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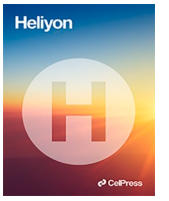

Research article

Association between oxidative balance score and self-reported severe headache or migraine based on NHANES 1999 to 2004 data: A cross-sectional study

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ABSTRACT

Purpose: The pathophysiological mechanisms underlying migraine remain elusive, with oxidative stress hypothesized as a potential etiological factor. The Oxidative Balance Score (OBS) is a comprehensive tool for assessing the impact of diet and lifestyle on oxidative stress, thereby gauging an individual's overall antioxidant capacity. In this cross-sectional study, we explored the correlation between OBS and migraine prevalence among a cohort of US adults.

Methods: We analyzed data from 6195 participants aged 20 years and above, drawn from the National Health and Nutrition Examination Survey (NHANES) conducted between 1999 and 2004. We employed multiple logistic regression, coupled with sensitivity analyses, to investigate the relationship between OBS and migraine. Subsequent subgroup analyses and interaction tests were performed to assess the consistency of this association across the population.

Results: Multiple logistic regression revealed an inverse relationship between OBS and the likelihood of experiencing migraines. Specifically, individuals in the highest OBS quartile exhibited a significantly reduced migraine risk compared to those in the lowest quartile (OR = 0.98, 95% Confidence Interval (CI): 0.97–0.99, P = 0.0001). Furthermore, restricted cubic spline curves indicated a non-linear association between dietary OBS and migraine incidence (non-linear P = 0.0258).

Discussion: Our findings suggest that adherence to an antioxidant-rich diet may be an effective strategy for mitigating migraine, potentially by influencing oxidative balance.

1. Introduction

Migraine is a prevalent, debilitating neurovascular disorder with significant personal and societal impacts [1]. It is characterized by recurrent, unilateral, pulsating, and severe headaches, often accompanied by symptoms such as nausea, vomiting, and heightened sensitivity to light (photophobia) and sound (phonophobia) [2]. These symptoms, exacerbated by physical activity, can persist for 4–72 h. Migraine is frequently associated with comorbidities [3], including psychiatric disorders like depression and anxiety, sleep disturbances, fatigue, and cardiovascular risk factors such as hypertension, diabetes mellitus, high cholesterol levels, and obesity. Additionally, it is linked to cardiovascular and cerebrovascular conditions, including patent foramen ovale and stroke [4].

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The exact pathophysiological mechanisms of migraine are not fully understood, but current evidence strongly suggests a link with increased oxidative stress. Migraine triggers include dietary components such as alcohol and nitrates, behavioral factors like stress, mental exertion, and irregular sleep patterns, as well as environmental and pharmaceutical elements, with nitroglycerin being particularly noteworthy [5,6]. Notably, most recognized or suspected migraine triggers are associated with oxidative stress [7].

Oxidative stress results from an imbalance between the production of reactive oxygen species (ROS) during normal metabolic activities and the body's innate antioxidant defenses [7]. This defense system, primarily relying on enzymes like catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx), and glutathione reductase (GSR), plays a crucial role in protecting the body from ROS/RNS-induced cellular damage [8]. Excessive ROS production, overwhelming the antioxidant defense capacity, can lead to cellular damage, affecting proteins, lipids, and DNA [9]. Oxidative stress can cause cellular dysfunction, contributing to

