


Mental health consequences of dietary restriction: increased depressive symptoms in biological men and populations with elevated BMI

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To cite: Menniti G, Meshkat S, Lin Q, *et al.* Mental health consequences of dietary restriction: increased depressive symptoms in biological men and populations with elevated BMI. *BMJ Nutrition, Prevention & Health* 2025;**0**. doi:10.1136/bmjnp-2025-001167

► Additional supplemental material is published online only. To view, please visit the journal online (<https://doi.org/10.1136/bmjnp-2025-001167>).

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Received 3 January 2025
Accepted 18 March 2025



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ABSTRACT

Introduction The literature primarily examines the mental health effects of dietary patterns, with ‘healthy’ diets linked to fewer depressive symptoms, although no standardised definition of a ‘healthy’ diet exists. Many individuals adopt restrictive diets such as caloric or nutrient restriction or medically prescribed patterns (eg, diabetic diets) to improve health, yet their impact on depressive symptoms remains understudied. This study aims to evaluate the association between restrictive dietary patterns and depressive symptoms stratified by sex and body mass index (BMI).

Methods A cross-sectional study was performed using the National Health and Nutrition Examination Survey (NHANES) 2007–2018. Adults who completed dietary assessments and the Patient Health Questionnaire-9 (PHQ-9) for depressive symptom severity were included. Statistical analyses were performed using R. Multivariable linear regression was used to examine associations, and interaction effects were explored by including BMI or sex, with subgroup analysis performed when appropriate.

Results The study included 28 525 adults, of whom 7.79% reported depressive symptoms. Compared with individuals not following a specific diet, those adhering to calorie-restrictive diets had a 0.29 point increase in PHQ-9 scores (95% CI 0.06 to 0.52). Among overweight individuals, calorie-restricted diets were associated with a 0.46 point increase (95% CI 0.02 to 0.89) and nutrient-restricted diet was associated with a 0.61 point increase (95% CI 0.13 to 1.10) in PHQ-9 scores. Men who followed any diet showed higher somatic symptom scores than those not on a diet. Additionally, men on a nutrient-restrictive diet had a 0.40 point increase in cognitive-affective symptom scores (95% CI 0.10 to 0.70) compared with women not following a diet.

Conclusions There are potential implications of widely followed diets on depressive symptoms, and a need for tailored dietary recommendations based on BMI and sex.

INTRODUCTION

Major depression has emerged as a global crisis, with the incidence increasing approximately 50% between 1990 to 2017.¹ Major depressive disorder (MDD) is defined by the Diagnostic and Statistical Manual of Mental

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Diet affects depression risk, with healthy eating patterns associated with reduced symptoms and unhealthy diets linked to increased risk. However, dietary patterns extend beyond a simple ‘healthy’ versus ‘unhealthy’ distinction, as many individuals adopt specific diets for health benefits. Research on the relationship between these dietary patterns and depressive symptoms remains limited.

WHAT THIS STUDY ADDS

⇒ This study adds to the body of knowledge that diets such as low-calorie diets may be associated with depressive symptoms. Additionally, modifying your diet in a specific way may be associated with worsening depressive symptoms including somatic and/or cognitive-affective symptoms, with specific implications for subgroups such as biological men and those with raised BMI.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study provides insights for dietary recommendations provided by healthcare professionals in terms of considerations regarding risk factors for depression, particularly in biological men or those with raised BMI.

Disorders, Fifth Edition, Text Revision (DSM-5-TR) as at least 2 weeks of ≥5 cognitive-affective symptoms (low mood, anhedonia, feelings of guilt, trouble concentrating and suicidality) and somatic symptoms (sleep disturbances, low energy, appetite changes and psychomotor changes).^{2–4}

While numerous risk factors contribute to depression—including non-modifiable factors like biological sex and age⁵—the role of diet as a modifiable risk factor of depression has only recently received attention.^{5 6} Traditionally, many individuals view their diet as a means to enhance their physical health.⁷ A study by Helland and Nordbotten showed

that better physical health, including weight loss, is the main motivator for individuals to improve their diet.⁷ However, growing evidence underscores the profound influence of diet on mental well-being as well as on physical health.^{6,7}

Numerous studies have consistently focused on ‘healthy’ versus ‘unhealthy’ diets. They have shown that ‘healthy’ diets rich in minimally processed foods, fresh fruits and vegetables, whole grains, nuts, seeds, lean proteins and fish have been linked to a lower risk of depression.^{6,7} In contrast, an ‘unhealthy’ diet dominated by ultra-processed foods, refined carbohydrates, saturated fats, processed meats and sweets is associated with an increased risk of depressive symptoms.^{6,7} Therefore, individuals must adopt a perfectly healthy dietary pattern to reduce the risk of depressive symptoms.^{6,7} This dichotomy fails to capture the complexity of real-world eating habits. Limited research explores the effects of dietary patterns beyond the ‘healthy’ versus ‘unhealthy’ framework on depression. By focusing on idealised healthy diets, studies overlook the diversity of dietary patterns that may be considered healthy or unhealthy, yet are more representative of what the average American realistically follows. Some studies have examined dietary patterns aimed at neuroprotection, such as the Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet and a modified Mediterranean diet, the latter of which was shown to reduce depressive symptoms in an Australian clinical trial.⁸ However, research on real-world healthy dietary patterns and their impact on depression remains limited, despite its importance for broader applicability to the average American.

On the topic of real-world healthy diets, individuals often experiment with various dietary modification strategies in pursuit of a healthier diet. Common approaches include caloric restriction, nutrient-specific restrictions (eg, low sugar or low fat diets) and adopting established dietary patterns designed for specific health conditions (eg, diabetic diet, dietary approaches to stop hypertension or DASH diet).^{9,10} Despite the popularity of these strategies, research on their impact on depression remains sparse and, in some cases, inconclusive.^{11–13}

Recognising this gap, our study aims to compare the influence of different dietary modification strategies on depressive symptoms within a US population. We aim to assess how caloric restriction, nutrient-specific restriction and established dietary patterns are associated with depressive symptoms, while also exploring potential differences when stratified by biological sex and body mass index (BMI). We hypothesise that, because biological men typically have higher caloric needs, restrictive diets may pose unique challenges that influence mood differently compared with biological women.¹³ Moreover, preclinical studies indicate sexual dimorphisms in behavioural outcomes following consumption of obesogenic diets, which show anxiogenic effects in male but not female rats.^{14,15} Clinical studies have also shown that the association between restrictive diets and depressive symptoms

is more pronounced in overweight/obese individuals.¹² Therefore, we hypothesise that the association between restrictive diets and depressive symptoms varies across BMI groups.

