might be associated with a lower risk of expanding abdominal adiposity and obesity, both of which are the major symptoms of PCOS. It should also be noted that consuming vegetables can improve ovulation by increasing insulin sensitivity.[54,55] Furthermore, Hajivandi *et al.* found that adolescent girls with polycystic ovary syndrome had unhealthy eating habits with a low consumption of meat, fish, and seafood and a high intake of fats full of sugar, unhealthy and fatty foods, which, as a result, cause some problems in women with polycystic ovary syndrome and reproductive issues.[56]

Following the results of our study, Liao *et al.* showed that the risk of GDM was inversely associated with fruit intake. This relationship might be explainable by polyphenols and antioxidant complexes found in fruits, such as carotenoids and vitamins E and C. These compounds reduce oxidative stress in cells, interfering with glucose absorption and preventing the development of abnormal glucose tolerance. Moreover, the presence of fibre in fruits and vegetables can delay the absorption of food carbohydrates and avoid a rapid increase in blood sugar.[57] Gicevic *et al.* found that the food-based dietary diversity score did not correlate with GDM or HDPs. The following mechanism can describe this: the DDSs do not consider the specific types of carbohydrates, fats, and forms of animal protein sources in the total diet. Therefore, the inclusion of refined grains, saturated and trans fatty acids, and red and processed meats associated with a higher risk of several different chronic diseases, such as GDM or HDPs, may partly explain the findings from this study.[26]

Overview of evidence related to infant complications

An inappropriate maternal diet with insignificant diversity and quality would not meet the nutritional requirements of the mother and foetus, which could lead to an increased risk of in-pregnancy complications and harmful consequences of childbirth.[35]

In line with our assessments, articles also claimed that maternal nutritional status and micronutrient deficiencies during pregnancy may have severe consequences for the health of the developing foetus, thereby affecting birth size and the availability of nutrients to transfer to the foetus.[58,59]

In close agreement with Quansah *et al.*, our findings indicated that a diversified diet may be correlated with a reduced risk of infants with low-birth weight.[40]

In addition, a low diet diversity score during pregnancy can lead to a lack of essential micronutrients important for foetal growth and development. However, improved micronutrient intake, resulting in greater dietary diversity, can ameliorate infant complications through improved antioxidant and fibre intake.[60] According to Yang *et al.*, the contrasts in maternal diet diversity among mothers with or without low-birth-weight infants, lack of animal protein intake, and inadequate dietary patterns could be the main nutritional complication for Chinese pregnant women.[61]

Moreover, lower DDS may develop anaemia, impairing oxygen delivery to the foetus and hence interfering with normal intrauterine development, possibly leading to low-birth weight.[62]

Consistent with our assessment with Zerfu *et al.*, the findings of this review showed that diet diversity, an indicator of diet quality, was related to preterm birth and birth weight.[13] According to a prospective cohort study involving 66,000 pregnant women in Norway, ‘prudent’ dietary patterns that included fruits, vegetables, whole-grain cereals, fibre-rich bread, oils, and water reported a lower chance of premature delivery.[63]

This current study found that diet diversity score during pregnancy may be positively related to the Apgar score. As Quansah noted, consuming a diverse diet during pregnancy provides essential nutrients for foetal growth and development. Consuming fewer food groups could result in a lack of important nutrients needed during pregnancy for the foetus’s growth and development.[64] Since the Apgar score is related to birth weight, a low-birth-weight newborn probably has higher odds of having a low Apgar score due to poor nutrition throughout gestation.

One of the outcomes investigated in this review was the relationship between DDS and SGA infants. With the same result, Sun *et al.* indicated that having an underweight pre-pregnancy BMI and also an insufficient dietary diversity score increased the mother’s chance of delivering a small for gestational age infant.[43]

Besides, as claimed by Cano-Ibanez *et al.*, diet diversity improves nutrient status and provides benefits not offered by supplementation during pregnancy. This analysis suggests that women with a high total DDS and more intake of dairy items had a protective impact against being small for gestational age..[44] Additionally, the findings of Zerfu *et al.* showed a decreased risk of preterm birth and low-birth weight in pregnant women with a high diversity of dairy products,[65] which are a good source of key nutrients, like calcium and high biological value proteins.[66] These nutrients are related to preventing small for gestational age due to their contribution to foetal growth.

As a result of our review, which is supported by Yang *et al.*,[35] a probable mechanism by which an adequate diet diversity score during pregnancy can decrease congenital heart defect risk would be through improving antioxidant status. Likewise, enhanced dietary diversity has been reported to be correlated with reduced oxidative stress in women. Maternal oxidative stress in pregnancy may affect the normal growth of the foetal cardiovascular system.[35,67,68] These findings highlight the importance of an increase in diet diversity score, which has a practical impact on women’s reproductive health and infant healthiness.

The present study was conducted to conclude previous studies on the relationship between the diversity of food ingredients and reproductive issues, which reported a positive relationship between higher DDS and reduced risk of reproductive health and infant issues. These findings can increase clinical and pre-clinical knowledge to recommend a diversified diet for the target group.

Considering that the present systematic review is a qualitative study, we conducted a review incorporating different study designs, including all observational studies, to draw conclusions and obtain reliable results from the relevant studies. Furthermore, no restrictions were placed on the publication date or language in our study.

Lastly, the articles included used various methodologies reporting DDS; therefore, quantitative analysis regarding different score calculation methods, was not possible.

Conclusion

Due to our findings, a higher dietary diversity score was correlated with a lower probability of reproductive disorders, including maternal anaemia and polycystic ovary syndrome, and a better status in nutrition and bone health. Moreover, a diversified diet may be correlated with a reduced risk of adverse birth outcomes, such as preterm delivery, low-birth weight infants, congenital heart defect, and a higher Apgar score. Likewise, results showed that DDS may not be correlated with GDM and HDPs. However, further studies are required in this area.

Abbreviations

DDS: Diet Diversity Score; **GDM:** Gestational Diabetes Mellitus; **HDPs:** Hypertensive Disorders of Pregnancy; **LBW:** Low Birth Weight; **PCOS:** Polycystic Ovary Syndrome; **SGA:** Small for Gestational Age

Acknowledgements. We appreciate the reviewers dedicating their time and effort to evaluating the manuscript.

Author contributions. P.A. & N.B. proposed ideas and designed the protocol. N.B. was responsible for the planned search and data collection strategy. P.A was responsible for preparing the original draft. P.A, N.B, and B.A were responsible for data screening and interpretation. B.A reviewed the literature and edited the final manuscript. Each of the authors has confirmed the final version to be published and approved the conception of the study.

-Data is available upon reasonable requests from the corresponding author.

Funding. The authors received no financial support for the research.

Competing interests. The authors had no conflict of interest to declare.

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https://doi.org/10.1017/jns.2024.81 Published online by Cambridge University Press

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Journal of Nutritional Science

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Research Article

Cite this article: Hoefnagels FA, Versteegt S, Stafleu A, Huigens MC, and Battjes-Fries MCE (2024). Effectiveness of the 'Weet wat je eet' nutrition education programme in Dutch secondary schools. *Journal of Nutritional Science* **13**: e96, 1–8. doi: 10.1017/jns.2024.80

Received: 22 May 2024
Revised: 15 August 2024
Accepted: 23 October 2024

Keywords:
Adolescence; Effectiveness; Nutrition education; Secondary schools; Students

Abbreviations:
COM-B model, Capability, Opportunity, Motivation and Behaviour model; GDPR, General Data Protection Regulation; IQR, Interquartile Range; SD, Standard Deviation; WHO, World Health Organization; WWJE, 'Weet wat je eet'

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Effectiveness of the 'Weet wat je eet' nutrition education programme in Dutch secondary schools

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Abstract

To assess the effectiveness of the 'Weet wat je eet' ('Know what you eat') school-based nutrition education programme on behavioural determinants and behaviour among students aged 12–15 years. A quasi-experimental study design was used, collecting data at baseline and after implementing the programme in both an intervention and control group (in total 611 students) across the Netherlands. Students from eighteen Dutch secondary education schools completed two consecutive questionnaires, assessing knowledge, self-efficacy, attitude, subjective norm, intention, and behaviours related to healthy, safe, and sustainable nutrition. Multilevel regression analyses were conducted corrected for gender, grade, education level, and school location. The intervention group showed a significant higher increase in self-efficacy, attitude, intention to drink water (all three $P < 0.01$), and a significant higher decrease in the consumption of sugary drinks, snacks, and meat (all $P < 0.05$) than the control group. Both the groups scored significantly higher on knowledge during the post-test (both $P < 0.05$), although the intervention group not significantly higher than the control group ($P = 0.14$). No significant effects were observed for subjective norm, intention, and fruit, vegetable, and whole grain bread consumption. The results of this study showed positive effects of the 'Weet wat je eet' school-based nutrition education programme on self-efficacy and attitude towards healthy, safe and sustainable nutrition, intention to drink more water, and various healthy eating behaviours among secondary school students. Further research is necessary to assess the long-term sustainability of these results.

Introduction

For the promotion of good health and a sustainable environment, it is crucial for young people to adopt a healthy, safe, and sustainable eating pattern.[1,2] Optimal growth, development, and health during childhood and adolescence necessitate a healthy dietary pattern.[3] Research has demonstrated that a healthy diet enhances children's cognitive skills such as concentration and memory, improves mood and energy levels, and boosts academic performance.[4] Moreover, a healthy eating pattern can reduce the burden of non-communicable diseases.[3,5] Besides, shifting towards a more plant-based and less animal-based eating pattern is not only beneficial for health but also for the environment.[6] Raising awareness about sustainability and food safety, including aspects like food origin, food waste, and food hygiene, is vital for imparting knowledge to young people, enabling them to make healthy, safe, and sustainable food choices in the future. Nutrition behaviour established during childhood often persist into later stages of life.[7] Adolescence is an important period of mental, physical, and social development. Nutrition behaviour, as well as other health-related habits, developed during adolescence tend to persist into later stages of life, making the teenage years an important time to encourage healthy and sustainable eating behaviours.[7,8]

Schools provide an ideal environment for educating children and adolescents about healthy, safe, and sustainable nutrition due to their existing infrastructure. Students from diverse backgrounds spend a significant amount of time in schools each week, acquiring knowledge, skills, and adopting favourable behaviours can be of benefit later in life.[9,10] The school environment has been identified by the WHO as an ideal setting in which youth consume approximately one-third to one-half of their daily food intake.[11] In the Netherlands, the proportion of the daily food intake consumed at school compared to total food consumption in grams is, with approximately 15%, lower.[12] Contento defined nutrition education as 'any combination of educational strategies, accompanied by environmental supports, designed to facilitate voluntary adoption of food choices and other food and nutrition-related behaviours conducive to health and well-being'.[13] For primary education, numerous nutrition education programmes are available, of which the effectiveness have been proven.[13–16] Presently, there exists limited empirical evidence regarding the efficacy of nutritional education interventions targeting adolescents in general and within the context of Dutch secondary education.[17,18] However, little is known yet about the effectiveness of nutrition education among adolescents in Dutch secondary schools.

'Weet wat je eet' (it could be translated as 'Know what you eat', but this doesn't capture the wordplay in the original Dutch title) is a school-based nutrition education programme with the objective to empower young people to make healthy, safe, and sustainable food choices and foster a positive attitude towards these choices. The programme is developed by the Netherlands Nutrition Centre, aimed at the lower grades of secondary school education and secondary vocational education. In the Netherlands, secondary education is divided in different levels: university preparatory education, senior general secondary education and preparatory secondary vocational education. It is an online programme, consisting of six lessons that teach students about healthy, safe, and sustainable nutrition. The lessons cover various key topics, for example, the functions of different nutrients, making informed choices based on food labels, the importance of consuming food groups such as vegetables, and safe food handling and preparation practices to prevent foodborne illnesses. Additionally, the programme addresses the environmental impact of food choices, reducing food waste and understanding the environmental footprint of different foods. The intervention is delivered through a digital learning environment where students can engage independently with the material at their own pace on their own devices, such as a laptops, iPads, or smartphones. The digital learning environment includes interactive modules, videos, and quizzes. Practical assignments, such as meal planning and cooking exercises, allow students to apply theoretical knowledge in real-life scenarios. Personalised feedback is provided based on students' responses in the quizzes, addressing their questions and tailoring the programme to their needs. According to the Netherlands Nutrition Centre, more than 15,000 students followed the recently revised programme in 2022, which is freely available at wwje.nl.[19]

The focus of this study is to assess the effectiveness of the 'Weet wat je eet' programme of the Netherlands Nutrition Centre, specifically targeting students in all levels of secondary education in the lower grades and thereby contributing to insights in the effectiveness of nutrition education for adolescents in secondary education in general.

Methods

Study design

In a quasi-experimental study design, data on behavioural determinants and behaviour towards healthy, safe, and sustainable nutrition were collected, including a pre-test and post-test in an intervention group and control group. The pre- and post-test in the intervention group were conducted using digital questionnaires among secondary school students at baseline and after implementing the lessons from the programme in the autumn of 2022. This intervention group was compared to a control group consisting of similar secondary schools who also completed a digital questionnaire during both a pre-test and a post-test, but that did not implement the lessons during the same period.

Participants

Schools that had previously implemented the programme were contacted to participate in the study by the Netherlands Nutrition Centre. Additionally, schools were recruited through recruitment texts via LinkedIn, various newsletters, and within the digital 'Weet wat je eet' environment. Schools were also informed about this study at the conference of the Netherlands Institute for Biology. Interested teachers had the opportunity to sign up for participation by completing a contact form.

The following inclusion criteria were applied for participation in the study: students from the first and second grades of all levels of secondary education, who had not previously followed the programme. Prior to participating in the study, students were asked to provide informed consent. The goal was to recruit approximately 400 students from ten schools per condition (intervention or control group), based on the power calculation for the effect studies on the 'Taste Lessons' educational programme in primary education on behavioural determinants.[20] Out of the 23 schools that initially expressed interest in participating, a total of 18 schools actively took part in the study. Seven schools chose to implement the programme, while 11 were part of the control group. The remaining five schools withdrew from participation due to not meeting the inclusion criteria, for example, different school type.

Procedures

The teachers from participating schools received two personalised links to the digital questionnaires, one for the pre-test and one for the post-test, intended for all students in their class(es) to be filled out during school time. These links were distributed to the students using the teachers' preferred method, such as sharing them on the student portal or any other convenient means. The pre-test questionnaire was administered to both the intervention and control group before the students in the intervention group started with the first lesson of the programme, between September and November 2022. The post-test questionnaire was administered to the intervention group after they completed the lessons and to the control group during the same period, within a timeframe of three to seven weeks, from November 2022 to January 2023. Participants spent an average of 9 minutes completing the questionnaire. The students engaged in an average of 5.0 lessons out of the 6 lessons offered in the educational programme (SD: 1.60).

Participation in the research and the implementation of the programme were both voluntary. Students were not obliged to complete the questionnaires if they chose not to, they had the freedom to withdraw from the study at any time without having to explain why. All data collected were treated as confidential and processed in a coded manner to ensure anonymity and privacy. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. The medical ethical review committee of VU Amsterdam University exempted this study from ethical approval (reference: 2022.0305). As a token of appreciation for their participation, all classes that completed both questionnaires received a classroom flowerpot and they had the chance to win a food truck for one day.