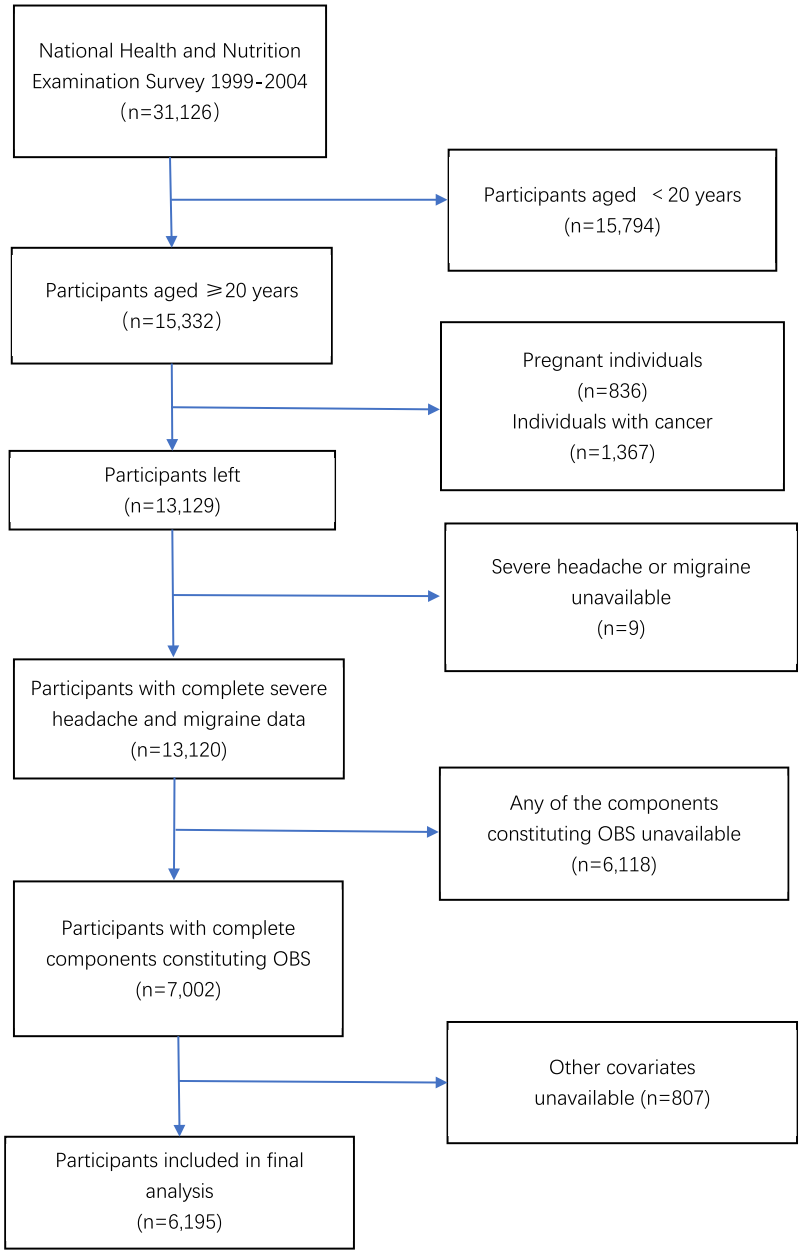

Fig.1.Flowchart of participants selection.

Fig. 1. Flowchart of participants selection.

neurodegenerative disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), and Huntington's disease (HD) [10], and is associated with medical conditions like ischemic strokes [11] and traumatic brain injury [12]. Furthermore, oxidative stress has been linked to various conditions, including migraine, neuropathic pain, and inflammatory pain [9]. The contribution of oxidative stress to migraine may involve brain energy deficits, mitochondrial ATP depletion, and increased levels of exogenous oxidants [13].

The Oxidative Balance Score (OBS) is a comprehensive metric assessing the balance between pro-oxidants and antioxidants in an individual's diet and lifestyle [7,14]. Generally, a higher OBS indicates a dominance of antioxidants over pro-oxidants. Numerous studies have established a negative correlation between OBS and the prevalence of various diseases, such as leukocyte telomere length [15], diabetes mellitus [14], osteoarthritis [16], and periodontitis [17]. However, research exploring the potential link between OBS and migraine is notably limited.

This study conducted a cross-sectional analysis using data from the 1999–2004 National Health and Nutrition Examination Survey (NHANES), aiming to explore the relationship between OBS and migraine incidence.