METHODS

Study design and cohort

This cross-sectional study used baseline data from six cycles of the National Health and Nutrition Examination Survey (NHANES) from 2007 to 2018. NHANES was conducted by the Division of Health and Nutrition Examination Surveys within the Centres for Disease Control and Prevention and the National Centre for Health Statistics. It was designed to assess the health and nutritional status of the US population. A complex multi-stage probability sampling design was used to ensure accurate representation of the non-institutionalised US population. Data are released in 2-year cycles, with approximately 10 000 voluntary participants per cycle who provided informed consent. Data collection included the household interview and examinations. More details regarding study design are described elsewhere.^{16,17} The study population was restricted to men and women aged 18 and older who completed both the Mental Health – Depression Screener (DPQ) and the Dietary Interview – Total Nutrient Intakes, First Day (DR1TOT) questionnaires. Participants with a BMI <18.5 kg/m² were excluded due to small sample sizes when stratified by dietary patterns.

Exposure variable

Dietary patterns were assessed as part of the dietary interview. Participants were asked: “(Are you) currently on any kind of diet, either to lose weight or for some other health-related reason?” If the participant responded “Yes”, a follow-up question: “What kind of diet (are you) on?” was asked. Only nine of the 13 diet options were available in all six cycles of NHANES from 2007 to 2018 and were included in the analysis.

To reflect common strategies for modifying a diet, dietary patterns were categorised into four groups: (1) calorie-restricted diets; (2) nutrient-restricted diets; (3) established dietary patterns; and (4) not on a diet. Group 1 (calorie-restricted diets) included all participants who responded “Yes” to a weight loss or low-calorie diet. Group 2 (nutrient-restricted diets) included all participants who responded “Yes” to only a low fat or cholesterol diet, or a sugar-free or low sugar diet, or a low salt or sodium diet, a low fibre diet or a low carbohydrate diet. We used data from 2007 to 2018 to test whether individuals who answered “Yes” to low sugar or low fibre diets were correlated with those on a low carbohydrate diet. The resulting coefficient of 0.11 indicated a weak association, which led us to group low sugar, low fibre and low carbohydrate diets together in Group 2. Group 3 (established dietary patterns) included all participants who responded “Yes” to a diabetic diet or to multiple questions that together insinuated they were following

the DASH diet. If they responded “Yes” to a low fat or cholesterol diet, a low salt or sodium diet or a sugar-free or low sugar diet, they were classified as DASH diet since NHANES did not specifically ask about this dietary pattern.¹⁸ No information was provided by NHANES to participants on what the term ‘diabetic diet’ entails, despite it being a commonly picked response option. Other dietary patterns that ideally would have been included in our investigation are the renal/kidney diet and the celiac/gluten-free diet. These diets were unfortunately not included as a response option in the questionnaire for many cycles until more recently. Nevertheless, very few participants (n=70) self-identified as consuming these diets, and therefore excluding them is unlikely to influence the results given all established dietary patterns were grouped together in this study. For all four groups we ensured that they were mutually exclusive.

Outcome variables

Depressive symptoms were assessed using the validated screening and diagnostic tool for depression known as the Patient Health Questionnaire (PHQ-9). The PHQ-9 includes nine total questions examining specific symptom frequencies across a 2-week period. The PHQ-9 scores range between 0 and 27, where participants with scores ≥ 10 are considered to have clinically meaningful depressive symptoms and those with scores < 10 are considered to have no depressive symptoms.¹⁹ The PHQ-9 also reflects cognitive-affective and somatic depressive symptoms. The cognitive-affective score was calculated by summing the responses to questions 1, 2, 6, 7 and 9, while the somatic score was derived from questions 3, 4, 5 and 8. This segmentation approach enables a detailed exploration of depressive symptom clusters analysed as continuous variables and their potential associations with dietary patterns.

Covariates

The following categorical covariates were also included: age groups (young adults: 18–44, middle-aged adults: 45–64, older adults: 65+); biological sex (male vs female); race/ethnicity (Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, other race including multiracial); educational level (< 9 th grade, 9–11th grade including 12th grade with no diploma, high school graduate/GED or equivalent, some college or AA degree, college graduate or above); marital status (married, widowed, divorced, separated, never married, living with partner); ratio of family income to poverty (PIR; low income < 1 , middle income 1–4, and high income > 4); smoking status (non-smoker vs smoker); drinking status (never drinker, light drinker, moderate drinker, heavy drinker); BMI (healthy weight=18.5–24.9 kg/m², overweight=25–29.9 kg/m², obese ≥ 30 kg/m²); adult food security category (full food security, marginal food security, low food security, very low food security); diabetes mellitus (yes vs no); and hypertension (yes vs no).

Statistical analyses

Statistical analyses were performed using R Studio (v 4.4.0), incorporating the ‘survey’ package to apply survey weights. The analysis used the ID variable *SDMVPSU* and the strata variable *SDMVSTRA*, with MEC survey weights adjusted by dividing *WTMEC2YR* by 6 (*WTMEC2YR*/6) to accommodate the combination of six survey cycles, as suggested by NHANES (<https://wwwn.cdc.gov/nchs/nhanes/tutorials/weighting.aspx>). Survey weights are essential for maintaining the representativeness of study findings to the broader US population. They account for the complex sampling design of NHANES, which includes oversampling of specific subpopulations and adjustments for non-response bias. By incorporating these weights, the estimates remain unbiased and accurately reflect population-level trends. In the table presenting demographic characteristics, unweighted frequencies were generated through the *CreateTableOne* function, while weighted percentages were calculated using the *svyCreateTableOne()* function. A χ^2 test of independence was applied to detect statistically significant differences ($p \leq 0.05$) in demographic characteristics across the various depressive symptom groups.

Multivariable linear regression models were used to assess the relationship between dietary patterns and depressive symptom severity, as well as symptom clusters. Interaction terms for sex and BMI were incorporated to explore potential interactions between these variables and dietary patterns. If significant interactions were found, subgroup models stratified by sex or BMI were analysed. All adjusted models consistently included relevant covariates. Missing survey data were handled using listwise deletion to avoid potential bias or inaccuracies that could arise from imputation, meaning cases with missing values in any variable included in the model were excluded from the analysis.

RESULTS

Descriptive statistics

A total of 28525 individuals participated in the study, comprising 14329 women (50.61%) and 14196 men (49.39%) (figure 1). The BMI distribution included 7995 participants (28.73%) classified as having a healthy weight, 9470 (33.27%) as overweight and 11 060 (37.99%) as obese. Overall, 2508 individuals (7.79%) reported experiencing depressive symptoms.