Outcome measures

The outcome measures were selected and the questionnaires were designed based on the COM-B model. The COM-B model demonstrates that human behaviour (B) results from the interaction between personal physical and psychological capabilities (C), to utilise social and environmental opportunities (O) via motivators (M) that are reflective (thinking with the head) or automatic (emotional-'thinking' with the heart).[21] In the

Table 1. Content of the questionnaires, answer scales and number of items

Determinant	Content of the questions	Example questions	Answer scale	Items
Demographic characteristics	Name, age, gender, grade, and education level	What is your gender?	Different answer options	5
Capacity				
Knowledge	Questions about healthy, safe, and sustainable nutrition	The temperature inside the fridge varies. Where in the refrigerator is it recommended to store dairy items such as milk, yogurt, and cream?	Multiple-choice with 4 options	9
Self-efficacy	I can . . .	I can provide examples of how influencers promote (un)healthy products.	Disagree (=1) to agree (=5)	6
Opportunity				
Subjective norm	My friends consider . . . to be . . .	My friends consider animal welfare to be . . .	Unimportant (=1) to important (=7)	4
Motivation				
Attitude	I consider . . . to be . . .	I consider healthy eating to be . . .	Unimportant (=1) to important (=7)	4
Intention	I plan to . . . in the upcoming month	I intend to drink more water in the upcoming month.	Disagree (=1) to agree (=5)	7
Behaviour				
Behaviour	I generally eat/drink . . .	I generally drink sugary beverages *(like cola, orange soda, and fruit juices)*	Times per week, per day or how often	6

questionnaire, each component of the COM-B model was aligned with the intervention. In the context of the intervention, capabilities were operationalised as knowledge and self-efficacy. For knowledge this included for example nutritional basics, food label interpretation, and safe food handling. Self-efficacy was assessed through questions on students' understanding and confidence in making informed food choices. Opportunities were operationalised as subjective norms through questions about students' perceptions of their social groups. Motivation was operationalised via attitudes and intentions, by assessing students' attitudes towards healthy, safe, and sustainable eating, and their intentions to adopt specific behaviours covered in the lesson content. The operationalisation of the COM-B model is also been used in other studies.[22] The interplay between these components is crucial for behaviour. By enhancing capabilities, providing opportunities and influencing motivators, the model creates a comprehensive approach to measure behaviour. The target behaviour was operationalised as several healthy, safe, and sustainable food behaviours, also as covered in the lesson content. The physical capabilities, for example, age and attendance, and environmental opportunities, for example, availability of the educational programme, were fixed and thus outside the scope of this research. The COM-B model has been successfully applied to understand children's health behaviours within the domains of medical and education science,[23] as well as in designing and evaluating interventions aimed at improving children's physical activity levels.[24]

To assess all outcome measures and the respondents' demographic characteristics, four to nine items per determinant and one to three questions per topic (healthy, safe, and sustainable) were included in the questionnaire (Table 1 for example questions). For the intervention group in the post-test questionnaire, supplementary questions were included to gather feedback on the programme, with an overall evaluation on a scale of 1–10 and specific parts of the programme on a scale ranging from low (=1) to high (=5). In the questions the same terminology was used as in the lessons, to ensure consistency. Furthermore, the questions' readability was examined against B1-level standard and discussed with teachers. To ensure usability and comprehensibility, a group of young individuals tested the questionnaire and subsequently the questions were adapted accordingly.

Statistical analyses

IBM SPSS version 26 was used for the analyses. Normally distributed continuous variables are reported as mean ± standard deviation while categorical variables are presented as n (number) and percentage. Non-normally distributed continuous variables are presented as median and interquartile range.

Responses of the pre- and post-test questionnaires were linked to each other based on the first name and class of the students. Out of the initial 1,072 responses on the pre-test questionnaire and 946 from the post-test questionnaire across eighteen participating schools, 611 students (57% of the pre-test responses) were successfully linked based on their first name and class, and these students answered at least all knowledge questions, which constituted the first substantive questions in the questionnaire. Of the students who answered all knowledge questions, 96% also answered the last question of the questionnaire.

The average scores for all determinants were calculated considering questions within a determinant only if their Cronbach's alpha was at least 0.7. The Cronbach's alpha for the determinants ranged from 0.71 to 0.84, indicating satisfactory internal consistency. The difficulty level of the knowledge questions was assessed using the facility index. Questions in the pre-test that were answered correctly by more than 80% of the students (considered too easy) or less than 20% of the students (considered too difficult) were excluded from further analyses.[25]

Table 2. Characteristics of the study sample (N = 611)

		Intervention group (n = 364)		Control group (n = 247)	
		Number	%	Number	%
Gender	*Boy*	178	49%	139	56%
	Girl	182	50%	106	43%
	Other	4	1%	2	1%
Age (mean ± SD)*		12.4 ± 0.74		12.9 ± 0.91	
Education level*	*Pre-vocational (general) secondary education*	218	60%	111	45%
	Senior general secondary and pre-university education	146	40%	136	55%
Grade*	*First*	218	60%	78	32%
	Second	146	40%	169	68%
School location*	*(Medium) large city*	151	42%	173	70%
	Town/small city	213	59%	74	30%

*Significant difference between the intervention group and control group, P < 0.01.

One knowledge question had a correct response rate exceeding 80% in the pre-test (specifically, the question 'what is a healthy food switch?' with 89% correct answers), and therefore, this question was excluded in the subsequent analyses.

Paired *t*-tests were used to compare pre- and post-test scores separately for the intervention and control groups. Independent *t*-tests were employed to assess differences between the control and intervention group at each measurement point. The change in the average score for each determinant was calculated by subtracting the pre-test score from the post-test score for each student. These change scores were used as outcome measures in the further analyses.

To account for the clustering effect of students within the same school, multilevel linear regression analyses were conducted. Within these analyses, initial analyses were performed to identify confounders and effect modifiers by sequentially adding individual socio-demographic characteristics to the baseline model with the change score of each determinant as the dependent variable and 'condition' (intervention or control group) as independent variable. The following student-level characteristics were included: gender (boy and girl), education level (vocational level and high school/university level), and grade (first and second). The school location serves as school-level characteristic, determined by the population figures of the municipality where the schools are situated. Based on this criterion, they classified into two categories: (medium) large cities (>40,000 inhabitants) or town/small cities (≤ 40,000 inhabitants).[26] Gender, education level, grade, and school location were found to be significant confounders for most determinants and behaviours. None of the characteristics were identified as significant effect modifiers, indicating that the effect of the programme did not differ for the determinants among subgroups defined by the measured characteristics. Subsequently, multivariate multilevel linear regression analyses were conducted, incorporating the change scores as dependent variables and including condition, grade, gender, education level, and school location as independent variables. Afterwards, the results of students who completed all (six) programme lessons were compared to those who completed only some. Results with a P-value <0.05 were interpreted as statistically significant.

Results

Characteristics of the study sample

The study sample included 611 students whose data were included in the analyses. The intervention group consisted of 364 respondents from eleven schools, while the control group consisted of 247 respondents from seven schools. The students had an average age of 12 ± 0.74 years, and the study sample displays a more or less balanced distribution across gender and grade (Table 2). The age of students and the percentage of second-year students in the intervention group were significantly lower compared to the control group (both P < 0.01). Furthermore, the intervention group had a significantly higher proportion of students at pre-vocational level (P < 0.01) and a higher number of students attending schools located in town/small cities compared to the control group (P < 0.01).

Appreciation of the programme

According to the responses from the students in the intervention group, the overall rating for the programme was 6.9 ± 1.7 on a scale of 1–10. On average, students gave neutral to positive scores for the lessons and components of the programme (3.4 ± 1.0). The questions and videos (3.3 ± 1.0) were rated slightly more positive on a scale of 1–5 than the lessons (3.1 ± 0.7) and assignments (3.2 ± 0.8).

Effect on behavioural determinants and behaviour

Capacity

In the pre-test, the students answered on average 43% of the knowledge questions correctly (Table 3). Most students (57%) answered the question regarding which product contains most dietary fibres correctly, while fewest students (29%) answered the question about the best choice of meat considering the environment correctly. Results showed a significant increase in knowledge between the pre-test and post-test in both the intervention and control group (both P < 0.01). The increase in knowledge among students in the intervention group (3.8 ± 1.6 vs.

Table 3. The average scores, the average change in scores of the intervention and control groups, and the results of the multilevel regression analyses

		Mean scores				Change scores		Difference in change	
		Pre-test		Post-test		Post-test – pre-test		Post-test – pre-test	
		Mean	(SD)	Mean	(SD)	Mean	(SD)	β	(SD)
Capacity									
Knowledge	Intervention group	3.81	(1.60)	4.34	(1.54)	0.35	(1.78)	0.00	(0.16)
	Control group	3.79	(1.47)	4.37	(1.60)	0.32	(1.67)		
Self-efficacy	Intervention group	3.34	(0.64)	3.63	(0.64)	0.29	(0.67)	**0.20**	**(0.07)****
	Control group	3.45	(0.74)	3.51	(0.75)	0.05	(0.80)		
Opportunity									
Subjective norm	Intervention group	5.05	(1.25)	5.07	(1.22)	0.02	(1.46)	0.10	(0.13)
	Control group	4.85	(1.23)	4.89	(1.34)	0.04	(1.33)		
Motivation									
Attitude	Intervention group	5.29	(1.09)	5.32	(1.06)	0.03	(1.09)		
								0.29	**(0.10)****
	Control group	5.32	(1.12)	5.21	(1.14)	−0.10	(1.09)		
Intention	Intervention group	3.30	(0.76)	3.34	(0.77)	0.04	(0.84)	0.11	(0.07)
	Control group	3.25	(0.76)	3.21	(0.78)	−0.05	(0.73)		
Behaviour[a]									
Fruit	Intervention group	5	(4)	5	(4)	0.13	(1.82)	0.31	(0.17)
	Control group	5	(4)	5	(4)	−0.15	(1.70)		
Vegetables	Intervention group	6	(2)	6	(2)	0.08	(1.52)	0.08	(0.13)
	Control group	7	(2)	6	(2)	−0.11	(1.25)		
Sugary drinks	Intervention group	3	(4)	3	(3)	−0.48	(2.10)	**−0.37**	**(0.19)***
	Control group	3	(5)	3	(3)	−0.15	(2.06)		
Whole grain bread	Intervention group	2	(2)	2	(2)	−0.13	(1.19)	0.01	(0.11)
	Control group	2.5	(2)	2	(2)	−0.13	(1.20)		
Sweet and savoury snacks	Intervention group	4	(1)	3	(1)	−0.21	(1.14)	**−0.29**	**(0.10)***
	Control group	3	(1)	3	(1)	−0.01	(1.05)		
Meat	Intervention group	5	(3)	5	(4)	−0.33	(1.87)	**−0.34**	**(0.16)***
	Control group	5	(4)	5	(4)	0.02	(1.66)		

The multilevel analyses are corrected for grade, gender, education level, and school location of the students.
[a]Data were not normally distributed, thus the median + interquartile range (IQR) is presented. The change scores were normally distributed.
*$P < 0.05$.
**$P < 0.01$.
Intervention group: n = 347–364; control group: n = 231–247.

4.3 ± 1.5) was not significantly higher than that among students in the control group (3.8 ± 1.5 vs. 4.3 ± 1.6) ($P = 0.14$).

Regarding self-efficacy, students rated themselves slightly positive during the pre-test (3.4 ± 0.7 on a scale of 1–5). Only the intervention group showed a significant increase in self-efficacy ($P < 0.01$), and this increase was significantly higher than in the control group ($P < 0.01$).

Opportunity

During the pre-test, students had a positive average score for subjective norm (5.30 ± 1.24 on a scale of 1–7). There was no significant difference observed between the pre-test and post-test in both the intervention and control group and in the change between the intervention and control group ($P = 0.44$).

Motivation

During the pre-test, students had on average a positive attitude towards healthy, safe, and sustainable food choices (5.30 ± 1.10 on a scale of 1–7). Although there was no significant difference between the pre-test and post-test scores in both the intervention and control group, the change in the direction of a more positive attitude among students in the intervention group was significantly higher than in the control group ($P < 0.01$).

Regarding intention, the students had a slightly positive average score in the pre-test (3.28 ± 0.76 on scale of 1–5). Students scored their intention to eat less meat neutrally to slightly negatively (2.47 ± 1.24), while the intention to drink more water scored the highest (3.92 ± 0.98) of all intention items. There was a significant decrease in the intention to drink more water observed between the pre-test and post-test in the control group ($P < 0.01$), while the intention to drink more water did not change in the intervention group ($P = 0.52$), resulting in a significant positive difference in the intervention group compared to the control group ($P = 0.04$). However, there was no significant difference in the average intention to change other behaviours ($P = 0.16$).

Behaviour

During the pre-test, students most often reported eating fruit and meat five days a week (both 5 ± 4 (median and IQR)) and vegetables six days a week (6 ± 2). Students most often consumed sugar-sweetened beverages three days a week (3 ± 4). Most students reported consuming sweet or savoury snacks almost every day of the week (3 ± 2) and whole grain bread was most usually chosen (3 ± 1). In the intervention group, significant positive differences were observed on various behaviours compared to the control group, including consuming fewer sugar-sweetened beverages ($P < 0.05$), eating fewer sweet and savoury snacks ($P < 0.01$), and consuming less meat ($P = 0.03$).

When comparing only the students in the intervention group who completed all six lessons (n = 196) with the control group, the same results were observed for all outcome measures as when including all students from the intervention group (n = 364) in the analyses.

Discussion

The results of the study indicate that students who participated in the ‘Weet wat je eet’ programme showed significant greater improvements in various behavioural determinants and behaviours related to healthy, safe, and sustainable nutrition compared to the control group. Specifically, the intervention group demonstrated a significant higher increase in self-efficacy, attitude, and intention to drink water, as well as in reducing consumption of sugary drinks, snacks, and meat than the control group. Both the intervention and control group showed increased knowledge scores at the post-test compared to the pre-test, but the intervention group did not show a statistically significant higher increase than the control group. There were no significant changes observed in subjective norms, intention, and behaviour related to consuming fruits, vegetables, and whole-grain bread between the intervention and control group. Although some positive outcomes were observed, other outcomes thus did not show significant changes. This mixed pattern of results suggests that the intervention may have limited overall efficacy and highlights the complexity of evaluating the effectiveness of educational interventions.

Reflection on the results

The ‘Weet wat je eet’ programme aims to empower students to make healthy, safe, and sustainable food choices and foster a positive attitude towards this. The objectives of the programme have been partially achieved, as positive effects have been observed in terms of motivation (attitude and intention to drink more water) and partially on capacity (self-efficacy and knowlegde) and changes in eating behaviour aligned with the food-based dietary guidelines of the Dutch Wheel of Five (Schijf van Vijf) such as reducing consumption of sugary drinks, snacks, and meat.[27] However, there is no observed effect on opportunity (subjective norm).

It is worth noting that this study did not reveal a significant effect on knowledge between the intervention and control group. This result was surprising given that schools are generally considered environments where knowledge is typically imparted, and previous nutrition education programmes have demonstrated increases in knowledge. This raises questions about the potential role of the assessment questionnaires themselves. It is possible that the act of completing these questionnaires increased awareness and prompted behavioural changes independent of the intervention content. Future studies should consider including additional control measures to isolate the effects of the intervention from the assessment process. Other Dutch programmes such as ‘Taste Lessons’[14,20] and foreign programmes described in the systematic reviews by Murimi *et al.* and Shepherd *et al.* have reported a positive effect on knowledge.[18,28] A possible explanation is that there is already increasing attention to healthy and sustainable food in secondary education in the Netherlands. It is plausible that the heightened focus on nutrition and lifestyle in recent times, as well as the inclusion of related topics in other subjects such as biology lessons, have contributed to the overall increase in knowledge levels observed in both groups. Therefore, the content of this programme may perhaps not have provided sufficiently distinct knowledge compared to what students already receive through other methods.

Literature shows that effective nutrition education should not solely focus on knowledge transfer but also focus on teaching skills, changing behaviour, and applying information in practice.[29,30] In this study, although no significant effect on knowledge was found, positive effects were observed on students’ self-efficacy and motivation towards favourable eating behaviour. These findings align with the systematic review by Shepherd *et al.*, which reported that six out of seven interventions targeting attitudes towards healthy eating were effective.[28]

It is well established that adolescence is a crucial period in the development of nutrition behaviour among young people. This period is marked by increased independence, as young people start making their own food choices and are influenced by their friends and peers.[31]

According to Bruening *et al.*, large-scale research illustrates that eating patterns among adolescent friendship groups show similar eating patterns.[32] This phenomenon may stem from young consumers’ desire to conform to social norms by mirroring their peer group’s eating behaviours. In the present study, no effect was observed regarding the opinions of friends of the students, which is in line with the lack of substantial change in subjective norm in other studies on nutrition education programmes fostering healthy eating among adolescents.[33,34] Further research in this area could delve into exploring how to change the social and physical environment on food choices among young people.

A significant positive effect on the intention to drink water was found, which seems to be explained by a decrease in intention among the control group rather than an increase in the intervention group. No significant effect was found on other items of intention, which is in line with the results of another study on the effect of nutrition education on healthy eating behaviour intention.[33] Despite the lack of effect on intention, which is seen as the most prominent predictor of healthy eating behaviour in adolescents,[35] significant changes were observed in certain behaviours among students in the intervention group. Specifically, students in the intervention group reported a significant positive change in the consumption of sugary drinks, snacking, and meat consumption. No significant changes were found in the consumption of fruits, vegetables, and whole grain bread consumption. Research on another Dutch nutrition education programme 'Krachtvoer' ('Power Food'), specifically targeting vocational education students, showed a significant increase in the consumption of vegetables and fruits, while no difference was observed in the consumption of fruit juices and high-fat snacks.[36] This appears to be almost the opposite of the current findings. Studies conducted in Europe examining the effects of nutrition education programmes have reported mixed results in terms of vegetable and fruit consumption.[37,38] According to a systematic review by Vézina-Im and colleagues focussing on soda consumption, which included 36 interventions, 65% of the education-based interventions led to a significant reduction in soda consumption among students.[39] The results from previous studies, therefore, appear to be inconsistent. The effects on snacking behaviour and meat consumption in the current study have not been demonstrated before, to the best of our knowledge. This suggests the need for further research to explore the effect of nutrition education interventions on eating behaviour of adolescents.