2. Materials and methods

2.1. Data sources and study population

The NHANES, a biennial survey encompassing adults and children, is conducted across the United States. This study utilized data from three 2-year cycles: 1999–2000, 2001–2002, and 2003–2004, focusing on participants reporting headaches or migraines. Of the 31,126 NHANES participants from 1999 to 2004, exclusions were made based on the following criteria: (1) under 20 years of age (n = 15,794), (2) pregnancy (n = 836), (3) a cancer diagnosis (n = 1367), (4) missing headache or migraine data (n = 9), and (5) incomplete OBS component information (n = 6118). Additional exclusions were made for missing covariate data (n = 807), resulting in a final cohort of 6195 participants. The National Center for Health Statistics' (NCHS) Research Ethics Review Board approved the NHANES survey, following informed consent from each participant (Fig. 1).

2.2. Calculation of oxidative balance score

The OBS combines data from 16 dietary and 4 lifestyle factors [18,19]. This comprehensive score encompasses evaluations of 15 antioxidants and 5 pro-oxidants. Higher OBS values indicate increased antioxidant activity in participants. Components of OBS calculation include dietary fiber, carotenoids, riboflavin, niacin, calcium, magnesium, zinc, total folate, vitamins B6, B12, C, E, total fat, selenium, copper, and dietary iron. Lifestyle factors considered are body mass index (BMI), cotinine levels, alcohol consumption patterns, and physical activity. Components are categorized as either pro-oxidants or antioxidants, with pro-oxidants including fats, iron, alcohol, cotinine, and BMI. All elements are divided into tertiles (Table 1), with pro-oxidants and antioxidants assigned scores from 2 to 0 and 0 to 2, respectively, within each tertile [19]. This scoring system facilitates a comprehensive evaluation of the oxidative

Table 1
Components of the oxidative balance score.

OBS components	Property	Male			Female		
		0	1	2	0	1	2
Dietary OBS components							
Dietary fiber (g/d)	A	<12.55	12.55–19.67	>19.67	<10.09	10.09–16.30	>16.30
Carotene (RE/d)	A	<98.68	98.68–306.04	>306.04	<97.96	97.96–383.46	>383.46
Riboflavin (mg/d)	A	<1.79	1.79–2.69	>2.69	<1.34	1.34–2.02	>2.02
Niacin (mg/d)	A	<20.65	20.65–29.74	>29.74	<14.51	14.51–21.86	>21.86
Vitamin B6 (mg/d)	A	<1.59	1.59–2.40	>2.40	<1.13	1.13–1.77	>1.77
Total folate (mcg/d)	A	<315.52	315.52–491.5	>491.5	<250.50	250.50–388.5	>388.5
Vitamin B12 (mcg/d)	A	<3.36	3.36–6.20	>6.20	<2.22	2.22–4.21	>4.21
Vitamin C (mg/d)	A	<42.40	42.40–113.20	>113.20	<38.00	38.00–98.30	>98.30
Vitamin E (ATE) (mg/d)	A	<5.82	5.82–9.41	>9.41	<4.53	4.53–7.51	>7.51
Calcium (mg/d)	A	<645.50	645.50–1070.78	>1070.78	<499.23	499.23–848.78	>848.78
Magnesium (mg/d)	A	<256.86	256.86–361.05	>361.05	<186.95	186.95–283.21	>283.21
Zinc (mg/d)	A	<9.74	9.74–15.09	>15.09	<6.73	6.73–10.74	>10.74
Copper (mg/d)	A	<1.12	1.12–1.57	>1.57	<0.85	0.85–1.28	>1.28
Selenium (mcg/d)	A	<94.92	94.92–141.60	>141.60	<67.75	67.75–99.40	>99.40
Total fat (g/d)	P	>107.43	69.83–107.43	<69.83	>75.79	50.98–75.79	<50.98
Iron (mg/d)	P	>19.17	12.88–19.17	<12.88	>14.32	9.65–14.32	<9.65
Lifestyle OBS components							
Physical activity (MET-minute/week)	A	<415.10	415.10–1134.00	>1134.00	<269.00	269.00–843.00	>843.00
Alcohol (drinks/d)	P	>3	2–3	≤2	>2	1–2	≤1
Body mass index (kg/m2)	P	>29.16	25.54–29.16	<25.54	>28.64	23.74–28.64	<23.74
Cotinine (ng/mL)	P	>1.12	0.04–1.12	<0.04	>0.17	0.04–0.17	<0.04

Antioxidant was represented by A; pro-oxidant was represented by P; retinol equivalent was represented by RE; alpha-tocopherol equivalent was represented by ATE; metabolic equivalent represented by MET.

balance.