Regarding dietary patterns, the majority of participants (n=25 009, 87.23%) reported not following any specific diet, while 2026 (8.10%) followed a calorie-restricted diet, 859 (2.90%) adhered to a nutrient-restricted diet, and 631 (1.77%) followed an established dietary pattern. Table 1 shows a detailed summary of participant characteristics stratified by depressive symptom status, while figure 2 shows the participant inclusion process. The distribution of dietary patterns among participants with and without depressive symptoms is further detailed in online supplemental table S1.

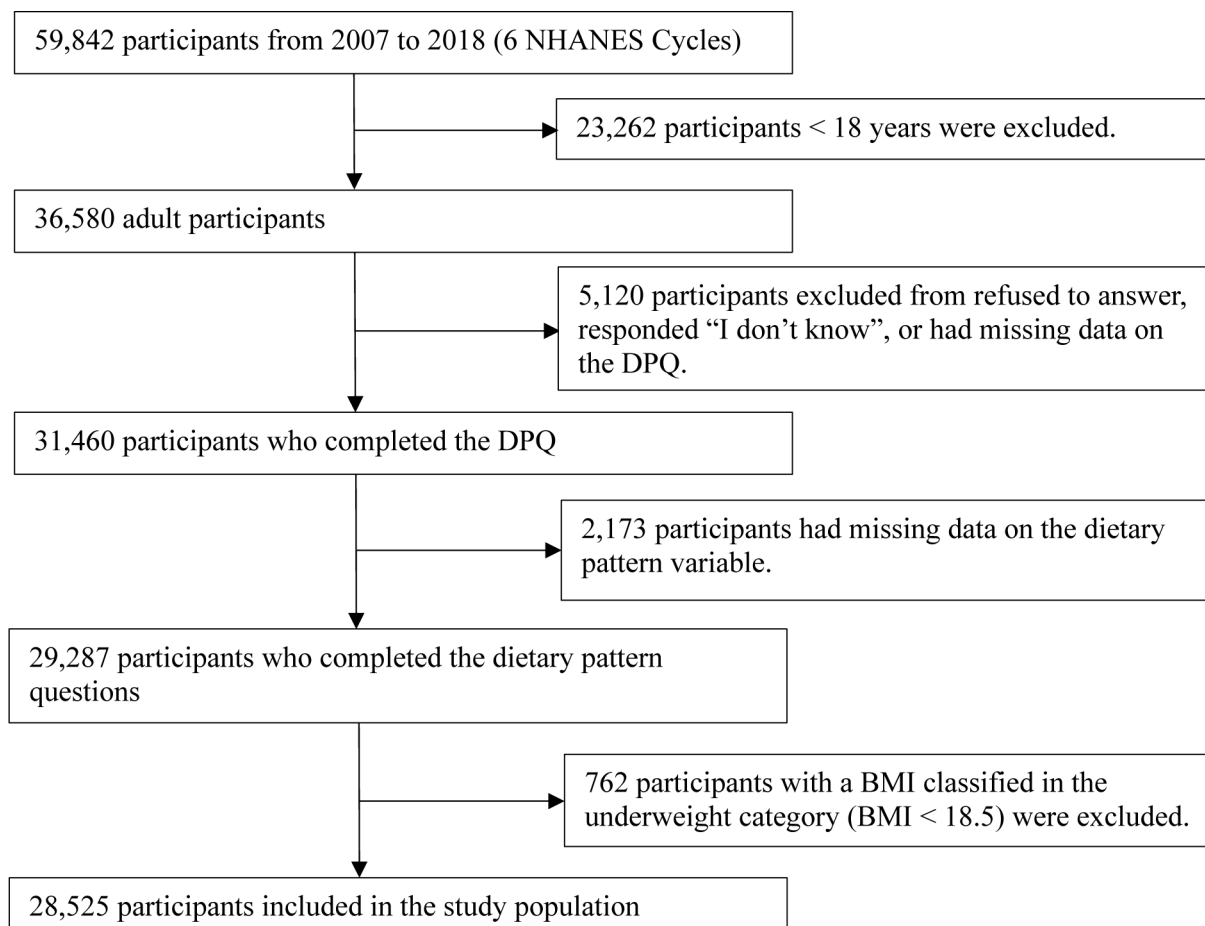


Figure 1 Participant inclusion flowchart. BMI, body mass index; DPQ, Mental Health – Depression Screener.

When stratified by sex, a greater proportion of men ($n=12\,772$, 89.81%) reported not following a diet compared with women ($n=12\,237$, 84.71%). In terms of BMI categories, calorie restriction was most commonly reported among obese participants ($n=1247$, 12.26%), followed by those who were overweight ($n=594$, 8.02%) and those with a healthy weight ($n=185$, 2.71%). The adoption of nutrient-restricted diets and established dietary patterns was relatively infrequent across all BMI groups, with the highest proportion of established dietary pattern users observed among obese participants ($n=359$, 2.77%). Online supplemental table S2 shows the full distribution of dietary patterns stratified by sex and BMI.

Association between dietary patterns and depressive symptom severity

Compared with non-diet users, individuals following a calorie-restricted diet had higher PHQ-9 scores (adjusted coefficient (aCoef)=0.29, 95% CI 0.06 to 0.52; $p=0.016$; table 2). In contrast, the use of a nutrient-restricted diet and an established dietary pattern was not associated with PHQ-9 scores, as indicated by p values >0.05 (table 2).

The interaction models indicated that the relationship between dietary patterns and depressive symptom severity varied by BMI, particularly among individuals with obesity following an established dietary pattern (table 3, figure 2). Subgroup analyses showed that, among overweight

individuals, both calorie-restricted diets (aCoef 0.46, 95% CI 0.02 to 0.89; $p=0.040$) and nutrient-restricted diets (aCoef 0.61, 95% CI 0.13 to 1.10; $p=0.014$) were associated with higher PHQ-9 scores (see online supplemental table S3). However, no associations were found in individuals classified as of healthy weight or obese. Furthermore, the relationship between dietary patterns and depressive symptom severity did not significantly differ by sex (online supplemental table S4), suggesting that the observed effects were consistent across men and women.

Association between dietary patterns and cognitive-affective and somatic symptoms

Participants following a calorie-restricted diet had a 0.16 unit increase in cognitive-affective scores (95% CI 0.02 to 0.29; $p=0.021$), while no significant associations were observed for those on nutrient-restricted or established dietary patterns (table 4).

Conversely, individuals on a nutrient-restricted diet experienced a 0.28 unit increase in somatic scores (95% CI 0.09 to 0.47; $p=0.005$), with no association found for calorie-restricted or established dietary pattern groups (table 4).