Limitations

To ensure a proper interpretation of the results, it is important to consider several limitations. The data obtained in this study relied on self-reported information assessed through (online) questionnaires, which may potentially be subject to social desirability biases. Additionally, it is unclear whether the students were capable of accurately translating their opinions into the appropriate questionnaire responses, and whether the questionnaire used the most suitable questions and response categories. However, efforts were made to address these concerns during questionnaire development and testing, such as controlling the terminology at a suitable level and involving students and a teacher in the pilot testing. Although the questionnaire was carefully developed and pre-tested, not all students completed it in its entirety. Analysis revealed a higher number (4%) of unanswered questions on the last pages of the questionnaire.

The decision was made to include in the analysis only those students who used the same first name in both the pre-test and post-test and were in the same class. As a consequence, this led to the exclusion of a substantial portion (43%) of responses from the pre-test in the analysis. Possible reason for the drop-out include the effort to minimise the collection of personal data to protect student privacy, which may have led to a lack of follow-up. A baseline comparison on characteristics and the outcome measures between completers and non-completers showed no significant differences, suggesting that the drop-out did not introduce bias in the sample. It is conceivable that this exclusion resulted in a selective group of students who may have filled out the questionnaire twice, or who might have been easily distracted, had difficulty completing questionnaires, or lacked the motivation to complete the questionnaire seriously. This may have led to the absence or underrepresentation of a selective group of students in the results, potentially affecting the generalisability of the findings. Since the dropout of participants occurred in both the intervention and control groups, it is not expected to have influenced the results. However, the dropout of participants may have resulted in lower statistical power, potentially leading to the failure to detect true effects.

In this study, multiple statistical tests were conducted to examine various outcomes related to the intervention. This increases the risk of Type I errors. To address this, the use of Bonferroni correction and other statistical adjustments was considered. However, it is also recognised that such adjustments can elevate the risk of Type II errors, potentially obscuring meaningful findings. Therefore, a balanced approach was taken in interpreting the results, considering both statistical significance and practical relevance.

A quasi-experimental research design was used. The inclusion of a control group and the use of a pre-test provide some confidence in attributing the observed results to the programme. However, the allocation of schools to the intervention or control group was not random but based on their voluntary participation in implementing the 'Weet wat je eet' programme. In this study, the intervention and control group differed in terms of socio-demographic characteristics. In the Netherlands, the distribution of students in the first and second grade is approximately fifty-fifty, with slightly more boys than girls in the lower years.[26] The distribution of grade and gender in both the intervention and control groups in the current study deviated from this general distribution. The analyses on change scores between the intervention and control group were adjusted for these differences, including grade, gender, education level, and school location. Nevertheless, differences between the groups may have influenced the results. For future research, it is recommended to ensure a comparable distribution of characteristics to enhance the reliability and generalisability of the results.

Conclusions

The findings of this study indicate that the 'Weet wat je eet' school-based nutrition education programme is effective in enhancing self-efficacy and attitude towards healthy, safe, and sustainable nutrition, as well as the intention to increase water consumption and certain self-reported behaviours among secondary school students aged 12–15 years. No significant effects were observed for knowledge, subjective norm, intention, and fruit, vegetable, and whole grain bread consumption. Although there was an increase in students' nutrition knowledge, it did not show a statistically significant greater improvement in the intervention group compared to students who did not receive the programme. Further research is necessary to examine the long-term sustainability of these positive changes over an extended period.

Acknowledgements. We like to thank the teachers and students for their participation in this study. We would also like to thank the Netherlands Nutrition Centre for their comments and the constructive help in the execution of the study.

Authorship. FAH and MCEB were responsible for formulating the research questions, designing the study, collecting and analysing the data, and writing the article. SV, AS, and MCH assisted with formulating the research questions and reviewing the article. All authors have approved the final article.

Financial support. This work was supported by the Ministry of Agriculture, Nature and Food Quality and Rijksprogramma Jong Leren Eten via the Netherlands Enterprise Agency. The funders had no role in the design, data analyses, or writing of the manuscript.

Competing interests. None.

Ethical standards disclosure. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. The medical ethical review committee of VU Amsterdam University exempted this study from ethical approval (reference: 2022.0305). Written informed consent was obtained from all students. The collection, processing, and storage of data were carried out in accordance with the implementations of the General Data Protection Regulation (GDPR).

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Journal of Nutritional Science

cambridge.org/jns

The relationship between temperament with nutritional status and anthropometric measurements in adult individuals

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Research Article

Cite this article: Icer MA, Çelik E, Köse AG, and Gezmen-Karadag M (2025). The relationship between temperament with nutritional status and anthropometric measurements in adult individuals. *Journal of Nutritional Science* **13**: e97, 1–8. doi: 10.1017/jns.2024.89

Received: 14 August 2024
Revised: 4 October 2024
Accepted: 2 December 2024

Keywords:
Body mass index; Nine types of temperament; Nutritional status; Temperament types

Abbreviations:
CHO:, Carbohydrate; NTTM:, Nine Types of Temperament Model; BMI:, Body Mass Index; WHO:, World Health Organization; BeBiS:, Nutrition Information System; SD:, Standard Deviations; PUFA:, Polyunsaturated Fatty Acid; MUFA:, Monounsaturated Fatty Acid

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We accept to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Abstract

Interest in studies examining the effect of temperament types on nutrition has recently increased. The aim of this study was to evaluate the relationship between nine types of temperament, anthropometric measurements, and nutrition in adults. This study was conducted on 1317 individuals aged between 18 and 55 years. Descriptive information, dietary habits and anthropometric measurements of the participants were questioned. The Nine Types of Temperament Scale was administered to the individuals and food consumption records were obtained with a 24-hour retrospective reminder method. Type 2 scores of obese participants were higher than those of underweight and normal body weight; Type 8 scores of overweight participants were higher than those of normal body weight. Daily dietary intake of protein, riboflavin, folate, vitamins K, C, calcium, iron, and cholesterol were negatively associated with Type 1 score; protein, magnesium, iron, zinc intake, and water consumption were negatively associated with Type 2 score. Type 3 score was negatively associated with dietary CHO (%), dietary magnesium, iron, and zinc intake and positively associated with water consumption. The results of the study indicate significant relationships between temperament types, dietary habits, and anthropometric measures. In this context, considering temperament types when planning dietary patterns of individuals may be a new approach.

Introduction

Temperament is defined as the structural integrity of the individual, which is shaped in early childhood and undergoes relatively little change.[1] A personality component present from early infancy, temperament has a biological background and is partly genetically determined. It is more stable and immutable than other personality traits.[2] Temperament traits lead each individual to different motivations and create different behaviours in individuals with these motivations. Different temperament types can explain this situation. Temperament types are the stable characteristics of an individual that make it possible for them to react similarly in every situation that constitutes their behaviour and attitudes.[3]

One of these scales, The Nine Types of Temperament Model (NTTM), associates individual differences that enable the perception of human behaviours, actions, and attitudes with temperament. In this model, individuals are thought to be born with one of nine different temperament types.[3,4] While the individual shows other behaviours, the basic temperament is essential. The temperament types defined in the nine types of temperament models are named after the most basic needs and pursuits of individuals belonging to that type. These are: 1. Temperament Type Seeking Perfection, 2. Temperament Type Seeking to Feel Emotions, 3. Temperament Type Seeking Admirable Self-Image, 4. Temperament Type Seeking Meaning of Emotions, 5. Temperament Type Seeking the Meaning of Knowledge, 6. Temperament Type Seeking Intellectual Serenity, 7. Temperament Type Seeking the Joy of Discovery, 8. Temperament Type Seeking Absolute Power, 9. Temperament Type Seeking Sensory Movement Comfort.[3]

Previous studies have focused on the role of specific temperamental traits in dieting outcomes. Temperament elements that have been specifically suggested to have potential effects on eating behaviours include impulsivity,[5,6] extraversion,[7] negative emotionality, and self-regulation or effortful control.[8,9] Rothbart *et al.* conceptualised temperament into two main reactivity styles: negative emotionality and boldness/extroversion. Both negative emotionality and boldness are characterised by traits that may increase the consumption of energy-dense, nutrient-poor foods.[10]

When the studies investigating the relationship between temperament and body mass index (BMI) in the literature are examined, it is seen that two temperament traits, agility and resilience,

Table 1. Evaluation of the nine types of temperament scale questions

Types of temperament		Question number	Number of questions
TYPE1	Temperament Type Seeking Perfection	1, 7, 21, 27, 45, 58, 69, 78, 86, 90	10
TYPE2	Temperament Type Seeking to Feel Emotions	11, 19, 33, 41, 44, 49, 53, 61, 70, 83	10
TYPE3	Temperament Type Seeking Admirable Self-Image	12, 18, 26, 35, 38, 59, 65, 87, 88	9
TYPE4	Temperament Type Seeking the Meaning of Emotions	3, 8, 15, 20, 23, 29, 51, 54, 73, 79	10
TYPE5	Temperament Type Seeking the Meaning of Knowledge	2, 9, 13, 34, 39, 47, 57, 60, 66, 71, 76, 81	12
TYPE6	Temperament Type Seeking Intellectual Serenity	4, 6, 28, 36, 48, 55, 64, 68, 82, 84	10
TYPE7	Temperament Type Seeking the Pleasure of Discovery	5, 14, 22, 32, 40, 46, 52, 56, 62, 72	10
TYPE8	Absolute Power Seeking Temperament Type	16, 24, 37, 42, 43, 50, 63, 67, 75, 80	10
TYPE9	Sensory Movement Comfort Seeking Temperament Type	10, 17, 25, 30, 31, 74, 77, 85, 89, 91	10

partially mediate the relationship between gender and BMI.[11,12] Two studies with children[13] and adults[14] found that certain temperament traits, such as high emotionality and novelty seeking, were associated with higher BMI. Sutin *et al.* showed that high neuroticism is associated with higher BMI, while conscientiousness, extraversion, and openness are protectiveness.[15–17] In these studies, it is seen that different scales have been used to determine temperament types.[5–7,18]

Despite the increasing interest in the role of temperament, studies examining the relationship between the nine types of temperament scale, anthropometric measurements and nutritional status in adults are limited. This study aimed to investigate the relationships between temperament types determined using the nine types of temperament model, nutritional status, and anthropometric measurements in adults.

Methods

Work plan and participants

This cross-sectional study was conducted with 1317 volunteer adults (673 women and 644 men) aged between 18 and 55. In determining the number of people included in the study, the study of Braet *et al.*[5] was taken as reference. As a result of the power analysis, it was determined that at least 766 individuals were required to participate in the study in order to have a Type I error (a) = 0.05, effect size 0.24, and power 95%.

Care was taken to ensure that the individuals participating in the study were homogeneously distributed in terms of gender. Individuals younger than 18 and older than 55 years of age, illiterate, pregnant, and lactating women were excluded from the study. This study protocol was approved by the Gazi University Ethics Commission at its meeting dated 13.09.2022 and numbered 15. At the start of questionnaire, full review was obtained and written consent of participants was obtained.

The research data were obtained by the face-to-face questionnaire method. The questionnaire contained information about the participants' demographic characteristics (age, gender, educational status, place of residence, number of siblings, disease status, use of supplements), anthropometric measurements, dietary habits, and the Nine Types of Temperament Scale. In addition, food consumption records were obtained from the participants with a 24-hour retrospective reminder method.

Anthropometric measurements

Participants' body weight and height were taken on a self-reported basis. Body mass index values were calculated as body weight divided by the square of height in metres. Body mass index values were classified according to the World Health Organization's (WHO) classification. Accordingly, those below 18.5 kg/m^2 were categorised as underweight, 18.5–24.9 kg/m^2 as normal weight, 25.0–29.9 kg/m^2 as overweight, and 30.0 kg/m^2 and above as obese.[19]

The nine types of temperament scale

The Nine Types of Temperament Scale developed by Yılmaz *et al.* (2014) is a self-report scale for measuring the characteristics of temperament types.[3] The scale consists of 91 items on nine subscales representing nine different temperament types. Participants were asked to respond using a 3-point Likert scale (2 = yes, 1 = sometimes, 0 = no). Higher scores from a temperament type indicate that the participant has more traits related to that temperament type. Cronbach's alpha value for the total scale was calculated as 0.75.[3] The question numbers indicating the temperament types in the scale are given in Table 1.

Assessment of nutritional status

To determine the energy and nutrient intake of the participants with the daily diet, 24-hour retrospective diet records were taken by the researchers. "Standard Food Recipes for Institutions Serving Mass Nutrition" and "Examples from Turkish Cuisine" were used to determine the amounts of food and the amounts in the portions of the meals.[16,17] The average energy and nutrient values of the foods consumed daily were calculated using the Nutrition Information System (BeBiS) software version 9.0.[20]

Statistical analysis

SPSS 25.0 program was used for data analysis. The chi-square test was used to compare categorical data. Independent Samples *t*-test was used to compare the means ($\bar{X}$) and standard deviations (SD) of qualitative data obtained from two groups with normal distribution; the One-Way ANOVA test was used to evaluate the qualitative data of more than two groups. The relationship between two values was analysed using the Pearson correlation test if the data were normally distributed and the Spearman correlation test if not. The limit for statistical significance was set as $P < 0.05$.

Results

This study was conducted to determine the relationship between nine types of temperament and nutritional behaviours and anthropometric measurements in adults. Table 2 shows the demographic characteristics of the participants according to gender. The mean age of the individuals was 25.4 ± 7.2 years, and it was found that male participants were older than females ($P < 0.05$).

Table 3 shows the relationship between the participants' dietary behaviours according to the NTTM. A statistically significant relationship was found between the number of main meals and the Type 9 score. It was found that the frequency of consuming three main meals was higher than the frequency of consuming one main meal in individuals with high Type 9 scores ($P < 0.05$). There was a relationship between the number of snacks and Type 4, Type 5, and Type 8 scores. The frequency of not consuming snacks and consuming three snacks of individuals with higher Type 4 scores was higher than that of consuming two snacks; the frequency of consuming three snacks of participants with higher Type 4 scores was higher than that of consuming one snack ($P < 0.05$). Individuals with higher Type 5 and Type 8 scores had a higher frequency of not consuming snacks than the frequency of consuming one or two snacks ($P < 0.05$). Participants with high Type 1, Type 3, Type 4, Type 7, and Type 8 scores had higher rates of smoking, while participants with high Type 9 scores had lower rates of smoking ($P < 0.05$). Participants with higher Type 3, Type 4, Type 7, and Type 8 scores had higher rates of alcoholic beverage consumption, while participants with higher Type 9 scores had lower rates of alcoholic beverage consumption ($P < 0.05$). Considering the amount of alcoholic beverages consumed, individuals who consumed 1001–3000 ml of alcoholic beverages had higher Type 7 scores compared to those who consumed <1000 ml ($P < 0.05$). Participants who ate with friends had higher Type 1 scores than those who ate alone or with family ($P < 0.05$). Participants who ate alone had higher Type 3 and Type 5 scores than other groups, and participants who ate alone had higher Type 4 and Type 6 scores than those who ate with family ($P < 0.05$). Type 8 scores of the participants who ate with their families were higher than the other groups; Type 9 scores of the participants who ate with their families were higher than those who ate alone ($P < 0.05$).

The BMI evaluation of the participants according to temperament types is given in Table 4. A statistically significant difference was found between BMI and Type 2, Type 5, and Type 8 scores ($P < 0.05$). Obese participants had higher Type 2 scores than underweight and normal body weight participants ($P < 0.05$). Underweight participants had higher Type 5 scores than obese participants ($P < 0.05$). Overweight participants had higher Type 8 scores than those with normal body weight ($P < 0.05$).