2.3. Definition of severe headache or migraine

Consistent with methodologies in prior NHANES-based studies [20], migraine status determination primarily relied on responses from the Pain Questionnaire. A severe headache or migraine was defined by an affirmative answer to: "In the past 3 months, have you

Table 2
The baseline characteristics by migraine: National Health and Nutrition Examination Survey 1999–2004.

Characteristics	Total	Migraine	Control	P value
	n = 6195	n = 1229	n = 4966	
	N = 200,475,396	N = 42,239,668	N = 158,235,728	
Age,years, Mean (S.E)	43.81 ± 0.37	40.32 ± 0.38	44.75 ± 0.42	<0.0001
Age group, years,n (%)				<0.0001
20–39	2309 (41.95)	567 (48.72)	1742 (40.14)	
40–59	2159 (42.33)	498 (44.55)	1661 (41.74)	
≥60	1727 (15.71)	164 (6.73)	1563 (18.11)	
Gender,n (%)				<0.0001
male	3338 (53.88)	478 (38.12)	2860 (55.73)	
female	2857 (46.12)	751 (61.88)	2106 (44.27)	
Race,n (%)				0.59
Mexican American	1267 (20.45)	278 (7.21)	989 (6.19)	
Non-Hispanic White	1020 (16.46)	212 (9.23)	808 (8.47)	
Non-Hispanic Black	3443 (55.58)	644 (74.82)	2799 (76.16)	
Other Hispanic	253 (4.08)	58 (5.06)	195 (4.71)	
Other Race	212 (3.42)	37 (3.68)	175 (4.47)	
Marital Status,n (%)				0.21
non-single	4034 (65.12)	777 (65.81)	3257 (68.05)	
single	2161 (34.88)	452 (34.19)	1709 (31.95)	
Education level,n (%)				<0.001
Less than high school	641 (10.35)	117 (4.05)	524 (4.09)	
High school or GED	2329 (37.59)	518 (41.02)	1811 (33.64)	
Above high school	3225 (52.06)	594 (54.92)	2631 (62.27)	
PIR, Mean (S.E)	3.20 ± 0.06	2.80 ± 0.06	3.30 ± 0.06	<0.0001
PIR group,n (%)				<0.0001
≤1.3	1445 (17.00)	360 (23.03)	1085 (15.40)	
>1.3, ≤ 1.85	702 (9.22)	166 (11.66)	536 (8.56)	
>1.85	4048 (73.78)	703 (65.31)	3345 (76.04)	
Total energy intake, Mean (S.E)	2278.69 ± 14.53	2243.77 ± 27.57	2288.02 ± 17.53	0.2
Dietary OBS, Mean (S.E)	19.80 ± 0.22	18.99 ± 0.27	20.01 ± 0.23	<0.001
Lifestyle OBS, Mean (S.E)	3.61 ± 0.06	3.37 ± 0.09	3.67 ± 0.06	<0.001
OBS, Mean (S.E)	19.80 ± 0.22	18.99 ± 0.27	20.01 ± 0.23	<0.001
White blood cells(×103 cells/ml)	7.18 ± 0.05	7.47 ± 0.08	7.10 ± 0.05	<0.0001
CRP(mg/dL)	0.38 ± 0.01	0.41 ± 0.02	0.37 ± 0.01	0.06
Triglycerides (mmol/L)	1.56 ± 0.02	1.56 ± 0.04	1.56 ± 0.03	0.93
Total cholesterol (mmol/L)	5.20 ± 0.02	5.17 ± 0.03	5.21 ± 0.03	0.4
HDL-C (mmol/L)	1.36 ± 0.01	1.33 ± 0.01	1.36 ± 0.01	0.01
LDL-C (mmol/L)	3.10 ± 0.03	3.07 ± 0.05	3.11 ± 0.03	0.36
SII	588.07 ± 4.55	610.12 ± 9.57	582.18 ± 4.80	0.01
CVD,n (%)				0.8
No	5670 (91.53)	1136 (93.49)	4534 (93.72)	
Yes	525 (8.47)	93 (6.51)	432 (6.28)	
CKD,n (%)				0.01
No	5291 (85.41)	1106 (92.41)	4185 (88.66)	
Yes	904 (14.59)	123 (7.59)	781 (11.34)	
Diabetes,n (%)				0.11
No	5538 (89.39)	1114 (93.44)	4424 (92.12)	
Yes	657 (10.61)	115 (6.56)	542 (7.88)	
Hypertension,n (%)				0.17
No	3920 (63.28)	829 (69.95)	3091 (68.02)	
Yes	2275 (36.72)	400 (30.05)	1875 (31.98)	
Hyperlipidemia,n (%)				0.76
No	1662 (26.83)	340 (28.08)	1322 (28.71)	
Yes	4533 (73.17)	889 (71.92)	3644 (71.29)	