The association between dietary patterns and both symptom clusters varied by sex (table 5). Specifically, among men, a nutrient-restricted diet was associated

Table 1 Demographic characteristics stratified by presence of depressive symptoms (n=28 525)

Characteristic	Total	Depressive symptoms		P value
		No	Yes	
Sample size	28 525	26 017	2508	
Total PHQ-9 scores, mean (SD)	3.03 (4.06)	2.10 (2.37)	14.04 (3.79)	<0.001
Cognitive-affective scores, mean (SD)	1.18 (2.18)	0.70 (1.23)	6.83 (2.94)	<0.001
Somatic scores, mean (SD)	1.86 (2.28)	1.41 (1.61)	7.21 (2.19)	<0.001
Dietary patterns, n (%)				<0.001
Not on a diet	25 009 (87.23)	22 886 (87.43)	2123 (84.82)	
Calorie-restricted	2026 (8.10)	1824 (8.06)	202 (8.59)	
Nutrient-restricted	859 (2.90)	766 (2.86)	93 (3.42)	
Established dietary pattern	631 (1.77)	541 (1.65)	90 (3.18)	
Age, n (%)				<0.001
Young adults	12 745 (46.92)	11 679 (46.99)	1066 (46.17)	
Middle-aged adults	9425 (35.21)	8400 (34.78)	1025 (40.28)	
Older adults	6355 (17.87)	5938 (18.23)	417 (13.55)	
Sex, n (%)				<0.001
Female	14 329 (50.61)	12 736 (49.55)	1593 (63.20)	
Male	14 196 (49.39)	13 281 (50.45)	915 (36.80)	
Race, n (%)				<0.001
Mexican American	4474 (8.75)	4086 (8.79)	388 (8.23)	
Other Hispanic	2993 (5.81)	2638 (5.61)	355 (8.22)	
Non-Hispanic White	11 778 (67.02)	10 732 (67.34)	1046 (63.23)	
Non-Hispanic Black	6141 (11.09)	5605 (10.92)	536 (13.18)	
Other race (including multi-racial)	3139 (7.33)	2956 (7.34)	183 (7.15)	
Education, n (%)				<0.001
<9th grade	2648 (4.75)	2305 (4.49)	343 (7.80)	
9–11th grade	4265 (11.12)	3730 (10.58)	535 (17.49)	
High school graduate GED	6824 (24.09)	6191 (23.76)	633 (27.99)	
Some college AA degree	8075 (30.79)	7358 (30.59)	717 (33.14)	
College graduate or above	6321 (29.25)	6065 (30.58)	256 (13.57)	
Marital status, n (%)				<0.001
Married	13 883 (55.34)	13 043 (56.92)	840 (36.62)	
Widowed	2021 (5.45)	1796 (5.26)	225 (7.77)	
Divorced	3018 (10.31)	2592 (9.70)	426 (17.39)	
Separated	925 (2.37)	755 (2.10)	170 (5.57)	
Never married	4991 (18.19)	4473 (17.85)	518 (22.20)	
Living with partner	2271 (8.34)	2036 (8.16)	235 (10.45)	
PIR, n (%)				<0.001
Middle income	13 787 (48.72)	12 624 (48.40)	1163 (52.48)	
Low income	5691 (14.55)	4818 (13.30)	873 (29.30)	
High income	6532 (36.73)	6293 (38.30)	239 (18.22)	
Smoking status, n (%)				<0.001
Non-smoker	22 212 (80.56)	20 662 (82.25)	1550 (60.63)	
Smoker	5592 (19.44)	4681 (17.75)	911 (39.37)	

Continued

Table 1 Continued

Characteristic	Total	Depressive symptoms		P value
		No	Yes	
Drinking status, n (%)				<0.001
Never drinker	9027 (25.76)	8127 (25.32)	900 (31.06)	
Light drinker	5690 (19.14)	5091 (18.67)	599 (24.62)	
Moderate drinker	9477 (38.27)	8803 (38.89)	674 (30.86)	
Heavy drinker	3815 (16.83)	3518 (17.12)	297 (13.47)	
BMI, n (%)				<0.001
Healthy weight	7995 (28.73)	7417 (29.05)	578 (24.94)	
Overweight	9470 (33.27)	8801 (33.85)	669 (26.35)	
Obese	11 060 (37.99)	9799 (37.09)	1261 (48.70)	
Food security, n (%)				<0.001
Full	18980 (75.43)	17 882 (77.63)	1098 (49.27)	
Marginal	3484 (9.61)	3141 (9.26)	343 (13.70)	
Low	3218 (8.33)	2752 (7.63)	466 (16.63)	
Very low	2403 (6.63)	1851 (5.48)	552 (20.39)	
Diabetes, n (%)				<0.001
No	25 020 (90.87)	22 981 (91.35)	2039 (85.19)	
Yes	3489 (9.13)	3025 (8.65)	464 (14.81)	
Hypertension, n (%)				<0.001
No	20 509 (74.88)	19 000 (75.89)	1509 (62.90)	
Yes	7935 (25.12)	6943 (24.11)	992 (37.10)	

Categorical characteristics reported as unweighted frequency and weighted percent.
Continuous characteristics reported as weighted mean and SD.
The first level of categorical variables is the reference level.
P values <0.05 denote significant differences between depressive symptoms versus no depressive symptoms and are reported as weighted p values.
BMI, body mass index; PHQ-9, 9-item Patient Health Questionnaire; PIR, ratio of family income to poverty.

with higher cognitive-affective symptom scores compared with women not on a diet (aCoef 0.40, 95% CI 0.10 to 0.70; $p=0.010$) (table 5). Additionally, men following a calorie-restricted diet had higher somatic symptom scores compared with women not on a diet (aCoef 0.50, 95% CI 0.15 to 0.85; $p=0.005$) (table 6). Subgroup analyses further showed that, among men, a nutrient-restricted diet was associated with increased cognitive-affective scores (aCoef 0.26, 95% CI 0.05 to 0.47; $p=0.016$), while all three dietary patterns were positively associated with somatic scores. In contrast, no significant associations were observed among women for either symptom cluster (see online supplemental table S5).

Table 6 shows that obese individuals following an established dietary pattern had, on average, a 0.70 unit higher cognitive-affective symptom score (95% CI 0.06 to 1.33; $p=0.031$) and a 0.75 unit higher somatic symptom score (95% CI 0.16 to 1.33; $p=0.013$) compared with healthy-weight individuals not following a diet. In subgroup analyses, overweight individuals adhering to a nutrient-restricted diet had higher somatic scores (aCoef 0.44, 95% CI 0.15 to 0.73; $p=0.003$), while no associations were

observed in the healthy weight or obese groups (see online supplemental table S6).