The correlation between temperament types and nutrients is given in Fig. 1. There was a negative and statistically significant correlation between daily dietary protein, daily fat intake percentage, riboflavin, folate, vitamins K and C, calcium, iron, cholesterol intake, and Type 1 score, and a positive correlation between dietary CHO (%) and dietary polyunsaturated fatty acid (PUFA) and Type 1 score ($P < 0.05$). Type 2 score was negatively correlated with daily dietary protein, magnesium, iron, zinc intake, and water consumption ($P < 0.05$). Type 3 score was negatively correlated with dietary CHO (%), dietary magnesium, iron, and zinc intake, and positively correlated with water consumption ($P < 0.05$). There was a positive correlation between daily dietary protein, PUFA, and w-3 fatty acid intake and Type 4 score ($P < 0.05$). Type 5 score was negatively correlated with daily dietary energy and niacin intake and positively correlated with water consumption ($P < 0.05$). There was a negative correlation between water consumption and Type 6 score and a positive correlation between Type 7 and Type 8 scores ($P < 0.05$). There was a positive correlation between daily dietary cholesterol intake and Type 8 score ($P < 0.05$). There was a positive correlation between daily dietary fat consumption percentage and Type 9 ($P < 0.05$).

Discussion

This study examined the relationship between temperament types determined by the nine types of temperament model and anthropometric measurements and dietary habits. The study results show a relationship between temperament types and anthropometric measurements, some eating habits, and dietary intake of various nutrients. The limited number of studies examining this issue in the literature and the fact that these studies generally focus on negative affect reveals the deficiencies in this field.

Research consistently emphasises the negative impact of skipping meals, especially breakfast, on physical and mental health.[21–23] In one study, neglecting breakfast was associated with reduced energy intake.[21] Skipping meals is positively correlated with mental distress.[23] A study conducted on children reported that children with extroverted and internalising temperaments were likelier to skip breakfast every day. In contrast, children with average sociability scores had regular breakfast habits.[22] This study found that the frequency of consuming three main meals was higher in individuals with high Type 9 scores compared to individuals who consumed one main meal. The study population consisted of adult individuals, and no other study in the literature examines the relationship between temperament types and meal skipping in adults. Therefore, this study is predicted to lead to new studies on whether temperament types are effective in meal skipping.

Research shows that smoking and alcohol consumption are associated with poor eating habits, such as low intake of fruits, vegetables, and cereals and high fat and alcohol consumption.[24–26] These habits can lead to lower nutrient intake and poorer food choices, negatively affecting overall health and nutrition. It has been reported that smokers tend to have lower fear and control and drive higher anger and unstable emotional temperament.[27] Another study supported this by showing that smokers have lower harm avoidance and higher novelty-seeking, reward dependence, and persistence.[28] Participants with higher Type 1, Type 3, Type 4, Type 7, and Type 8 scores had higher rates of smoking, while participants with higher Type 9 scores had lower rates of smoking ($P < 0.05$). Participants with higher Type 3, Type 4, Type 7, and Type 8 scores had higher rates of alcoholic beverage consumption, while participants with higher Type 9 scores had lower rates of alcoholic beverage consumption. In line with the results obtained from the literature and this study, it can be concluded that temperament types may be effective on smoking and alcohol use habits. However, there is a need for more

Table 2. General information and sociodemographic characteristics of participants by gender

	Women (n = 673) n (%)	Men (n = 644) n (%)	Total (n = 1317) n (%)	x^2	P
Age group					
18–30 years	589 (87.5)	515 (80.0)	1104 (83.8)	13.84	**<0.001**
31–55 years	84 (12.5)	129 (20.0)	213 (16.2)		
Age (X̄ ± SD)	24.5 ± 6.6	26.4 ± 7.6	25.4 ± 7.2	df = 1315	**<0.001**
Education Status					
Primary/Secondary School	14 (2.0)	16 (2.5)	30 (2.3)	13.48	**0.009**
High School	59 (8.8)	93 (14.4)	152 (11.5)		
University	570 (84.7)	496 (77.0)	1066 (80.9)		
Master's Degree/PhD	30 (4.5)	39 (6.1)	69 (5.2)		
Marital status					
Married	89 (13.2)	149 (23.1)	238 (18.1)	23.20	**<0.001**
Single	583 (86.6)	492 (76.4)	1075 (81.6)		
Profession					
Student	461 (68.5)	341 (53.0)	802 (60.9)	92.13	**<0.001**
Officer	65 (9.7)	105 (16.3)	170 (12.9)		
Worker	22 (3.3)	72 (11.2)	94 (7.1)		
Self-employment	65 (9.7)	94 (14.6)	159 (12.1)		
Housewife	36 (5.3)	–	36 (2.7)		
Other	24 (3.6)	32 (5.0)	56 (4.3)		
Place of living					
Urban	621 (92.3)	617 (95.8)	1238 (94.0)	7.29	**0.07**
Rural	52 (7.7)	27 (4.2)	79 (6.0)		
Number of siblings					
None	60 (8.9)	56 (8.7)	116 (8.8)	1.04	0.90
One	220 (32.7)	208 (32.2)	428 (32.5)		
Two	195 (29.0)	191 (29.7)	386 (29.3)		
Three and more	198 (29.4)	189 (29.3)	387 (29.4)		
Presence of diagnosed disease					
Yes	62 (9.2)	52 (8.1)	114 (8.7)	0.54	0.46
No	611 (90.8)	592 (91.9)	1203 (91.3)		
Chronic disease					
Cardiovascular diseases	6 (9.7)	8 (15.4)	14 (12.3)	0.86	0.36
Diabetes	3 (4.8)	9 (17.3)	12 (10.5)	4.70	**0.03**
Hypertension	2 (3,2)	10 (19.2)	12 (10.5)	7.69	**0.006**
Digestive system diseases	7 (11.3)	8 (15.4)	15 (13.2)	0.42	0.52
Respiratory system diseases	8 (12.9)	5 (9.6)	13 (11.4)	0.30	0.58
Mental disorders	6 (9.7)	5 (9.6)	11 (9.6)	<0.001	0.99
Musculoskeletal system diseases	9 (14.5)	9 (17.3)	18 (15.8)	0.17	0.68
Endocrine diseases	8 (12.9)	3 (5.8)	11 (9.6)	1.65	0.19
Vitamin/Mineral deficiencies	15 (24.2)	5 (9.6)	20 (17.5)	4.15	**0.04**
Neurological diseases	9 (14.5)	–	9 (7.9)	8.19	**0.004**
Vitamin/mineral supplements					
Yes	94 (14.0)	62 (9.6)	156 (11.8)	5.94	**0.02**
No	579 (86.0)	582 (90.4)	1161 (88.2)		

https://doi.org/10.1017/jns.2024.89 Published online by Cambridge University Press

Table 3. Evaluation of participants' dietary habits and smoking status according to temperament types

Nutrition Behaviours	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6	Type 7	Type 8	Type 9
Number of main meals									
One	19.5 ± 3.9	11.4 ± 4.3	9.1 ± 3.6	10.7 ± 4.2	10.8 ± 5.4	13.1 ± 3.7	10.2 ± 3.1	8.8 ± 4.4	9.8 ± 4.8[a]
Two	18.8 ± 3.9	12.0 ± 4.2	9.5 ± 3.6	9.8 ± 4.3	10.7 ± 5.1	12.7 ± 3.9	10.5 ± 3.5	8.9 ± 4.6	11.2 ± 3.9
Three	18.4 ± 4.0	11.7 ± 4.1	9.5 ± 3.4	9.7 ± 4.2	10.9 ± 4.8	12.6 ± 3.8	10.7 ± 3.5	9.3 ± 4.60	11.6 ± 3.7[a]
P	0.07	0.27	0.75	0.43	0.54	0.22	0.45	0.43	**<0.01**
Number of snacks									
None	19.0 ± 3.2	11.6 ± 2.9	10.3 ± 2.8	10.7 ± 3.4[a]	12.8 ± 4.2[a.b]	11.7 ± 3.3	11.2 ± 2.6	10.7 ± 4.0[a.b]	11.8 ± 2.8
One	18.5 ± 4.0	11.7 ± 4.1	9.5 ± 3.4	9.7 ± 4.4[b]	10.4 ± 5.0[a]	12.6 ± 3.8	10.6 ± 3.5	9.1 ± 4.6[a]	11.4 ± 4.1
Two	18.8 ± 4.1	11.9 ± 4.3	9.3 ± 3.6	9.6 ± 4.2[a.c]	10.8 ± 5.0[b]	12.7 ± 3.9	10.4 ± 3.5	8.7 ± 4.8[b]	11.2 ± 3.9
Three	18.3 ± 3.8	11.8 ± 4.2	9.5 ± 3.4	10.7 ± 4.2[b.c]	11.4 ± 4.6	12.4 ± 3.5	11.0 ± 3.4	9.6 ± 4.2	11.5 ± 3.7
P	0.38	0.86	0.11	**<0.01**	**<0.01**	0.15	0.06	**<0.01**	0.46
Smoking									
Yes	18.9 ± 4.1	12.1 ± 4.4	9.8 ± 3.5	10.3 ± 4.1	10.6 ± 5.1	12.3 ± 3.9	11.1 ± 3.3	10.1 ± 4.5	10.9 ± 3.9
No	18.5 ± 3.9	11.7 ± 4.1	9.4 ± 3.4	9.6 ± 4.3	10.9 ± 4.9	12.7 ± 3.8	10.4 ± 3.5	8.8 ± 4.6	11.6 ± 3.8
P	**0.05**	0.09	**0.03**	**0.01**	0.24	0.11	**<0.01**	**<0.01**	**<0.01**
Number of cigarettes smoked per day									
1–10	18.9 ± 3.9	12.3 ± 4.3	9.9 ± 3.4	10.2 ± 4.0	10.5 ± 4.8	12.2 ± 3.8	10.9 ± 3.3	9.7 ± 4.3	11.1 ± 3.9
11–20	19.0 ± 4.2	12.0 ± 4.4	9.6 ± 3.6	10.5 ± 4.3	10.6 ± 5.3	12.5 ± 4.1	11.4 ± 3.2	10.4 ± 4.7	10.8 ± 3.8
>20	19.1 ± 4.9	11.2 ± 4.8	11.5 ± 3.8	9.5 ± 3.8	10.5 ± 5.7	11.4 ± 3.8	10.9 ± 3.4	12.1 ± 5.0	9.4 ± 3.8
P	0.99	0.10	0.24	0.73	0.16	0.76	0.08	0.12	0.25
Consumption of alcoholic beverages									
Yes	18.8 ± 4.4	11.9 ± 4.7	10.1 ± 3.6	10.5 ± 4.1	10.5 ± 4.9	12.7 ± 3.9	11.4 ± 3.5	9.7 ± 5.0	10.9 ± 4.0
No	18.6 ± 3.9	11.8 ± 4.0	9.4 ± 3.4	9.6 ± 4.2	10.9 ± 4.9	12.5 ± 3.8	10.5 ± 3.4	9.0 ± 4.5	11.5 ± 3.8
P	0.46	0.63	**<0.01**	**0.01**	0.25	0.60	**<0.01**	**0.04**	**0.05**
Amount of alcoholic beverages consumed									
0–1000 ml	18.8 ± 4.5	12.0 ± 4.7	9.9 ± 3.5	10.4 ± 4.2	10.6 ± 5.1	12.9 ± 3.9	11.1 ± 3.6[a]	9.5 ± 5.0	11.1 ± 3.9
1001–3000 ml	19.5 ± 4.1	11.7 ± 4.7	10.8 ± 4.0	10.9 ± 3.5	9.3 ± 5.9	11.9 ± 3.8	12.7 ± 2.7[a]	10.8 ± 5.1	9.5 ± 4.5
>3000 ml	16.0 ± 4.2	9.9 ± 4.0	12.3 ± 4.6	9.5 ± 5.1	11.6 ± 6.3	10.8 ± 5.1	12.8 ± 3.6	12.5 ± 3.4	11.9 ± 3.4
P	0.14	0.43	0.11	0.70	0.42	0.19	**0.05**	0.13	0.13
The person with whom you eat									
Family	18.4 ± 3.8[a]	11.9 ± 3.9	9.4 ± 3.3[a]	9.6 ± 4.4[a]	10.9 ± 4.8[a.b]	12.2 ± 3.9[a]	10.5 ± 3.4	9.5 ± 4.4[a]	11.6 ± 3.7[a]
Lonely	18.3 ± 4.3[b]	11.7 ± 4.5	10.1 ± 3.6[a.b]	10.5 ± 4.1[a]	11.8 ± 5.1[a.c]	13.2 ± 3.6[a]	10.8 ± 3.7	9.4 ± 5.2[b]	10.8 ± 4.3[a]
Friends	19.3 ± 4.1[a.b]	11.8 ± 4.4	9.3 ± 3.6[b]	9.8 ± 4.2	10.1 ± 4.9[b.c]	12.8 ± 3.7	10.7 ± 3.4	8.4 ± 4.5[a.b]	11.3 ± 3.8
P	**<0.01**	0.73	**0.01**	**0.01**	**<0.01**	**<0.01**	0.44	**<0.01**	**0.02**

There is a statistically significant difference between the data indicated with the same letter.

Table 4. Evaluation of BMI of participants according to temperament types

	BMI				
Temperament Types	Underweight (n = 119)	Normal (n = 807)	Overweight (n = 319)	Obese (n = 72)	P
TYPE1	18.7 ± 3.4	18.7 ± 4.1	14.5 ± 3.9	18.4 ± 3.8	0.73
TYPE2	11.6 ± 3.9[a]	11.7 ± 4.1[b]	11.9 ± 4.4	13.2 ± 4.1[a,b]	**0.02**
TYPE3	9.7 ± 3.3	9.4 ± 3.4	9.6 ± 3.7	9.2 ± 3.5	0.65
TYPE4	10.8 ± 4.7	9.7 ± 4.1	9.8 ± 4.3	9.3 ± 4.2	0.06
TYPE5	11.7 ± 4.5[a]	10.7 ± 4.9	11.1 ± 4.9	9.8 ± 5.2[a]	**0.03**
TYPE6	12.8 ± 3.6	12.7 ± 3.8	12.3 ± 3.8	11.8 ± 4.1	0.12
TYPE7	10.6 ± 3.3	10.6 ± 3.50	10.5 ± 3.4	11.1 ± 3.4	0.60
TYPE8	9.3 ± 4.7	8.7 ± 4.6[a]	9.9 ± 4.4[a]	10.1 ± 4.9	**<0.001**
TYPE9	11.2 ± 3.4	11.1 ± 3.9	11.6 ± 3.8	12.2 ± 4.0	0,14

BMI, Body Mass Index: a statistically significant difference exists between the data shown with the same letter.

Nutrients	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6	Type 7	Type 8	Type 9
Energy	-0.02	-0.03	0.001	0.03	**-0.29**	0.01	0.01	0.001	0.02
Protein	**-0.33**	**-0.06**	0.04	-0.01	-0.11	-0.01	0.03	0.02	0.005
Protein (%)	-0.02	-0.05	0.05	**0.06**	0.02	-0.03	0.02	0.02	-0.02
Fat	-0.04	-0.03	0.05	0.03	-0.02	0.01	0.02	0.01	0.03
Fat (%)	**-0.06**	0.001	0.03	0.001	0.02	0.001	0.01	0.02	**0.07**
CHO	0.02	-0.02	-0.02	0.03	-0.03	0.02	-0.002	-0.02	-0.002
CHO (%)	**0.07**	0.03	**-0.06**	0.02	-0.02	0.02	-0.02	-0.03	-0.05
Fiber	-0.04	-0.02	-0.02	0.03	-0.01	0.001	0.03	-0.01	0.01
Vitamin E	-0.04	-0.02	-0.02	0.03	-0.01	0.03	-0.01	-0.003	0.05
Vitamin K	**-0.08**	-0.01	-0.01	0.02	0.02	0.02	-0.002	0.05	-0.05
Thiamine	-0.02	-0.03	-0.03	0.03	-0.02	0.01	0.04	-0.005	0.01
Riboflavin	**-0.07**	-0.01	-0.01	0.001	-0.01	-0.004	0.04	0.02	0.005
Niacin	0.01	-0.04	-0.04	-0.04	**-0.06**	0.002	0.04	-0.03	0.001
Vitamin B_5	0.02	-0.02	-0.02	0.001	-0.02	-0.01	0.05	-0.01	0.02
Vitamin B_6	-0.01	-0.05	-0.05	0.03	0.01	0.01	0.03	0.001	0.002
Folate	**-0.08**	-0.02	-0.02	0.03	0.02	0.02	0.003	-0.01	0.009
Vitamin C	**-0.06**	0.001	0.001	0.03	0.01	0.02	-0.03	-0.004	-0.02
Potassium	-0.05	-0.04	-0.04	0.03	0.01	-0.01	0.02	0.004	0.01
Calcium	**-0.08**	-0.01	-0.01	0.01	0.03	-0.008	-0.01	0.02	0.009
Magnesium	-0.02	**-0.05**	**-0.05**	0.01	-0.02	-0.003	0.03	0.002	0.001
Phosphorus	-0.04	-0.05	-0.05	0.001	-0.02	-0.008	0.04	0.01	0.01
Iron	**-0.05**	**-0.05**	**-0.05**	-0.02	-0.02	-0.009	0.01	0.006	0.008
Zinc	-0.05	**-0.06**	**-0.06**	-0.04	-0.05	-0.01	0.009	0.004	0.03
Copper	0.001	-0.03	-0.03	0.02	-0.03	-0.006	0.04	-0.009	-0.006
MUFA	-0.01	-0.03	-0.03	0.02	-0.03	0.01	0.02	0.003	0.04
PUFA	**0.09**	-0.02	-0.02	**0.06**	-0.01	0.03	0.009	0.001	0.02
Cholesterol	**-0.06**	0.04	-0.04	-0.05	-0.02	-0.005	-0.004	**0.38**	0.03
w-3	-0.02	0.04	0.05	**0.07**	0.03	0.001	0.008	0.05	-0.03
w-6	0.001	-0.01	0	0.05	-0.01	0.03	0.008	-0.005	0.03
Water	-0.04	**-0.06**	**0.08**	0.04	**0.06**	**-0.06**	**0.09**	**0.11**	0.02

Data shown in bold are statistically significant. Blue indicates a negative correlation; red indicates a positive correlation. The correlation strength increases as we move to the left and right of the color scale.