PIR: poverty to income ratio;*OBS*:oxidative balance score; *CRP*, C-reactive protein; *SII*, systemic immune inflammation index; *HDL-C*, high density lipoprotein cholesterol; *LDL-C*, low density lipoprotein cholesterol; *CVD*, cardiovascular disease; *CKD*, chronic kidney disease.
Mean (S.E) for continuous variables: P-value was calculated by weighted linear regression model; *n (%)* for categorical variables: P-value was calculated by weighted x^2 test.
n: Unweighted number of observations in data set; *N*: Weighted number of observations in data set.

experienced a severe headache or migraine?"

2.4. Other covariates

A broad range of covariates was carefully evaluated, guided by existing literature [16–20]. These included demographic variables such as age, race/ethnicity, gender, educational attainment, marital status, poverty-to-income ratio (PIR), and total energy intake (kcal) [19]. Race/ethnicity was categorized into five groups: non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic, and other. Educational levels were classified into less than high school, high school, and beyond high school. Marital status was reported as non-single or single. Household income was categorized using PIR into low (PIR ≤1.3), moderate (PIR >1.3 to 1.85), and high (PIR >1.85).

Table 3
The baseline characteristics by oxidative balance score: National Health and Nutrition Examination Survey 1999–2004.

Characteristic	Total	Q1 [5,16]	Q2 [17,22]	Q3 [23,24]	Q4 [25,26]	P value
	n = 6195	n = 1543	n = 1498	n = 1561	n = 1593	
	N = 98,292,929	N = 22,307,789	N = 22,541,339	N = 25,486,178	N = 27,957,623	
Age,years, Mean (S.E)	43.69 ± 0.33	43.01 ± 0.54	44.36 ± 0.46	43.79 ± 0.55	43.61 ± 0.43	0.21
Gender,n (%)						0.46
Male	3338 (53.88)	875 (54.21)	810 (52.42)	827 (52.16)	826 (50.64)	
Female	2857 (46.12)	668 (45.79)	688 (47.58)	734 (47.84)	767 (49.36)	
Race,n (%)						<0.0001
Mexican American	1267 (20.45)	319 (6.60)	320 (6.45)	337 (6.33)	291 (5.68)	
Non-Hispanic White	1020 (16.46)	383 (13.59)	277 (10.13)	214 (6.80)	146 (4.33)	
Non-Hispanic Black	3443 (55.58)	718 (69.15)	790 (73.63)	888 (77.09)	1047 (82.65)	
Other Hispanic	253 (4.08)	70 (6.07)	62 (5.36)	68 (5.48)	53 (3.79)	
Other Race	212 (3.42)	53 (4.59)	49 (4.43)	54 (4.31)	56 (3.54)	