DISCUSSION

Our study suggests that caloric-restricted diets are the only dietary pattern among the examined patterns that is associated with significantly increased depressive symptom scores compared with those not on a diet. These findings contrast with previous studies indicating that low-calorie diets improve depressive symptoms.²⁰ This discrepancy may arise because prior studies were primarily randomised controlled trials (RCTs) where participants adhered to carefully designed diets ensuring balanced nutrient intake.^{20 21} In contrast, real-life calorie-restricted diets and obesity often result in nutritional deficiencies (particularly in protein, essential vitamins/minerals) and induce physiological stress, which can exacerbate depressive symptomatology including cognitive-affective symptoms.^{22–25} However, it is important to note that the use of self-reported dietary data in our study may limit objectivity, such that the true calorie intakes remain unknown.

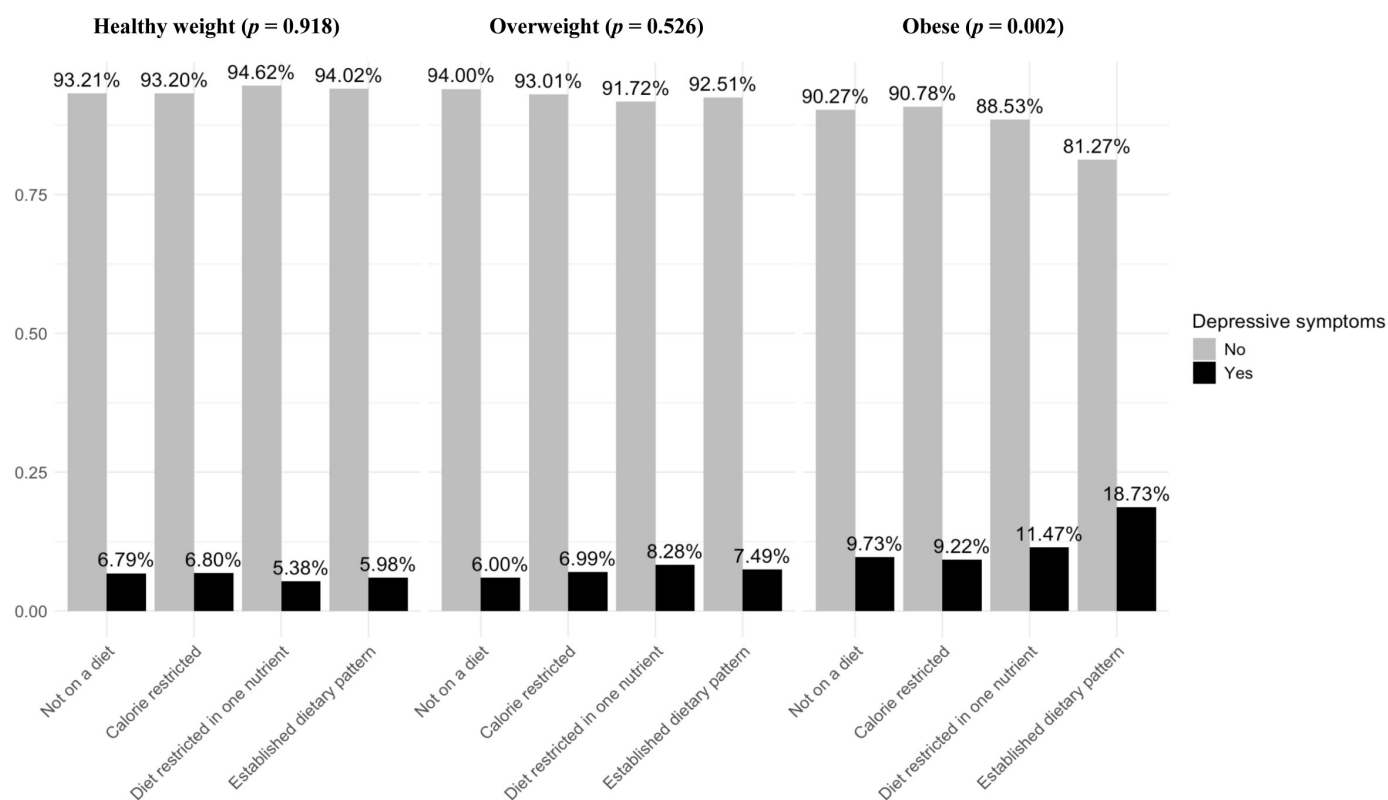


Figure 2 Prevalence of depressive symptoms by dietary pattern category stratified by body mass index.

The association between low-calorie diets and depressive symptoms remains inconclusive, highlighting the need for studies examining realistic calorie-restricted diets with their potential nutritional deficiencies.

Our findings indicated no significant association between other dietary patterns and depressive symptoms. We also found no significant association between consuming a nutrient-restricted diet and depressive symptoms. These findings align with prior studies on low carbohydrate and low fat diets.^{11 12} However, our results are inconsistent with other NHANES-based studies showing significant associations between nutrient-restricted diets and depressive symptoms. Cheng *et al* and Zhang *et al* found associations between low carbohydrate and low sugar diets and reduced depressive symptoms, respectively.^{26 27} However, these studies relied on 24-hour recalls, which can introduce recall bias and may

not reflect usual dietary patterns.^{26 27} Mao *et al* showed associations between low fibre intake, also a carbohydrate, and increased depressive symptoms.²⁸ Participants' limited knowledge on what constitutes a carbohydrate may explain why low carbohydrate diets appeared beneficial while low fibre diets showed the opposite, potentially causing a cancelling out effect in our study and leading to insignificant findings.

Furthermore, no significant association was observed between established dietary patterns (diabetic or DASH diet) and depressive symptoms. While some studies suggest the DASH diet may benefit depressive symptoms, results remain inconsistent.²⁹ Additionally, an important caveat is that self-reported adherence to a diabetic diet does not guarantee participants fully adhered to this diet. Further real-world and epidemiological research is needed to explore these patterns, particularly diets

Table 2 Main effect models for the association between dietary patterns and depressive symptom severity (PHQ-9 scores)

Total PHQ-9 scores				
Dietary patterns	Coef (95% CI)	P value	aCoef (95% CI)	P value
Calorie-restricted	0.31 (0.07 to 0.55)	0.011	0.29 (0.06 to 0.52)	0.016
Nutrient-restricted	0.52 (0.18 to 0.87)	0.003	0.29 (−0.01 to 0.60)	0.056
Established dietary pattern	1.32 (0.78 to 1.86)	<0.001	0.47 (−0.01 to 0.95)	0.053

Coef=unadjusted coefficient estimate (model including only the exposure variable), aCoef=adjusted coefficient estimate (model including exposure variable and all predefined covariates).
The reference level for dietary patterns is "not on a diet".
P values <0.05 denote statistical significance.
PHQ-9, 9-item Patient Health Questionnaire.