Fig. 1. The relationship between participants' temperament types and macro and micronutrient intakes.

comprehensive studies using similar temperament scales to reveal more clearly which temperament types affect these habits.

Although there has been a recent increase in studies investigating the effects of temperament types on the risk of obesity development, these studies have often focused on children.[9,29] In adults, neurotic personality trait is associated with higher body mass index.[30,31] In contrast, people with self-regulation and conscientiousness temperaments are reported to have a lower risk of developing obesity.[9,31] van Eeden *et al.* (2020) found that higher negative affect and lower effortful control in pre-adolescence were generally associated with higher BMI in young adulthood. The same study predicted that common effortful control may consistently increase the development of obesity in adolescents.[29] In this study, it was observed that obese individuals had higher scores in the temperament type seeking to feel emotions (Type 2) and absolute power-seeking temperament type (Type 8) and lower scores in the temperament type seeking the meaning of information (Type 5). This result suggests that individuals predisposed to Type 2 and Type 8 temperament types may be at higher risk for obesity, but more studies are needed in this regard.

Negative affect temperament type is generally associated with consuming more energy-dense, nutrient-poor foods, high-sugar foods, and sugar-sweetened beverages. Kidwell *et al.* (2023) found that adolescents with high negative affectivity were more likely to consume foods and beverages and sugar than those with low negative affectivity. Another result of that study was that having internalising (anxious and dependent) and externalising (hyperactive and aggressive) temperaments in 18-month-olds is a risk factor for greater consumption of sweet drinks and foods at ages three and seven.[32] Another study showed that active and social temperament in 18-month-old infants was associated with higher daily fruit and vegetable consumption at three and seven years of age.[7] In the present study, Type 2 score was negatively correlated with daily dietary protein, magnesium, iron, zinc intake, and water consumption. There was a positive correlation between daily dietary protein, PUFA, and w-3 fatty acid intake and Type 4 score. Another study result was a negative correlation between the score of perfection-seeking temperament type (Type 1) and daily dietary intake of protein and protein, vitamin K, riboflavin, folate, vitamin C, calcium, iron, and cholesterol. In light of the present data, it can be considered that predisposition to some temperament types may affect nutritional status and dietary intake levels of some macro/micronutrients and bring potential health risks, especially obesity.

This study has some limitations. The Nine Types of Temperament Scale used in this study has yet to be used before to assess the relationship between temperament and nutritional status in adults. Although this adds a unique value to the study, it needs to be clearer to compare the results of the survey with the results of other studies using different scales to determine temperament types.

Conclusions

The results obtained from this cross-sectional study show that some temperament types in the nine types of temperament models are associated with anthropometric measurements and nutritional behaviours. When it is considered that genetics is influential in determining temperament and does not change frequently, it is essential to design interventions by considering temperament characteristics instead of only providing nutritional recommendations to individuals. With the new studies to be conducted, it can be thought that determining the diet according to temperament types may be a new alternative strategy to improve the nutritional status and prevent the development of obesity by revealing the effects of temperament types on nutritional status more clearly. Our study anticipates accelerating new studies to be planned for this purpose.

Acknowledgements. The authors declare that they have no conflicts of interest and that they did not reveive any funding. The authors would also like to thank everyone who participated in this study. Author contributions are as follows: EÇ and MGK: Conceptualisation; Contributing to the design of the study. MAI, EÇ, AGK, and MGK: Conceptualisation; data curation; formal analysis; investigation; methodology; project administration; resources; software; supervision; visualisation; writing—original draft; writing—review and editing.

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Journal of Nutritional Science

cambridge.org/jns

Chia seed supplementation and inflammatory biomarkers: a systematic review and meta-analysis

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Review

Cite this article: Pam P, Asemani S, Azizi MH, and Jamilian P (2024). Chia seed supplementation and inflammatory biomarkers: a systematic review and meta-analysis. *Journal of Nutritional Science* **13**: e91, 1–9. doi: 10.1017/jns.2024.70

Received: 15 June 2024
Revised: 20 August 2024
Accepted: 9 September 2024

Keywords:
C-reactive protein; Chia seed; Inflammation; Interleukin-6; Tumour necrosis factor-alpha

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Abstract

Chia seeds have gained attention for their potential anti-inflammatory properties, which may be attributed to their high content of omega-3 fatty acids, dietary fibre, and antioxidants. This study aims to provide an overview of the current understanding regarding the effects of chia seeds on inflammatory markers, specifically C-reactive protein (CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α). A comprehensive literature search was conducted on PubMed, Scopus, Web of Science, Cochrane, and Google Scholar up to June 2024. Randomized controlled trials (RCTs) assessing the effect of chia seed on CRP or/and IL-6 or/and TNF-α. Data were extracted and analysed using a random-effects model, and reported as weighted mean differences (WMD) with 95% confidence intervals (CI). Subgroup and sensitivity analyses were also performed. Four RCTs involving 210 participants were included in the meta-analysis. The results showed that chia consumption significantly decreased CRP (WMD: –0.64 mg/dl; 95% CI: –1.24, –0.04; P = 0.03). But it had no significant effect on IL-6 (WMD: 0.29 pg/dl; 95% CI: –0.40, 0.98; P = 0.41), and TNF-α (WMD: 0.05%; 95% CI: –0.21 to 0.30; P = 0.72). Chia consumption can significantly decrease CRP, but no significant effect was observed on IL-6 and TNF-α. To prove our findings, more studies with a larger sample size are needed.

Introduction

Cellular damage prompts alarm signals in our biological system, triggering an inflammatory response aimed at countering the damage and maintaining internal stability.[1] In conditions like cancer, sepsis, and autoimmune disorders, there is disruption in the regulation of inflammatory processes.[2] Inflammation plays a crucial role after infections or physical trauma by orchestrating tissue repair, restoring equilibrium, and bolstering the host's defences against external pathogens.[3] The inflammatory cascade begins with a rapid induction phase, followed by pro-inflammatory stages and resolution phases. Disruption in these coordinated phases leads to uncontrolled inflammation, contributing to various inflammatory diseases such as neurological disorders, autoimmune conditions, cancer, sepsis, cardiovascular diseases, and obesity.[4]

Chia (Salvia hispanica L.), originally from northern Guatemala and southern Mexico, is an annual herbaceous plant that has spread worldwide.[5] Chia seeds are valued for their functional and nutritional properties, including their ability to form a gel-like consistency when mixed with water due to their high mucilage content. This property enhances the texture of various recipes. Chia seeds are versatile and are used in different forms such as oils, flours, and whole seeds. They are integrated into a wide range of foods such as fruit smoothies, salads, dairy beverages, cereal bars, breads, cookies, yogurts, fruits, and cakes. Furthermore, chia seeds are utilized as effective thickening agents in sauces and soups, making them highly versatile in culinary applications.[6–8]

It is noteworthy that chia seeds contain approximately 20% protein.[9] The proteins obtained from digested chia seeds have anti-inflammatory properties. Specifically, these proteins inhibit PPARγ, which in turn reduces the expression of nuclear factor-kappa B (NF-κB), thereby mitigating inflammation.[10] Research indicates that during adipogenesis, digested proteins like albumin and glutelin inhibit the expression of sterol regulatory element-binding protein 1 (SREBP), which is known to activate PPARγ.[11] This reduction in SREBP expression leads to a diminished stimulation of NF-κB, resulting in decreased inflammation.[10] Studies investigating the neuroprotective effects of chia peptides have identified three fractions (1, 1–3, and 3–5 k Dalton) derived from enzymatic hydrolysis of chia proteins. These peptides demonstrate protective and anti-inflammatory effects on nervous system cells, particularly HMC3 microglia cells, by reducing reactive oxygen species (ROS) and inflammatory mediators such as TNF-α, IL-6, H2O2, and NO.[12]

Three meta-analyses have been conducted, and published in 2018[13] and 2023.[14,15] The studies by NikPayam *et al.* in 2023 and Teoh *et al.* in 2018,[13] included research that integrated additional supplements alongside chia as interventions, potentially confounding the true impact of chia. Given this limitation, our study specifically focuses on investigations solely examining chia as the intervention. Furthermore, Sarmiento *et al.*[15] conducted a meta-analysis showing reduced serum CRP levels with chia seed consumption, but they did not evaluate TNF and interleukin as inflammatory indicators. Their analysis included data from 3 publications (5 studies) up to 2021. In contrast, another study combined data from 4 publications (6 studies) and employed a random-effects model to better handle the heterogeneous data observed across the studies, which was deemed more suitable than the fixed model used by Sarmiento *et al.*

Our study stands out for its exclusive focus on chia seeds, unlike previous meta-analyses that included studies with additional supplements.[13,14] In addition, we have compiled a larger data set than previous meta-analyses.[15] Using a random-effects model addresses the heterogeneity among studies, thereby strengthening the reliability of our findings. This meticulous approach aims to advance the understanding of chia's therapeutic potential in managing inflammatory biomarkers.

Materials and methods

The current study adhered to the guidelines outlined in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[16] The study's protocol has been officially recorded on PROSPERO with the registration code **CRD42023442343**.

Search strategy

We conducted a comprehensive literature search on PubMed, Scopus, Web of Science, Cochrane, and Google Scholar up to June 2024. The merge of MESH and non-MESH terms were used as follows: ("chia" OR "chia seed" OR "Salvia hispanica") AND ("randomized controlled trial"). The details of the search strategy are provided in Supplementary Table 1. To ensure the comprehensive inclusion of relevant studies, we also manually searched the reference lists of eligible studies, as well as relevant reviews and meta-analyses (hand-search method). The language was limited to English studies only.

Eligibility criteria

The guidelines for selecting studies and conducting the meta-analysis were established using the PICO approach, which involves outlining the Population (P), Intervention (I), Comparison (C), and Outcome (O) criteria for the search process. This meta-analysis incorporated studies meeting the following criteria: (a) randomized controlled trials (RCTs), (b) involving participants aged 18 years and above, (c) reporting at least one of the following outcomes: CRP and/or IL-6 and/or TNF-α.

Studies were excluded if they met any of the following criteria: (a) non-randomized study design, (b) assessed the effects of chia in combination with other interventions, (c) lacked sufficient information regarding the outcomes of interest, (d) had a follow-up period of less than one week, or (e) were conducted exclusively on children, pregnant or lactating women. We used only published studies and did not use grey literature.

Data extraction

The study selection process was conducted independently by two researchers (P.P and S.A.), with a chief investigator (P.J.) available to resolve any discrepancies. In cases where data were unavailable, we contacted the corresponding author via email to request the necessary information. The following details were extracted from each included study: the first author's name, year of publication, study location, duration of the study, participant demographics including gender, mean age, and mean body mass index (BMI), study design, the health status of the study population, sample size in each group, chia seed consumption dosage, as well as measurements of CRP and/or IL-6 and/or TNF-α before and after the intervention.

Data synthesis

The primary outcome was assessed by calculating the mean and standard deviation (SD) differences between before and after supplementation in CRP, IL-6, and TNF-α in intervention and placebo groups. A random-effects model was employed to estimate the overall effect size using restricted maximum likelihood (REML) method. As the measurement units of outcomes were the same, the data were reported as weighted mean difference (WMD). To convert the standard error of the mean (SEM) into SD, the following formula was used: $SD = SEM \times \sqrt{n}$ (where n represents the number of participants in each group). The Cochrane's Q test (with significance set at $P < 0.1$) and I^2 statistic were utilized to assess heterogeneity among studies, with an I^2 value >50% indicating substantial heterogeneity.[17] Predefined subgroup analyses were conducted based on intervention duration, chia seed dosage, gender, and health status to explore potential sources of heterogeneity. Additionally, a sensitivity analysis was performed to examine the impact of individual studies or groups of studies on the overall results. Because the number of observations for each outcome was less than 8, Begg's and Egger's tests and visual inspection of the funnel plot were not performed. Statistical analyses were conducted using STATA software (version 14.0; StatCorp, College Station, TX, USA). A significance level of $P < 0.05$ was predetermined to indicate statistical significance.

Quality assessment

Version 2 of the Cochrane risk-of-bias tool (RoB2) was used to assess the quality of the included studies (Table 1).[18] This system consists of five criteria to evaluate the risk of bias, which are as follows: Randomization Process, Deviation from Intended Interventions, Missing Outcome Data, Measurement of the Outcome, and Selection of the Reported Result. After evaluating these domains for each study, an overall judgement of 'low risk', 'some concerns', or 'high risk' is assigned to indicate the level of bias in the study. The assessment of the overall risk of bias was determined based on specific criteria: studies were categorized as having a low risk of bias when all domains were deemed to have a low risk; categorized as having some concerns when at least one domain raised some concerns but none were considered high risk; and categorized as high risk of bias if at least one domain was assessed as high risk or if multiple domains raised some concerns.

https://doi.org/10.1017/jns.2024.70 Published online by Cambridge University Press

Table 1. Quality assessment

Studies	Randomization Process	Deviation from Intended Interventions	Selection of the Reported Result	Measurement of the Outcome	Missing Outcome Data	General risk of bias
Nieman *et al.* 2009(A)	U	L	L	L	L	some concerns
Nieman *et al.* 2009(B)	U	L	L	L	L	some concerns
Nieman *et al.* 2012(A)	U	L	L	L	L	some concerns
Nieman *et al.* 2012(B)	U	L	L	L	L	some concerns
Vuksan *et al.* 2017	L	L	L	L	L	Low
Vuksan *et al.* 2007	L	L	L	H	H	High

Abbreviations: U, Unclear risk' of bias; L, Low risk of bias; H, High risk of bias.

Certainty assessment

The overall certainty of evidence across the studies was graded according to the guidelines of the GRADE (Grading of Recommendations Assessment, Development, and Evaluation) Working Group. The quality of evidence was classified into four categories, according to the corresponding evaluation criteria: high, moderate, low, and very low.[19]

Results

Study selection

Out of the initial pool of 341 articles identified through the primary search, 146 duplicate studies were removed. Following the screening of titles and abstracts, an additional 195 studies were excluded based on the predetermined inclusion criteria: studies with unrelated titles and abstracts (n = 168), animal studies (n = 21), and review articles (n = 6). As a result, 23 articles remained for full-text screening, and after this evaluation, 19 articles were ineligible for inclusion because they lacked essential information, such as the absence of a control group, focus on paediatric subjects, inclusion of co-supplements, or involvement of animal-based studies. Ultimately, 4 studies (6 effect size) fulfilled all the inclusion criteria and were included in the meta-analysis. The search process and study selection are visually presented in Fig. 1, following the PRISMA flow diagram.

Study characteristics

Overall, four studies with 6 datasets, comprising 210 participants (106 cases and 104 controls), were included. The included studies were published between 2007 and 2023. The follow-up period ranged from 10[20] to 24[21] weeks, and the sample size of the included studies ranged from 20[22] to 58[21] participants. All of the studies were parallel RCTs, except for one study that had a cross-over design.[22] Selected studies enrolled subjects with type 2 diabetes mellitus[21,22] and overweight.[20,23] The investigations were conducted in the USA[20,23] and Canada.[21,22] One data set enrolled only males, three only enrolled females, and two involved both genders. Characteristics of the included studies are abstracted in Table 2.