Table 3 Models for interaction of BMI and dietary patterns on depressive symptom severity (total PHQ-9 scores)

Total PHQ-9 scores				
Dietary patterns × BMI	Coef (95% CI)	P value	aCoef (95% CI)	P value
Calorie-restricted × overweight	0.13 (−0.71 to 0.96)	0.767	−0.08 (−0.93 to 0.78)	0.854
Nutrient-restricted × overweight	0.77 (−0.07 to 1.61)	0.070	0.81 (−0.06 to 1.68)	0.068
Established dietary pattern × overweight	0.30 (−0.75 to 1.34)	0.574	0.36 (−0.65 to 1.37)	0.477
Calorie-restricted × obese	−0.16 (−0.94 to 0.62)	0.689	−0.35 (−1.18 to 0.48)	0.404
Nutrient-restricted × obese	0.68 (−0.18 to 1.54)	0.117	0.59 (−0.39 to 1.57)	0.231
Established dietary pattern × obese	1.47 (0.24 to 2.71)	0.020	1.44 (0.35 to 2.54)	0.011

Coef=unadjusted coefficient estimate (model including only the exposure variable), aCoef=adjusted coefficient estimate (model including exposure variable and all predefined covariates).

The reference level for dietary patterns is “not on a diet” and the reference level for BMI is “healthy weight”.

P values <0.05 denote statistical significance.

BMI, body mass index; PHQ-9, 9-item Patient Health Questionnaire.

designed for specific health conditions such as renal or gluten-free diets and their impacts on mental health.

A key finding of our study was that, among overweight individuals, low-calorie and nutrient-restricted diets were associated with higher depressive symptom scores. This contradicts previous literature showing that low-calorie diets lower depressive symptoms in overweight individuals; however, weight loss has consistently proved to be an important moderator of this relationship.¹³ Controlled settings such as RCTs often yield better adherence to diets, leading to increased weight loss and reduced depressive symptoms.³⁰ This may not translate to real-world settings where barriers like insufficient support and motivation exist. Lack of weight loss or weight cycling while dieting in a real-world setting may lead to worsening depressive symptoms, which may explain our findings. Our cross-sectional design captures participants’ real-life experiences, emphasising the need for studies comparing control and real-world settings. Overweight individuals also showed an association with higher somatic symptom scores when consuming nutrient-restricted diets, consistent with literature linking restrictive dieting to somatic

symptoms like low energy, poor sleep and concentration difficulties.³¹

In biological men, all three dietary patterns were associated with higher somatic symptom scores while nutrient-restrictive diets were associated with increased cognitive-affective symptoms. These findings align with our hypothesis that sex differences influence the relationship between diet and depressive symptoms.¹⁴ Men have higher nutritional requirements, making them more susceptible to deficiencies when adopting any restrictive diet.^{14 32 33} Deficiencies in essential nutrients (eg, vitamin B12, folate and iron) can impair energy metabolism, exacerbating somatic symptoms of depression.^{14 32 33} Several factors may explain our findings that nutrient-restrictive diets were associated with higher cognitive-affective symptoms. Men tend to prefer fatty meals, snack on sweets and frequent fast-food restaurants more than women¹⁴ and are less likely to believe they need dietary changes.³⁴ Nutrient-restrictive diets, low in fat or carbohydrates, may conflict with these preferences and increase cognitive-affective symptoms like depressed mood. Additionally, glucose (the brain’s primary energy source) and omega-3

Table 4 Main effects models for the association between dietary patterns and symptom clusters

Dietary patterns	Coef (95% CI)	P value	aCoef (95% CI)	P value
Cognitive-affective symptom cluster				
Calorie-restricted	0.11 (−0.03 to 0.25)	0.109	0.16 (0.02 to 0.29)	0.021
Nutrient-restricted	0.11 (−0.06 to 0.27)	0.205	0.01 (−0.14 to 0.17)	0.848
Established dietary pattern	0.62 (0.33 to 0.90)	<0.001	0.26 (−0.01 to 0.53)	0.056
Somatic symptom cluster				
Calorie-restricted	0.20 (0.07 to 0.33)	0.003	0.13 (0.00 to 0.26)	0.051
Nutrient-restricted	0.42 (0.20 to 0.63)	<0.001	0.28 (0.09 to 0.47)	0.005
Established dietary pattern	0.70 (0.41 to 1.00)	<0.001	0.21 (−0.05 to 0.47)	0.118

Coef=unadjusted coefficient estimate (model including only the exposure variable), aCoef=adjusted coefficient estimate (model including exposure variable and all predefined covariates).

The reference level for dietary patterns is “not on a diet”.

P values <0.05 denote statistical significance.

Table 5 Models for interaction of sex and dietary patterns on symptom clusters

Dietary patterns × sex	Coef (95% CI)	P value	aCoef (95% CI)	P value
Cognitive-affective symptom cluster				
Calorie-restricted × male	0.09 (−0.22 to 0.40)	0.558	0.09 (−0.23 to 0.40)	0.583
Nutrient-restricted × male	0.24 (−0.06 to 0.54)	0.120	0.40 (0.10 to 0.70)	0.010
Established dietary pattern × male	−0.58 (−1.14 to −0.03)	0.038	−0.23 (−0.77 to 0.32)	0.406
Somatic symptom cluster				
Calorie-restricted × male	0.46 (0.14 to 0.79)	0.006	0.50 (0.15 to 0.85)	0.005
Nutrient-restricted × male	−0.16 (−0.58 to 0.27)	0.465	0.03 (−0.37 to 0.43)	0.879
Established dietary pattern × male	−0.26 (−0.77 to 0.26)	0.325	0.10 (−0.42 to 0.61)	0.710

Coef=unadjusted coefficient estimate (model including only the exposure variable), aCoef=adjusted coefficient estimate (model including exposure variable and all predefined covariates).

The reference level for dietary patterns is “not on a diet”, the reference level for sex is “female”,

P values <0.05 denote statistical significance.

fatty acids (essential for cognitive function) are critical for brain health.^{35 36} Diets low in carbohydrates (glucose) or fats (omega-3s) may theoretically worsen brain function and exacerbate cognitive-affective symptoms, especially in men with greater nutritional needs.^{37 38} Future studies should investigate sex and gender differences to guide tailored dietary counselling based on symptom profiles.

The strengths of our study include using a nationally representative cohort of US adults with comprehensive demographic, dietary and health data. Additionally, the PHQ-9, a validated tool, was used to assess depressive symptoms.³⁹ Moreover, we included multiple covariates in the multivariate regression models including social and income status, which were individually shown to

be significantly associated with depressive symptoms to account for any potential confounding effects. Furthermore, our comparison of multiple dietary patterns, rather than focusing on a single type, provides a novel perspective. Lastly, subgroup analyses by sex and BMI group further enhance the study's relevance. However, our study has several limitations. The cross-sectional design establishes correlation but not causation and prevents the determination of directionality of associations. The non-specific response options for dietary patterns introduce response bias, as participants may misclassify their diets. For instance, the response option ‘diabetic diet’ did not include a description. Similarly, our analysis showed that low sugar and low fibre diets together could not be a

Table 6 Models for interaction of BMI and dietary patterns on symptom clusters

Dietary patterns × BMI	Coef (95% CI)	P value	aCoef (95% CI)	P value
Cognitive-affective symptom cluster				
Calorie-restricted × overweight	0.07 (−0.41 to 0.54)	0.785	−0.07 (−0.55 to 0.41)	0.770
Nutrient-restricted × overweight	0.37 (−0.03 to 0.77)	0.067	0.36 (−0.05 to 0.77)	0.083
Established dietary pattern × overweight	−0.03 (−0.65, 0.59)	0.921	−0.03 (−0.68, 0.61)	0.916
Calorie-restricted × obese	0.01 (−0.44 to 0.46)	0.972	−0.15 (−0.61 to 0.32)	0.532
Nutrient-restricted × obese	0.32 (−0.06 to 0.69)	0.095	0.25 (−0.19 to 0.69)	0.261
Established dietary pattern × obese	0.70 (0.01 to 1.39)	0.046	0.70 (0.06 to 1.33)	0.031
Somatic symptom cluster				
Calorie-restricted × overweight	0.06 (−0.37 to 0.49)	0.786	−0.01 (−0.46 to 0.44)	0.970
Nutrient-restricted × overweight	0.40 (−0.12 to 0.93)	0.132	0.45 (−0.09 to 0.99)	0.103
Established dietary pattern × overweight	0.33 (−0.24 to 0.90)	0.258	0.39 (−0.14 to 0.92)	0.142
Calorie-restricted × obese	−0.17 (−0.56 to 0.23)	0.409	−0.20 (−0.63 to 0.23)	0.351
Nutrient-restricted × obese	0.37 (−0.22 to 0.96)	0.217	0.34 (−0.29 to 0.97)	0.284
Established dietary pattern × obese	0.77 (0.12 to 1.43)	0.022	0.75 (0.16 to 1.33)	0.013

Coef=unadjusted coefficient estimate (model including only the exposure variable), aCoef=adjusted coefficient estimate (model including exposure variable and all predefined covariates).

The reference level for dietary patterns is “not on a diet”, the reference level for BMI is “healthy weight”.

P values <0.05 denote statistical significance.

BMI, body mass index.

proxy for low carbohydrate diets, despite sugar and fibre being carbohydrates. NHANES data cycle 2005–2006 was thus removed as low carbohydrate was not a response option at that time and there was no appropriate proxy. This demonstrates that participants may not be accurately self-reporting their diets. Although the use of self-reported data may represent a limitation, objective data in the form of 24-hour recalls can introduce recall bias, and 2 days of intake may not accurately represent typical dietary intake. Studies suggest up to eight 24-hour recalls are necessary to accurately represent dietary intake.⁴⁰ Similarly, while objective data such as total energy intake is an important factor in dietary research, self-reported dietary behaviours such as caloric restriction may better capture the psychological and behavioural aspects influencing depressive symptoms. Therefore, our approach allows for the assessment of diet and depression in a way that aligns with how individuals perceive and implement dietary modifications in daily life.⁴⁰

CONCLUSION

Calorie-restricted diets were associated with higher depressive symptom scores, which contrasts with the findings of earlier controlled studies. Future studies simulating real-world dieting are important as challenges such as nutritional deficiencies may arise and contribute to worsening depressive symptoms. Additionally, biological men and overweight individuals appear more vulnerable to the negative effects of restrictive eating. Future intervention studies testing tailored dietary recommendations may provide more personalised strategies for mitigating depression risk.

Contributors GM contributed to study conceptualisation, methodology, investigation, project administration, writing (original draft) and writing (review and editing). SM contributed to study conceptualisation, methodology and writing (review and editing). QL contributed to methodology, formal analysis, writing (original draft) and writing (review and editing). WL contributed to methodology, formal analysis and writing (review and editing). AR contributed to writing (review and editing). VB contributed to study conceptualisation, methodology, investigation, project administration, supervision, writing (original draft) and writing (review and editing). VB is the guarantor. All authors provided critical revisions to the manuscript for intellectual content and contributed to editing, and read and approved the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests VB is supported by an Academic Scholar Award from the University of Toronto Department of Psychiatry and has received research support from the Canadian Institutes of Health Research, Brain & Behavior Foundation, Ontario Ministry of Health Innovation Funds, Royal College of Physicians and Surgeons of Canada, Department of National Defence (Government of Canada), New Frontiers in Research Fund, Associated Medical Services Inc. Healthcare, American Foundation for Suicide Prevention, Roche Canada, Novartis and Eisai.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the National Center for Health Statistics Research Ethics Review Board. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement The data that support the findings of this study are available from the corresponding author, VB, upon reasonable request.