Effect of chia supplementation on CRP

Four publications (with 6 effect size), containing 210 participants (106 cases and 104 controls), examined the effects of chia on CRP. The overall effect size showed that chia supplementation had a significant effect on CRP reduction (WMD: −0.64 mg/dl; 95% CI: −1.24 to −0.04; P = 0.03) (Fig. 2a), with no degree of heterogeneity ($I^2 = 0\%$, P = 0.92). The results of the subgroup analysis showed that chia supplementation was effective in decreasing CRP in patients with T2DM, duration of intervention ≥12 weeks, and intervention dosage >35 g/day (Table 3).

Effect of chia on IL-6

Pooling effect sizes from two publications (with 4 effect sizes), including 132 participants (69 cases and 63 controls), indicated that chia had no significant effect on IL-6, compared with placebo (WMD: 0.29 pg/dl; 95% CI: −0.40 to 0.98; P = 0.41), with no between-study heterogeneity ($I^2 = 0\%$, P = 0.90) (Fig. 2b).

Effect of chia on TNF-α

Two publications (4 effect size), including 132 participants (69 cases and 63 controls), examined the effects of chia on TNF-α. The overall effect size showed that chia had no significant effect on TNF-α (WMD: 0.05 %; 95% CI: −0.21 to 0.30; P = 0.72), with no between-study heterogeneity ($I^2 = 0\%$, P = 0.70) (Fig. 2c). Subgroup analysis confirmed non-significance in all subgroups (Table 3).

Sensitivity analysis

To ascertain each study's impact on the overall effect size, we omitted each trial from the analysis, step by step. After deleting the study of Vuksan *et al.* 2007[22] and Vuksan *et al.* 2017[21] the overall effect of chia on CRP changed to (WMD: −0.49, CI 95%: −1.24, 0.25) and (WMD: −0.53, CI 95%: −1.22, 0.14). No significant effect was observed for IL-6 and TNF-α following sensitivity analysis.

Quality assessment

The methodological quality and risk of bias for the eligible trials are detailed in Table 1. The majority of trials showed some concerns regarding their quality according to the RoB2 tool criteria.

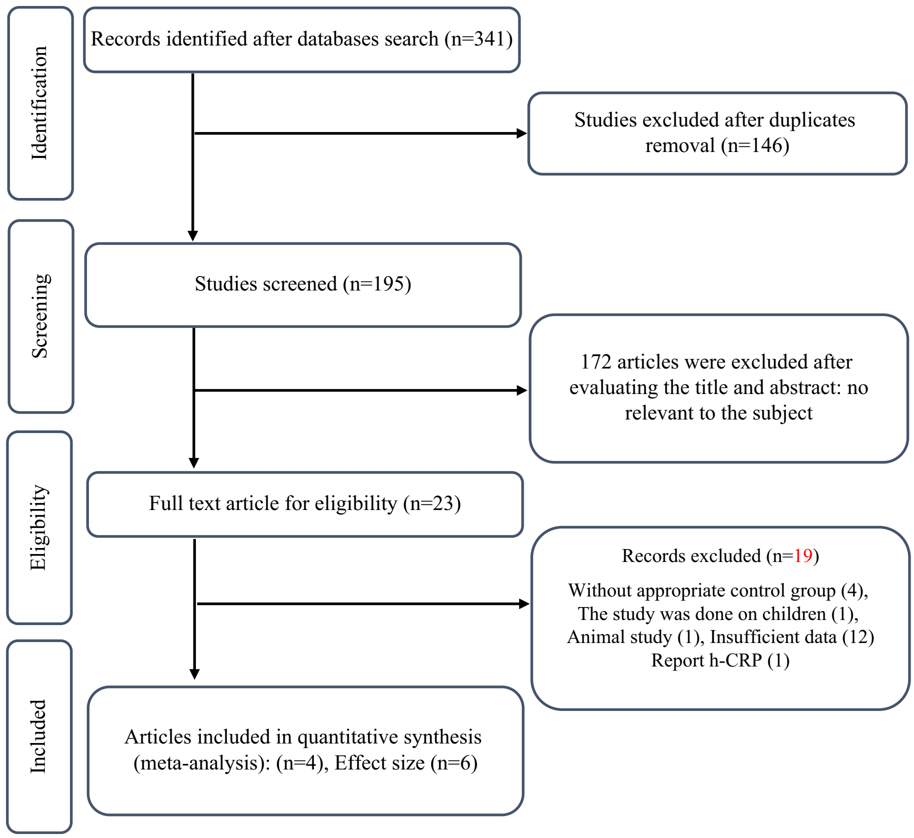

Figure 1. Flow diagram of study selection.

Specifically, four studies[20,23] displayed a risk of bias categorized as 'some concerns', one study[21] indicated a low risk of bias, and one study was identified as having a high risk of bias.[22] The GRADE protocol was used to assess the certainty of the evidence (Table 4). The effect evaluates of CRP and TNF-α were regarded as moderate quality. The evidence for IL-6 was downgraded to low quality for serious heterogeneity and imprecision. The overall quality of the body of evidence of the present systematic review and meta-analysis was regarded as moderate.

Discussion

This meta-analysis aimed to examine the influence of chia consumption on three inflammatory markers: CRP, IL-6, and TNF-α in individuals with type 2 diabetes mellitus or overweight. The analysis involved four studies with 236 participants for CRP, two studies with 132 participants for IL-6, and two studies with 132 participants for TNF-α. The analysis of the effects of chia supplementation on CRP in patients with type 2 diabetes mellitus and overweight individuals revealed significant CRP reduction, particularly in those with a 12-week or longer intervention and an intervention dosage of over 35 grams per day. This suggests that the full effects of chia products might require a longer duration and higher doses to become apparent. The analysis did not reveal a significant effect of chia on IL-6 and TNF-α. Subgroup analysis also confirmed the lack of significance in all subgroups for TNF-α. This suggests that chia supplementation may not have a significant impact on these specific inflammatory markers in the studied populations. These results of the meta-analysis are consistent with some earlier studies that have suggested the consumption of chia may potentially reduce inflammation. For instance, a study by Vuksan *et al.*[24] reported that Salba-chia intervention reduced inflammatory factors like hs-CRP levels in overweight and obese adults with type 2 diabetes. Furthermore, in another study by Vuksan,[25] 12-week dietary supplementation with the novel whole grain Salba (*Salvia hispanica L.*) was associated with decreased hs-CRP level and TNF-α but not IL6 in individuals with T2DM. While Nieman *et al.*,[26,27] indicated that inflammation did not differ between chia seed (whole or milled) and placebo groups in overweight adults. Furthermore, Nikpayam *et al.*[14] revealed that supplementation of chia did not significantly affect hs-CRP and TNF-α. However, the significant variation seen among studies, possibly due to differences in study populations, methodologies, and treatment protocols, could explain some of the inconsistencies. In addition, Teoh *et al.* indicated that participants who consumed chia seed showed no significant difference in any of the inflammation markers compared to the control group.[13] However, the study population was more diverse than our study, which could potentially undermine the results. The varying outcomes imply that the impact of consuming chia seeds may be influenced by contextual factors and may differ depending on an individual's health condition and other related factors. Moreover, investigating the possible mechanisms that underlie the impact of chia seed consumption on various health outcomes could be valuable. For instance, chia seeds contain high levels of dietary fibre and omega-3 fatty acids, which could play a role in their potential health advantages. Conducting additional studies on the biological pathways by which chia seeds may influence inflammation, weight loss, and disease risk factors could provide insights into their potential health benefits and offer guidance for future interventions.

Table 2. Characteristic of included studies in meta-analysis

studies	Country	Study Design	Participant	Sample size and Sex	Sample size		Trial Duration (Week)	Means Age		Means BMI		chia dose (g/d)
					IG	CG		IG	CG	IG	CG	
Nieman *et al.* 2009(A)	USA	parallel, R, PC, SB	overweight adults	M:28	14	14	12	NR	NR	NR	NR	50
Nieman *et al.* 2009(B)	USA	parallel, R, PC, SB	overweight adults	F:48	25	23	12	NR	NR	NR	NR	50
Nieman *et al.* 2012(A)	USA	parallel, R, PC, DB	overweight women	F:42	16	26	10	60.4 ± 1.6	58.5 ± 1.1	32.9 ± 1.3	33.1 ± 0.9	25
Nieman *et al.* 2012(B)	USA	parallel, R, PC, DB	overweight women	F:40	14	26	10	57.2 ± 1.7	58.5 ± 1.1	33.5 ± 1.5	33.1 ± 0.9	25
Vuksan *et al.* 2017	Canada	parallel, R, PC, DB	overweight and obese patients with type 2 diabetes	M/F (F:40, M:18)	27	31	24	60 ± 2	60 ± 2	31 ± 0.9	30.7 ± 0.7	50
Vuksan *et al.* 2007	Canada	Cross-over, R, PC, SB	T2DM	M/F (F:9, M:11)	10	10	12	64 ± 8	64 ± 8	28 ± 4	28 ± 4	37

Abbreviations: IG, intervention group; CG, control group; DB, double-blinded; SB, single-blinded; PC, placebo-controlled; R, randomized; NR, not reported; F, Female; M, Male; NR, not reported.

Moreover, a sensitivity analysis was carried out to determine the influence of each study on the overall effect size, and the results highlight the effect of removing two studies conducted by Vuksan *et al.* in 2007[25] and 2017[24] on the overall effect size of chia on CRP. So, it is recommended to approach the results of this meta-analysis with prudence, and further research is necessary to validate these outcomes.

There are mechanisms underlying the effects of chia on inflammation markers. Chia seeds contain high amounts of omega-3 fatty acids, specifically alpha-linolenic acid (ALA), which serves as a precursor to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the two most extensively researched and recognized omega-3 fatty acids.[28] Apart from omega-3 fatty acids and fibre, chia seeds also comprise polyphenols, which are plant-based substances possessing antioxidant and anti-inflammatory characteristics. Studies have demonstrated that polyphenols present in chia seeds, including caffeic acid and chlorogenic acid, possess anti-inflammatory properties by decreasing the production of pro-inflammatory cytokines such as TNF and IL-6.[29] Polyphenols possess the capability to scavenge free radicals, which are molecules that can inflict harm on cells and lead to inflammation and chronic illnesses.[30] Their capacity to improve blood sugar control and insulin sensitivity, both of which are linked to reduced levels of inflammation in the body, is one-way chia seeds might impact inflammation markers such as TNF, CRP, and IL-6.[31]

One explanation for the significant decrease in CRP but not in IL-6 and TNF-α could be the different biological pathways and sensitivities of these markers to dietary interventions. CRP is an acute-phase protein that responds rapidly to inflammation and may be more sensitive to dietary changes, whereas IL-6 and TNF-α are cytokines involved in chronic inflammation and might require more specific or potent interventions to show changes.[32,33]

This meta-analysis provides a comprehensive overview of the potential anti-inflammatory effects of chia seeds, integrating data from multiple randomized controlled trials. The study's systematic approach and sensitivity analysis enhance the reliability of the findings, despite the limited number of included studies.

According to the assessment using the RoB2 tool criteria, the majority of the trials showed some concerns regarding their quality. This suggests that certain aspects of these studies raised potential biases or methodological limitations. The results revealed that four studies exhibited some concerns regarding bias, implying potential limitations or biases that could influence the reliability of their findings. On the other hand, one study demonstrated a low risk of bias, suggesting a relatively higher methodological quality. In contrast, one study was found to have a high risk of bias, indicating significant methodological limitations or biases that may impact the accuracy of its results. These findings highlight the importance of considering potential biases and limitations when interpreting the overall results of studies investigating the effects of chia on inflammatory markers. It is crucial for future research to address these methodological concerns to enhance the quality and validity of evidence in this field.

Our study was limited by the number of eligible studies, which may limit the generalizability of the findings. Also, the majority of the trials showed some concerns regarding their quality. Furthermore, the included studies varied in terms of sample size, intervention duration, and population characteristics, which may

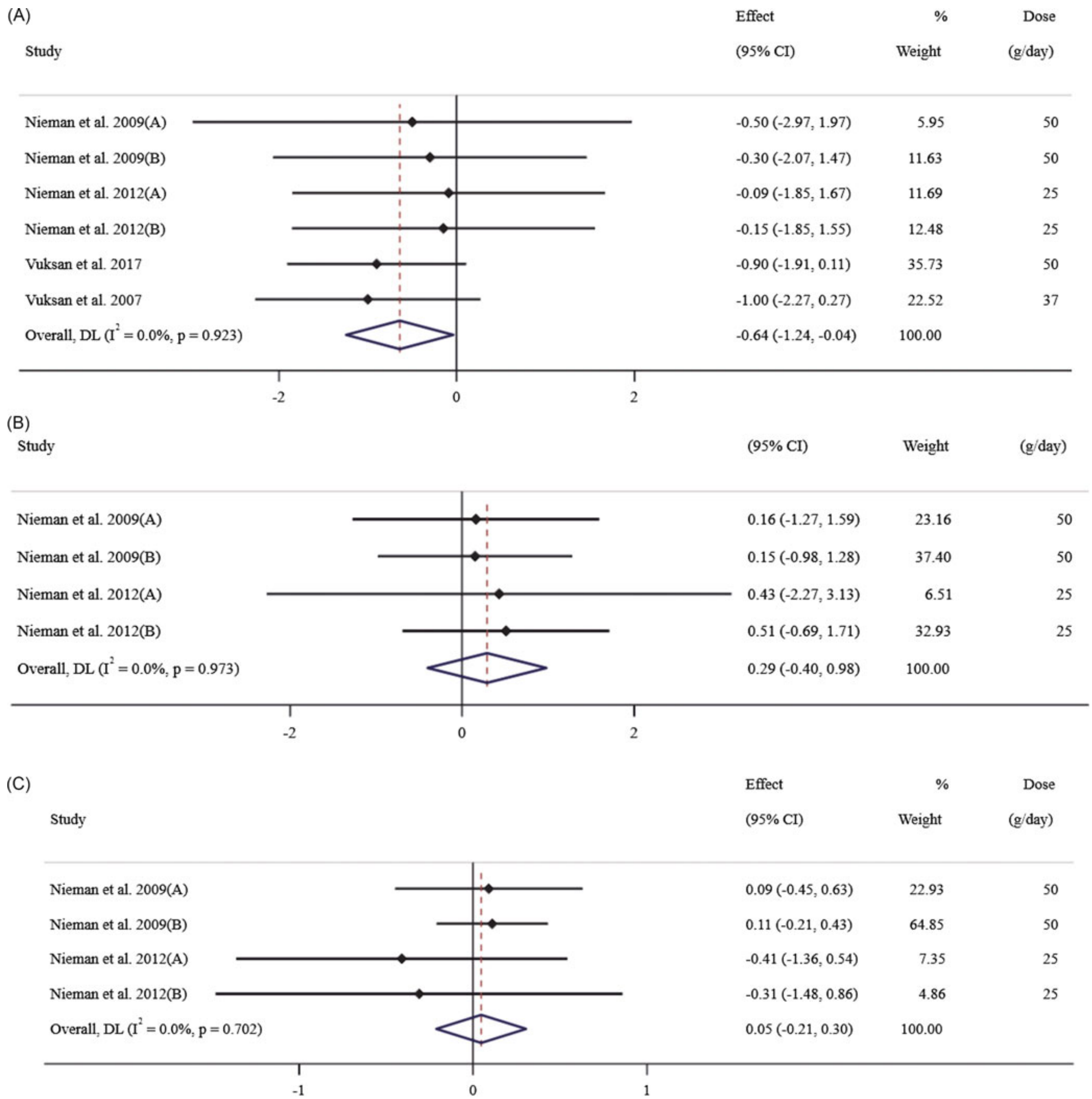

Figure 2. Forest plot for the effects of chia seed ingestion on CRP (A), IL-6 (B), and TNF-α (C).

limit the comparability of the results. However, the certainty of the evidence was classified as low to moderate, which means that the true effect of chia on inflammatory markers is uncertain.

Conclusion

This meta-analysis suggests that chia seed consumption may have a positive impact on reducing CRP levels, but does not show significant effects on IL-6 and TNF-α levels. Due to the limited number of studies and the overall low quality of evidence, several key areas for future research are highlighted. It is essential to conduct studies with larger sample sizes to enhance statistical power and reliability. Additionally, addressing the methodological limitations and potential biases identified through the RoB2 tool is crucial for improving evidence quality. Future research should also focus on determining the optimal dosage and duration of chia seed consumption to better understand its impact on inflammation. While chia seeds may offer some anti-inflammatory benefits, the current evidence is insufficient for definitive conclusions. Further investigation is needed to clarify the efficacy of chia seeds and elucidate the mechanisms behind their effects on inflammation.