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REFERENCES

- Liu Q, He H, Yang J, et al. Changes in the global burden of depression from 1990 to 2017: Findings from the Global Burden of Disease study. *J Psychiatr Res* 2020;126:134–40.
- Storch EA, Roberti JW, Roth DA. Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory–Second Edition in a sample of college students. *Depress Anxiety* 2004;19:187–9.
- American Psychiatric Association. Depressive Disorders. *Diagnostic and Statistical Manual of Mental Disorders* 2022;5. Available: https://psychiatryonline.org/doi/full/10.1176/appi.books.9780890425787.x04_Depressive_Disorders
- Mohammadi S, Seyedmirzaei H, Salehi MA, et al. Brain-based Sex Differences in Depression: A Systematic Review of Neuroimaging Studies. *Brain Imaging Behav* 2023;17:541–69.
- Li Y, Lv M-R, Wei Y-J, et al. Dietary patterns and depression risk: A meta-analysis. *Psychiatry Res* 2017;253:373–82.
- Selvaraj R, Selvamani TY, Zahra A, et al. Association Between Dietary Habits and Depression: A Systematic Review. *Cureus* 2022;14:e32359.
- Helland MH, Nordbotten GL. Dietary Changes, Motivators, and Barriers Affecting Diet and Physical Activity among Overweight and Obese: A Mixed Methods Approach. *Int J Environ Res Public Health* 2021;18:10582.
- Liu X, Morris MC, Dhana K, et al. Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) study: Rationale, design and baseline characteristics of a randomized control trial of the MIND diet on cognitive decline. *Contemp Clin Trials* 2021;102:106270.
- Jacka FN, O’Neil A, Opie R, et al. A randomised controlled trial of dietary improvement for adults with major depression (the “SMILES” trial). *BMC Med* 2017;15:23.
- Kim JY. Optimal Diet Strategies for Weight Loss and Weight Loss Maintenance. *J Obes Metab Syndr* 2021;30:20–31.
- Varaee H, Darand M, Hassanizadeh S, et al. Effect of low-carbohydrate diet on depression and anxiety: A systematic review and meta-analysis of controlled trials. *J Affect Disord* 2023;325:206–14.
- Soltani S, Sangsefidi ZS, Asoudeh F, et al. Effect of Low-Fat Diet on Depression Score in Adults: A Systematic Review and Meta-Analysis of Randomized Controlled Clinical Trials. *Nutr Rev* 2025;83:e741–50.
- Applewhite B, Penninx BWJH, Young AH, et al. The effect of a low-calorie diet on depressive symptoms in individuals with overweight or obesity: a systematic review and meta-analysis of interventional studies. *Psychol Med* 2024;54:1671–83.
- Chen Y, Kim M, Paye S, et al. Sex as a Biological Variable in Nutrition Research: From Human Studies to Animal Models. *Annu Rev Nutr* 2022;42:227–50.
- Patsalos O, Keeler J, Schmidt U, et al. Diet, Obesity, and Depression: A Systematic Review. *J Pers Med* 2021;11:176.
- National Center for Health Statistics. National Health and Nutrition Examination Survey: sample design, 2007–2010. 2013. Available: https://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf
- National Center for Health Statistics. National Health and Nutrition Examination Survey: plan and operations, 1999–2010. 2013. Available: https://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf
- Challa HJ, Ameer MA, Uppaluri KR. *DASH Diet To Stop Hypertension*. Treasure Island (FL): StatPearls, 2024.

- 19 Kroenke K, Wu J, Yu Z, *et al.* Patient Health Questionnaire Anxiety and Depression Scale: Initial Validation in Three Clinical Trials. *Psychosom Med* 2016;78:716–27.
- 20 Ein N, Armstrong B, Vickers K. The effect of a very low calorie diet on subjective depressive symptoms and anxiety: meta-analysis and systematic review. *Int J Obes (Lond)* 2019;43:1444–55.
- 21 Manchishi SM, Cui RJ, Zou XH, *et al.* Effect of caloric restriction on depression. *J Cell Mol Med* 2018;22:2528–35.
- 22 Fulgoni VL III, Agler A, Ricciuto L, *et al.* Impact of Simulated Caloric Reduction on Nutrient Adequacy Among U.S. Adults with Overweight or Obesity (National Health and Nutrition Examination Survey [NHANES] 2015–2018). *J Nutr* 2024;154:2732–42.
- 23 Zielińska M, Łuszczki E, Dereń K. Dietary Nutrient Deficiencies and Risk of Depression (Review Article 2018–2023). *Nutrients* 2023;15:2433.
- 24 Kaidar-Person O, Person B, Szomstein S, *et al.* Nutritional deficiencies in morbidly obese patients: a new form of malnutrition? Part A: vitamins. *Obes Surg* 2008;18:870–6.
- 25 Pankevich DE, Teegarden SL, Hedin AD, *et al.* Caloric restriction experience reprograms stress and orexigenic pathways and promotes binge eating. *J Neurosci* 2010;30:16399–407.
- 26 Cheng Z, Fu F, Lian Y, *et al.* Low-carbohydrate-diet score, dietary macronutrient intake, and depression among adults in the United States. *J Affect Disord* 2024;352:125–32.
- 27 Zhang L, Sun H, Liu Z, *et al.* Association between dietary sugar intake and depression in US adults: a cross-sectional study using data from the National Health and Nutrition Examination Survey 2011–2018. *BMC Psychiatry* 2024;24:110.
- 28 Mao Y, Li X, Zhu S, *et al.* Association Between Dietary Fiber Intake and Risk of Depression in Patients With or Without Type 2 Diabetes. *Front Neurosci* 2022;16:920845.
- 29 Tan J, Wang C, Tomiyama AJ. Dietary Approaches to Stop Hypertension (DASH) diet and mental well-being: a systematic review. *Nutr Rev* 2023;82:60–75.
- 30 Fabricatore AN, Wadden TA, Higginbotham AJ, *et al.* Intentional weight loss and changes in symptoms of depression: a systematic review and meta-analysis. *Int J Obes (Lond)* 2011;35:1363–76.
- 31 Habib A, Ali T, Nazir Z, *et al.* Unintended consequences of dieting: How restrictive eating habits can harm your health. *Int J Surg Open* 2023;60:100703.
- 32 Bennett E, Peters SAE, Woodward M. Sex differences in macronutrient intake and adherence to dietary recommendations: findings from the UK Biobank. *BMJ Open* 2018;8:e020017.
- 33 Huskisson E, Maggini S, Ruf M. The role of vitamins and minerals in energy metabolism and well-being. *J Int Med Res* 2007;35:277–89.
- 34 Grzymisławska M, Puch EA, Zawada A, *et al.* Do nutritional behaviors depend on biological sex and cultural gender? *Adv Clin Exp Med* 2020;29:165–72.
- 35 Mergenthaler P, Lindauer U, Dienel GA, *et al.* Sugar for the brain: the role of glucose in physiological and pathological brain function. *Trends Neurosci* 2013;36:587–97.
- 36 DiNicolantonio JJ, O’Keefe JH. The Importance of Marine Omega-3s for Brain Development and the Prevention and Treatment of Behavior, Mood, and Other Brain Disorders. *Nutrients* 2020;12:2333.
- 37 Verhulst CEM, Fabricius TW, Neffs G, *et al.* Consistent Effects of Hypoglycemia on Cognitive Function in People With or Without Diabetes. *Diabetes Care* 2022;45:2103–10.
- 38 Zhou L, Xiong J-Y, Chai Y-Q, *et al.* Possible antidepressant mechanisms of omega-3 polyunsaturated fatty acids acting on the central nervous system. *Front Psychiatry* 2022;13:933704.
- 39 Levis B, Benedetti A, Thombs BD, *et al.* Accuracy of Patient Health Questionnaire-9 (PHQ-9) for screening to detect major depression: individual participant data meta-analysis. *BMJ* 2019;365:1476.
- 40 Raina SK. Limitations of 24-hour Recall Method: Micronutrient Intake and the Presence of the Metabolic Syndrome. *N Am J Med Sci* 2013;5:498.