Table 3. Subgroup analyses of chia on FBS, Insulin, and HbA1c in adults

				heterogeneity		
	NO	WMD (95% CI)	P within group	P heterogeneity	I^2	P between subgroups
Subgroup analyses of chia on CRP						
Overall effect	6	−0.64 (−1.24, −0.04)	**0.03**	0.92	0%	
Trial duration (week)						
≥12	4	−0.81 (−1.50, −0.11)	**0.02**	0.91	0%	0.34
<12	2	−0.12 (−1.35, 1.10)	0.84	0.96	0%	
Intervention dose (g/day)						
>35	4	−0.81 (−1.50, −0.11)	**0.02**	0.9	0%	0.34
≤35	2	−0.12 (−1.35, 1.10)	0.84	0.96	0%	
Health status						
Overweight	4	−0.22 (−1.16, 0.71)	0.63	0.99	0%	0.25
T2DM	2	−0.94 (−1.73, −0.15)	**0.02**	0.90	0%	
Sex						
Both sexes	2	−0.94 (−1.73, −0.15)	**0.02**	0.90	0%	0.50
Male	1	−0.50 (−2.97, 1.97)	0.69	–	0%	
Female	3	−0.18 (−1.19, 0.83)	0.07	0.98	0%	
Subgroup analyses of chia on IL-6						
Overall effect	4	0.29 (−0.40, 0.98)	0.41	0.97	0%	
Trial duration (week)						
≥12	2	0.15 (−0.73, 1.04)	0.73	0.99	0%	0.63
<12	2	0.50 (−0.60, 1.59)	0.37	0.95	0%	
Intervention dose (g/day)						
>35	2	0.15 (−0.73, 1.04)	0.73	0.99	0%	0.63
≤35	2	0.50 (−0.60, 1.59)	0.37	0.95	0%	
Health status						
Overweight	4	0.29 (−0.40, 0.98)	0.41	0.97	0%	
T2DM	0	–	–	–	–	
Sex						
Both sexes	0	–	**–**	–	–	0.84
Male	1	0.16 (−1.27, 1.59)	0.82	–	00%	
Female	3	0.33 (−0.46, 1.11)	0.41	0.91	0%	
Subgroup analyses of chia on TNF-α						
Overall effect	4	0.05 (−0.21, 0.30)	0.72	0.70	0%	
Trial duration (week)						
≥12	2	0.10 (−0.17, 0.38)	0.45	0.95	0%	0.23
<12	2	−0.37 (−1.11, 0.37)	0.32	0.89	0%	
Intervention dose (g/day)						
>35	2	0.10 (−0.17, 0.38)	0.45	0.95	0%	0.23
≤35	2	−0.37 (−1.11, 0.37)	0.32	0.89	0%	
Health status						
Overweight	4	0.05 (−0.21, 0.30)	0.72	0.70	0%	
T2DM	0	–	–	–	–	

(*Continued*)

https://doi.org/10.1017/jns.2024.70 Published online by Cambridge University Press

Table 3. (*Continued*)

	NO	WMD (95% CI)	P within group	heterogeneity		P between subgroups
				P heterogeneity	I^2	
Sex						
Both sexes	0	–	–	–	–	0.85
Male	1	0.09 (−0.45, 0.63)	0.74	–	100%	
Female	3	0.03 (−0.26, 0.33)	0.82	0.50	0%	

Abbreviations: CI, confidence interval; WMD, weighted mean differences; T2D, type 2 diabetes; TC, total cholesterol.

Table 4. GRADE profile of chia on CRP, IL-6, and TNF-α in adults

Quality assessment						Summary of findings		
Outcomes	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Number of intervention/ control	WMD (95%CI)	Quality of evidence
CRP	No serious limitations	serious limitations[a]	No serious limitations	No serious limitations	No serious limitations	106/104	−0.64 (−1.24, −0.04)	irc; Moderate
IL-6	No serious limitations	serious limitations[b]	No serious limitations	serious limitations[c]	No serious limitations	69/63	0.29 (−0.40, 0.98)	irc;irc; Low
TNF-α	No serious limitations	No serious limitations	No serious limitations	serious limitations[c]	No serious limitations	69/63	0.05 (−0.21, 0.30)	irc; Moderate

[a]The test for heterogeneity is significant, and the I^2 is moderate, 53.3%.
[b]The test for heterogeneity is significant, and the I^2 is moderate, 56%.
[c]Values are distributed within opposite direction across studies, CRP; C-Reactive Protein, IL-6; Interleukin-6, TNF-α; Tumour Necrosis Factor-alpha.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/jns.2024.70

Data availability. The data used to support the findings of this study are available from the corresponding author upon request.

Acknowledgements. None.

Authors' contributions. PP and HA designed research; HA and PJ conducted research; SA performed statistical analysis; and PP, PJ, and HA wrote paper. PJ had primary responsibility for final content. All authors have read and approved the final manuscript.

Funding. None.

Competing interests. There is no conflict of interest in this study.

Ethical approval. Ethical approval was not applicable for this systematic review and meta-analysis.

Consent for publication. Not applicable.

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https://doi.org/10.1017/jns.2024.70 Published online by Cambridge University Press

Journal of Nutritional Science

cambridge.org/jns

Testing a proposed mathematical model of weight loss in women enrolled on a commercial weight-loss programme: the LighterLife study

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Research Article

Cite this article: Egan AM, Rayman JF, and Collins AL (2024). Testing a proposed mathematical model of weight loss in women enrolled on a commercial weight-loss programme: the LighterLife study. *Journal of Nutritional Science* **13**: e92, 1–6. doi: 10.1017/jns.2024.85

Received: 22 August 2024
Accepted: 5 November 2024

Keywords:
adaptive thermogenesis; fat-free mass; mathematical modelling; weight loss

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Abstract

Weight loss results in obligatory reductions in energy expenditure (EE) due to loss of metabolically active fat-free mass (FFM). This is accompanied by adaptive reductions (i.e. adaptive thermogenesis) designed to restore energy balance while in an energy crisis. While the '3500-kcal rule' is used to advise weight loss in clinical practice, the assumption that EE remains constant during energy restriction results in a large overestimation of weight loss. Thus, this work proposes a novel method of weight-loss prediction to more accurately account for the dynamic trajectory of EE. A mathematical model of weight loss was developed using ordinary differential equations relying on simple self-reported inputs of weight and energy intake to predict weight loss over a specified time. The model subdivides total daily EE into resting EE, physical activity EE, and diet-induced thermogenesis, modelling obligatory and adaptive changes in each compartment independently. The proposed model was tested and refined using commercial weight-loss data from participants enrolled on a very low-energy total-diet replacement programme (LighterLife UK, Essex). Mathematical modelling predicted post-intervention weight loss within 0.75% (1.07 kg) of that observed in females with overweight or obesity. Short-term weight loss was consistently underestimated, likely due to considerable FFM reductions reported on the onset of weight loss. The best model agreement was observed from 6 to 9 weeks where the predicted end-weight was within 0.35 kg of that observed. The proposed mathematical model simulated rapid weight loss with reasonable accuracy. Incorporated terms for energy partitioning and adaptive thermogenesis allow us to easily account for dynamic changes in EE, supporting the potential use of such a model in clinical practice.

Introduction

The energy balance (EB) principle, that is, the first law of thermodynamics, states that energy can be neither created nor destroyed, but only transformed. The human body is considered an open system, where energy (kcal) is added in the form of food ('calories in') and transformed to combustion to produce heat ('calories out')[1]. Thus, EB represents the relationship between energy intake (EI) and energy expenditure (EE). Any imbalance between EI and EE results in a shift in energy stores and a subsequent change in weight as fat mass (FM) and fat-free mass (FFM).

The EB equation provides the basis of weight-loss strategies currently implemented in clinical practice. The '3500-kcal rule' is a guidance used in clinical weight management that advises that a reduction of 500 kcal/d will result in 1 lb (~0.5 kg) weight loss per week[2]. This approach relies on the assumption that FFM accounts for 25% of total weight loss with the remaining 75% lost as FM. This represents a 'static model of weight loss', where weight is lost at a fixed rate, decreasing in a linear manner during periods of dynamic weight loss[3].

However, while the principle of EB appears straightforward, quantifying energy imbalance during periods of energy restriction is much more complex. The first inaccuracy of the 3500-kcal rule lies in the assumption that the proportion of weight lost as FM and FFM remains constant during the weight-loss phase[2]. In actuality, the fraction of weight lost as either component changes during the weight-loss phase ranges between 20–40% for FFM and 60–80% FM, respectively[4], resulting in dynamic shifts in energy imbalance.

Second, while traditionally assumed to be independent variables, the components of EB, that is, EI and EE, are in fact functionally interdependent, with changes in one side of the equation corresponding with alterations in the other side[5,6]. The evidence is very clear that if you restrict calories, EE will decline simultaneously[7]. This typically occurs in two ways: (i) obligatory reductions in EE resulting from a loss of metabolic tissue and (ii) adaptive reductions in EE resulting from 'adaptive thermogenesis' (AT), defined as the underfeeding-associated fall in resting energy expenditure (REE) independent of changes in FFM and FM[8,9].

Thus, a 500-kcal deficit per day does not directly translate into 1 lb (0.5 kg) weight loss as traditionally assumed, resulting in less-than-predicted weight loss[10,11].

To account for such shifts in EB during the weight-loss phase, various mathematical models[12] (also referred to as dynamic models) have been developed based on the EB principle, simulating the non-linear nature of weight loss by modelling the dynamic changes in EE resulting from FFM loss and AT. Such models vary in complexity depending on (i) the way EE is subdivided and (ii) the way body mass is compartmentalised. While accurate models do exist, they often require complex parameters that are unobtainable in a clinical setting, often limited to simple demographics (e.g. gender and age) and anthropometric measures (e.g. height, weight, waist circumference).

The aim of this study is to produce a working mathematical model of weight loss requiring only simple baseline parameters to more accurately describe the dynamic trajectory of weight loss over a given time. Subsequently, the model will be tested and refined using a large database of female clients enrolled on a commercial weight management programme (LighterLife, Essex, United Kingdom).

Methods

The present study describes a mathematical model of weight loss developed based on estimates of the components of EB and how these change during periods of weight loss. Values used in the development of the model are determined based on existing observational weight-loss data.

Model development

The proposed mathematical model uses baseline inputs of weight (kg), EI (kcal), and physical activity level (PAL) to predict weight loss over a specified time. The model subdivides total daily energy expenditure into resting energy expenditure (REE), physical activity energy expenditure (PAEE), and diet-induced thermogenesis (DIT), modelling each compartment independently. An energy conversion of 7700 kcal per kg is assumed to convert energy deficit to weight loss as described by Equation 1:

$$\frac{dw}{dt} = \frac{1}{7700}[EI - (REE + PAEE + DIT)]$$

Equation 1: Simple equation for weight loss. $\frac{dw}{dt}$ represents the rate of weight change over a given time. Weight loss is assumed as the discrepancy between EI and EE. EI is energy intake in kcal/d. Energy expenditure is subdivided into REE, PAEE, and DIT. REE is resting energy expenditure in kcal/d, PAEE is physical activity energy expenditure in kcal/d, and DIT is diet-induced thermogenesis in kcal/d. An energy conversion of 7700 kcal/kg is assumed to convert energy deficit to weight loss.

Energy intake and energy expenditure

EI (kcal/d) is used as a model input and is assumed true to that reported. REE (kcal/d) is estimated using the Cunningham Equation based on FFM[13]. Baseline FFM% is estimated using gender-specific expressions developed by our group as described by Equation 2:

$$FFM(\%)_{males} = \frac{(0.25w_0 + 43.5)}{w_0}$$

$$FFM(\%)_{females} = \frac{(0.25w_0 + 24.9)}{w_0}$$

Equation 2: Predictive equations for starting FFM (%). A linear relationship between starting body weight and FFM (kg) was assumed where starting values for FFM of a lean male and female were obtained from the literature and excess body weight was assumed to be composed of 75% FM and 25% FFM. This value was divided by starting body weight, allowing us to predict baseline FFM (%). *FFM*, fat-free mass; *FM*, fat mass; w_0, weight (kg).

PAL index[14] is used to account for PAEE (kcal/d), where TEE is calculated as REE multiplied by PAL (TEE = REE x PAL). Values range from 1.2 (chair-bound/bed-bound) to 2.4 (strenuous work or highly active leisure) and are self-reported to most accurately reflect the individual's lifestyle. DIT (kcal/d) is assumed a direct product of EI and is estimated using a coefficient of 10% in a lean population and 5% in an overweight/obese population[15].

Obligatory changes in energy expenditure

FFM is modelled as a function of weight loss. An exponential increase in FFM% is assumed on the onset of weight loss, persisting until an overall weight loss of 10% is achieved at which point FFM % stabilises approximately 10 percentage points higher than baseline. The predicted value for FFM% is multiplied by body weight at any given time to produce a predicted value for FFM (kg), as described by Equation 3:

$$FFM\ (kg) = \left(c + 0.1\left(1 - e^{-w_0 + w(t)}\right)\right)w(t)$$

Equation 3: Original expression for FFM change.

FFM, fat-free mass; *c*, baseline fat-free mass (%); w_0, starting weight; $w(t)$, weight at any given time.

Adaptive changes in energy expenditure

AT is modelled as a function of weight loss. An exponential increase in AT is assumed during the weight-loss phase, persisting until a total weight loss of 10% is achieved, at which point AT stabilises at a value equivalent to 15% of REE[16,17] as described by Equation 4:

$$AT\ (\%) = 0.15\left(1 - e^{-70\left(1 - \frac{w(t)}{w_0}\right)}\right)$$

Equation 4: Expression for adaptive thermogenesis.

AT, adaptive thermogenesis; w_0, starting weight; $w(t)$, weight at any given time.

Using the expressions above, a mathematical model of weight loss was assembled and defined as an ordinary differential equation as described by Equation 5:

$$\frac{dw}{dt} = \frac{1}{7700}\left(EI - PAL\left(1 - 0.15\left(1 - e^{-70\left(1 - \frac{w(t)}{w_0}\right)}\right)\right)\left(22.8\left(\left(c + 0.1\left(1 - e^{-w_0 + w(t)}\right)\right)w(t) + 484\right) - \beta(EI)\right)\right)$$

Equation 5: Original mathematical model of weight loss. $\frac{dw}{dt}$ represents the rate of weight change over a given time, w_0 is the starting weight, and w(t) is the weight at any given time. EI is energy intake in kcal/d. PAL is physical activity level, and a value of 1.6 is assumed representing a moderately active lifestyle[14]. This value is multiplied by REE to account for PAEE. REE is estimated using the Cunningham equation[13] based on FFM, adjusted for AT. An exponential increase in AT is assumed during the weight-loss phase, persisting until a total weight loss of 10% is achieved, at which point AT stabilises at a value equivalent to 15% of REE. The parameter c represents FFM% which is estimated using gender-specific expressions developed by our group and multiplied by w(t) to produce a value for FFM (kg). An exponential increase in FFM% is assumed during the weight-loss phase, persisting until a total weight loss of 10% is achieved at which point FFM% stabilises approximately 10 percentage points higher than baseline. The parameter β represents DIT coefficient which is assumed a value of 0.05 in an overweight/obese population[18,19]. This value is multiplied by EI to account for DIT. An energy conversion of 7700 kcal per kg is assumed to convert energy deficit to weight loss.

Model validation

The proposed mathematical model is tested using weight-loss data from a large retrospective database of female clients provided by LighterLife UK Ltd (LL; Essex, UK). Relevant data was extracted from the client database and anonymised by LL personnel prior to sharing with the study investigators for analysis.

Dietary intervention: LL is a commercial weight management company that offers a very low-energy total-diet replacement (TDR) plan. Clients consume four food packs per day (e.g. soups, shakes, pots, and meals) replacing all conventional food, providing 600–800 kcal/d, >50 g protein, 50–75 g carbohydrate, ~17 g fat, 14 g fibre and 22–28 essential micronutrients. Following the active weight-loss phase, clients follow a standardised protocol for food reintroduction where food packs are gradually decreased and replaced with conventional foods. Clients attend weekly group meetings delivered by trained LL counsellors consisting of weigh-ins and optional behaviour support.

Inclusion criteria: Individuals self-referring to a very low-energy TDR programme for 6–12 weeks from the years 2017–2021 were considered eligible for inclusion in the extracted data. Clients were considered ineligible if they had previously enrolled on a LL programme and/or gained weight during the programme. Once an appropriate study population was determined, clients were anonymised by LL personnel using a client ID. The anonymised data were then disseminated to the study investigators for analysis.

Data extraction: Demographic and anthropometric data from LL clients enrolled in TDR programme were extracted. Interventions 6–12 weeks in duration were examined to reflect a typical TDR programme duration. The proposed mathematical model was used to predict:

1.) End-weight and weight loss in LL participants enrolled on VLED programmes of different intervention lengths (6–12 weeks).
2.) End-weight and weight loss at 7-d intervals in a subset of LL participants with sequential weight measures.

Mean error, that is, the mean difference between actual and predicted end-weight expressed as a percentage of actual end-weight, was determined post-intervention and at weekly intervals.

$$Mean\ error(\delta) = \frac{\text{actual} - \text{predicted end weight}}{\text{actual end weight}} \times 100$$

Modelling energy intake: In the LL cohort, a step function was used to describe EI, where a one-step increase in kcal intake was assumed from week 10 to account for those in the food reintroduction phase and due to expected deviation from dietary prescription in the later stages of the intervention as described by Equation 6:

$$\text{EI} = \begin{cases} 800 & t < 70 \\ 1200 & \text{otherwise} \end{cases}$$

Equation 6: Step function for EI consisting of two intervals of different constant values with a jump between the horizontal line segments. For dieting durations <10 weeks, an intake of 800 kcal/d is assumed. For dieting durations >10 weeks, an intake of 1200 kcal/d is assumed. *EI*, energy intake; *t*, time.

Statistical analysis: Data are presented as mean and standard deviation. The Shapiro–Wilk test was used to test the normality of the data. A two-way ANOVA multiple comparisons test was performed to evaluate differences in actual weight loss vs. predicted weight loss across several timepoints. A one-way ANOVA or non-parametric Kruskal–Wallis test was performed to evaluate weekly weight changes. *P*-value <0.05 was considered as statistically significant.

Results

A total of 983 females were included in the present analysis. Clients were on average 47 years old with a mean body weight of 93.31 ± 19.18 kg and BMI of 34.51 ± 6.52 kg/m^2. The largest proportion of individuals enrolled in the programme for 8 weeks (n = 175), and the smallest proportion enrolled for 12 weeks (n = 84). The mean intervention length was 9 weeks.

Prediction of post-interventional weight loss

Across all intervention lengths, an overall mean weight loss of 7.90 ± 4.63 kg was observed. On average, participants lost 8.45 ± 4.55% of their starting body weight. Our mathematical model was associated with an average mean error of 0.75 ± 5.12% equivalent to 1.07 ± 4.52 kg. The best agreement between actual and predicted end-weight was observed in those enrolled on a 6-week intervention, with a mean error of −0.28 ± 4.22% (0.16 ± 3.65 kg). Mean error increased with intervention length, with the largest mean error observed in those enrolled on a 12-week intervention (2.36 ± 6.26% equivalent to 2.45 ± 5.57 kg). Table 1 summarises the post-intervention mean error of our mathematical model across all timepoints.

Prediction of weekly weight loss

Total weight loss increased with dieting duration (r = 0.96) ranging from 2.28 ± 1.56 kg at week 1 to 9.55 ± 4.92 at week 11. In contrast, a negative correlation was observed between the rate of weight loss and dieting duration (r = 0.75), decreasing from 2.28 ± 1.56 kg/week at week 1 to 0.74 ± 5.13 kg/week at week 12. Absolute weight loss (P <0.0001) and rate of weight loss (P < 0.0001) changed significantly over time as determined by one-way ANOVA.

Figure 1 summarises the mean error of our model at weekly intervals for 12 weeks ranging from −1.44 ± 2.52% (−1.13 ± 2.14 kg)

https://doi.org/10.1017/jns.2024.85 Published online by Cambridge University Press

Table 1. Post-intervention mean error of mathematical model in the LighterLife study

Weeks	n	δ
6	193	−0.28 (4.22)
7	181	0.59 (4.37)
8	196	0.40 (4.70)
9	158	0.47 (4.93)
10	155	1.09 (6.03)
11	133	1.75 (5.72)
12	93	2.36 (6.26)
Overall	1109	0.75 (5.12)

Data presented as mean (SD). Participants followed a very low-energy total-diet replacement intervention for 6–12 weeks providing 600–800 kcal/d. End-weight was predicted by our mathematical model and compared to that observed in LighterLife participants. δ, mean error ([actual-predicted end-weight/actual end-weight] × 100).

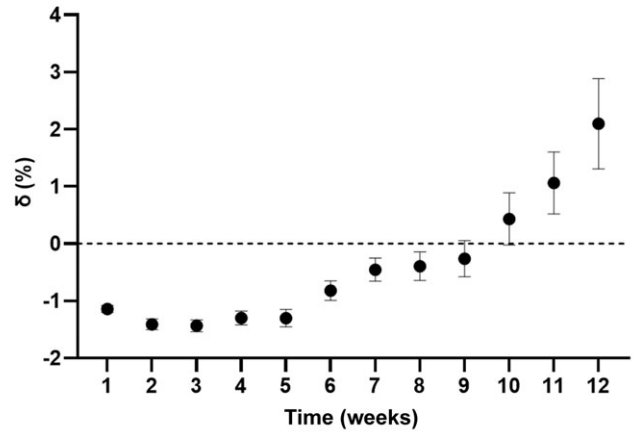

Fig. 1. Mean error of mathematical model at weekly intervals. Data presented as mean (SEM). End-weight in LighterLife participants was predicted by our mathematical model and compared to that observed. Week 1, −1.14 ± 1.68%, n = 713; week 2, −1.41 ± 2.4%, n = 656; week 3, −1.44 ± 2.5%, n = 606; week 4, −1.30 ± 2.93%, n = 599; week 5, −1.30 ± 3.64%, n = 565; week 6, −1.82 ± 4.09%, n = 578; week 7, −0.46 ± 4.4%, n = 465; week 8, −0.40 ± 4.91%, n = 388; week 9, −0.26 ± 5.27%, n = 277; week 10, 0.43 ± 6.28%, n = 190; week 11, 1.06 ± 5.96%, n = 121; week 12, 2.10 ± 6.07%, n = 59. δ, mean error ([actual-predicted end-weight/actual end-weight] × 100).

at week 3 to 2.09 ± 6.07% (2.24 ± 5.39 kg) at week 12. The best agreement was observed in 7–9-week interventions, predicting end-weight within 0.50% (<0.50 kg) of that observed.

When translated into predicted weight loss, our model underestimated weight loss in the first 7 weeks of the intervention and overestimated weight loss from week 8 onwards (Table 2).

Actual weight loss can be compared with predicted weight loss using the Bland and Altman method as illustrated in Fig. 2. Mean bias between actual weight loss and weight loss predicted by mathematical modelling was 0.61 ± 3.19 kg. The 95% confidence interval for the difference was −5.65–6.86 kg.

Discussion

In the present study, a proposed mathematical predicted post-intervention end-weight within 0.75% of that observed in females enrolled on a 6–12 weeks TDR intervention, reflecting a mean weight-loss overestimation of 1.07 kg. Mean error increased with intervention length, from −0.28%, equivalent to 0.16 kg in 6-week interventions, to 2.36% equivalent to 2.45 kg in 12-week interventions.

The extraction of weekly weights allowed us to determine where our mathematical model was most and least accurate. Findings showed that our model underestimated weight loss in the first 6 weeks of the intervention, particularly in the first week where weight loss was 75% greater than that predicted. This is likely due to the rapid depletion of glycogen pools and associated water and electrolytes (stored in a proportion of 1:3 g, respectively) in the first 7–10 d equivalent to a total weight loss of 1.5–2.0 kg[1,20–22].

Alternatively, mathematical modelling overestimated weight loss beyond week 8, possibly due to relaxed compliance in those enrolled on the programme for longer durations.

While data on the number of TDR products purchased per week were provided, this may not necessarily equate to TDR products consumed, particularly in participants enrolled on the intervention for over 8 weeks who may be using partial meal replacement or transitioning to a weight maintenance phase. Furthermore, the frequency of diet relapse is likely to increase with intervention length, particularly in those not participating in a gradual food reintroduction process, where rapid carbohydrate refeeding can result in a significant surge in hunger hormones and appetite. It is noteworthy that only 8.5% of the study population enrolled on the programme for 12 weeks, meaning relaxed compliance in one given participant will significantly impact mean weight loss.

Overall, our model was associated with a mean error of 0.60%, reflecting an average weight-loss overestimation of 0.27 kg. The best agreement between actual and predicted weight loss was observed from weeks 6–9 (ns $p > 0.3827$), representing the intervention length of ~48% of study participants, where predicted end-weight was within 0.30% (0.35 kg) of the observed. Furthermore, this range encompasses the mean intervention length of 8.7 weeks suggesting our model can produce intervention end-weight with reasonable accuracy. Despite this, our weekly analysis identifies points of the weight-loss phase where our model requires further refinement.

Based on present findings, the following refinements were applied to our mathematical model: (i) new predictor equation for REE: REE is estimated using the Mifflin equation based on FFM[23], developed in a population with both lean and obese individuals, thus more reflective of our study population, and (ii) new formulation for FFM: FFM is modelled as a function of weight loss. A linear increase in FFM% is assumed during the weight-loss phase, persisting until a total weight loss of 20% at which point FFM% stabilises approximately 10 percentage points higher than baseline, as described by Equation 7:

$$FFM\ (kg) \;=\; \left(\left\{ \begin{array}{cc} c + 0.1\,\frac{w_0 - w(t)}{0.2 w_0} & < 20\%\ weight\ loss \\ c + 0.1 & otherwise \end{array} \right\} \right) w(t)$$

Equation 7: Refined expression for FFM change.

FFM, fat-free mass; *c*, baseline fat-free mass (%); w_0, starting weight; *w(t)*, weight at any given time.

Strengths and limitations

The proposed model requires only simple inputs of EI and PAL and thus can be used without expensive lab equipment. While several mathematical weight-loss models do exist, few rely only on

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Table 2. Actual versus predicted weight loss at weekly intervals in females enrolled on the LighterLife study

Week	*n*	Predicted weight loss (kg)	Actual weight loss (kg)	Difference (kg)	*P*-value
1	713	1.29 (0.42)	2.28 (1.56)	−0.99 (1.49)	<0.001
2	656	2.55 (0.84)	3.70 (2.03)	−1.15 (1.97)	<0.001
3	606	3.60 (1.20)	4.73 (2.31)	−1.13 (2.14)	<0.001
4	599	4.64 (1.59)	5.58 (2.65)	−0.95 (2.54)	<0.001
5	565	5.61 (1.97)	6.46 (3.17)	−0.85 (3.09)	<0.001
6	578	6.64 (2.38)	6.97 (3.43)	−0.34 (3.52)	ns
7	465	7.56 (2.64)	7.57 (3.73)	−0.01 (3.81)	ns
8	388	8.44 (3.09)	8.34 (4.26)	0.10 (4.20)	ns
9	277	9.64 (3.39)	9.39 (4.63)	0.24 (4.57)	ns
10	190	10.41 (3.63)	9.47 (5.52)	0.93 (5.41)	0.0092
11	121	10.97 (3.43)	9.55 (4.92)	1.42 (5.12)	0.0006
12	59	11.18 (3.85)	8.94 (5.13)	2.24 (5.39)	<0.001

Data presented as mean (SD). Participants followed a very low-energy total-diet replacement intervention for 6–12 weeks providing 600–800 kcal/d. Weekly weight loss in LighterLife participants was predicted by our mathematical model and compared to that observed. The statistical significance of differences between actual and predicted weight loss was assessed using paired *t*-tests.

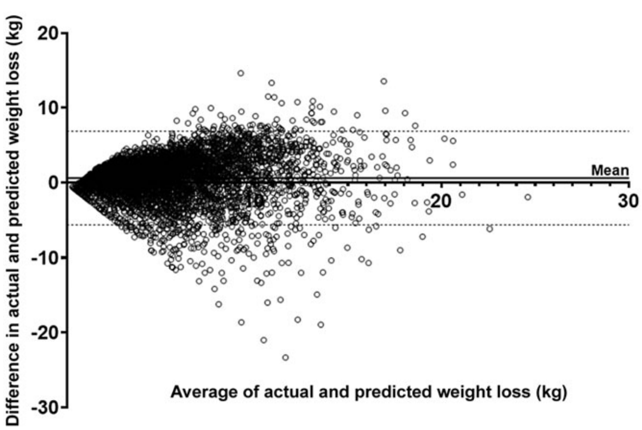

Fig. 2. Bland–Altman analysis to investigate the agreement between actual and predicted weight loss. Participants followed a very low-energy total-diet replacement intervention for 6–12 weeks providing 600–800 kcal/d. Average = (actual weight loss + predicted weight loss)/2. The dotted lines represent the upper and lower limits of agreement (± 2 SD). The solid line represents the average difference (kg).

clinically available anthropometric and demographic variables that are readily available in a clinical environment. Such a model may be used for (i) prescribing dietary intake in terms of energy deficit, (ii) setting target weights and timescales, (iii) monitoring dietary compliance, and (iv) patient/client motivation and self-management.

A key strength of the study is a large sample size strengthening the validity and reliability of our results. Individuals are self-referring and self-funding clients and thus are likely to be motivated and may exhibit higher levels of adherence.

The present study represents the nature of weight loss in free-living individuals, rather than study participants participating in a tightly regulated clinical trial. As such, the proposed mathematical model can be used to identify those deviating from the expected weight-loss trajectory who may require additional input. However, the accuracy of the proposed model is limited by the reliance on assumptions regarding EI and EE implemented to improve model applicability.

To quantify one side of the EB equation, that is, EE, it is important to keep the other side (EI) is as controlled as possible. Thus, to minimise potential error, our model is validated using weight-loss data from a TDR programme, using specially formulated food products of known energy content. If used as directed, TDR products are an accurate method of estimating EI.

However, it should be considered that there is likely much less variation in EI using TDR products than would be using food-based weight-loss programmes. Thus, it remains unclear if similar accuracy can be observed in food-based dietary interventions.

Predictor equations are developed using linear regression analysis which assumes a proportional increase in REE with increasing body weight. However, in morbidly obese subjects, excess weight is predominately FM rather than metabolic tissue. Furthermore, excess FFM is predominantly low-metabolic skeletal muscle rather than high-metabolic organs, for example, the brain, liver, and kidney[24,25]. Thus, REE increases at a slower rate in a more curvilinear manner[26–28] rather than a linear increase as assumed by most predictive equations[24].

Our model includes a term for AT based on observations from clinical weight-loss studies. While longitudinal studies support the existence of AT, significant between-individual variance is observed[3]. AT is suggested to be biologically determined, whereby two existing phenotypes ('thrifty' and 'spendthrift') differ in their capacities to regulate EE in response to altered energy availability[7,29]. Subsequently, it should be considered that the term for AT assumed by our model may not be representative of all study participants. The proposed model assumes an immediate adaptive response to energy deficit. However, the onset time for AT is a matter of debate with a considerable amount of research suggesting AT takes weeks to develop[15].

Finally, the proposed model relies on the assumption that those purchasing over twenty-eight products per week are following the prescribed four products per day. Additional intake from conventional foods was not recorded and therefore our model relies on assumptions regarding programme adherence. Thus, despite the prescriptive nature of TDR, true values for EI remain uncertain.

Conclusion

Mathematical models have been used to simulate EB and weight dynamics for decades, yet application in clinical weight loss remains limited. The model proposed in the present study estimates energy deficit by simulating dynamic changes in EE using simple baseline parameters of gender, weight, and EI. In a large cohort of weight-reducing females, the proposed model was associated with a mean bias of 0.61 kg. Limitations of model assumptions need to be examined before clinical application; however, there is no doubt that mathematical modelling has a valuable place in the treatment of overweight and obesity.

Acknowledgements. We wish to express our gratitude to Prof Margaret Rayman, University of Surrey, for providing invaluable writing assistance and for reviewing and proofreading the manuscript. We would like to acknowledge Dr Kelly Johnston for offering her valuable input in the planning and development of the project. Finally, we would like to thank the team at LighterLife UK Ltd for their collaboration and financial support.

Financial support. This work was supported by the University of Surrey Doctoral College Studentship Award and LighterLife UK Ltd.

Declaration of Interests. None.

Authorship. AME and ALC designed and implemented the research. AME and JFR developed all expressions and functions and performed all calculations relating to the mathematical model. AME and ALC acquired funding and obtained the commercial data. AME processed the data and analysed and interpreted the results. AME drafted the manuscript with inputs from ALC and JFR.

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