Marital Status,n (%)						0.01
Non-Single	4034 (65.12)	923 (63.04)	989 (68.51)	1037 (67.44)	1085 (70.87)	
Single	2161 (34.88)	620 (36.96)	509 (31.49)	524 (32.56)	508 (29.13)	
Education level,n (%)						<0.0001
Less than high school	641 (10.35)	211 (5.81)	193 (5.41)	142 (3.17)	95 (2.31)	
High school or GED	2329 (37.59)	689 (43.47)	586 (38.64)	613 (38.10)	441 (24.28)	
Above high school	3225 (52.06)	643 (50.72)	719 (55.95)	806 (58.73)	1057 (73.41)	
PIR, Mean (S.E)	3.21 ± 0.06	2.76 ± 0.06	3.06 ± 0.08	3.27 ± 0.06	3.63 ± 0.06	<0.0001
Total energy intake, Mean (S.E)	2284.15 ± 15.35	1632.07 ± 23.17	2028.02 ± 24.84	2445.74 ± 30.66	2863.65 ± 32.93	<0.0001
Dietary OBS, Mean (S.E)	19.64 ± 0.21	10.05 ± 0.08	16.20 ± 0.07	21.77 ± 0.07	28.12 ± 0.08	<0.0001
Lifestyle OBS, Mean (S.E)	3.63 ± 0.06	2.46 ± 0.04	3.35 ± 0.07	3.72 ± 0.06	4.70 ± 0.06	<0.0001
OBS, Mean (S.E)	23.27 ± 0.25	12.51 ± 0.09	19.55 ± 0.06	25.50 ± 0.06	32.82 ± 0.09	<0.0001
SII, Mean (S.E)	586.67 ± 4.99	603.93 ± 8.01	595.52 ± 8.87	584.40 ± 10.71	567.89 ± 9.63	0.02
White blood cells(×103 cells/ml)	7.16 ± 0.04	7.58 ± 0.08	7.22 ± 0.07	7.13 ± 0.06	6.80 ± 0.06	<0.0001
CRP(mg/dL)	0.37 ± 0.01	0.45 ± 0.02	0.38 ± 0.02	0.39 ± 0.03	0.28 ± 0.01	<0.0001
Triglycerides (mmol/L)	1.58 ± 0.03	1.74 ± 0.05	1.59 ± 0.06	1.56 ± 0.05	1.46 ± 0.05	<0.001
Total cholesterol (mmol/L)	5.18 ± 0.02	5.26 ± 0.04	5.20 ± 0.03	5.13 ± 0.03	5.14 ± 0.04	0.1
HDL-C (mmol/L)	1.34 ± 0.01	1.25 ± 0.01	1.33 ± 0.02	1.34 ± 0.01	1.42 ± 0.01	<0.0001
LDL-C (mmol/L)	3.13 ± 0.02	3.22 ± 0.04	3.16 ± 0.04	3.10 ± 0.04	3.06 ± 0.04	0.06
CVD,n (%)						0.01
No	5670 (91.53)	1393 (93.25)	1360 (93.30)	1414 (92.86)	1503 (95.68)	
Yes	525 (8.47)	150 (6.75)	138 (6.70)	147 (7.14)	90 (4.32)	
CKD,n (%)						<0.0001
No	5291 (85.41)	1267 (85.98)	1250 (89.33)	1354 (90.11)	1420 (92.24)	
Yes	904 (14.59)	276 (14.02)	248 (10.67)	207 (9.89)	173 (7.76)	
Diabetes,n (%)						0.02
No	5538 (89.39)	1351 (91.21)	1317 (92.06)	1402 (93.12)	1468 (93.89)	
Yes	657 (10.61)	192 (8.79)	181 (7.94)	159 (6.88)	125 (6.11)	
Hypertension,n (%)						<0.001
No	3920 (63.28)	902 (64.82)	900 (67.03)	1006 (70.57)	1112 (73.42)	
Yes	2275 (36.72)	641 (35.18)	598 (32.97)	555 (29.43)	481 (26.58)	
Hyperlipidemia,n (%)						<0.0001
No	1662 (26.83)	369 (22.97)	347 (24.63)	439 (30.49)	507 (33.70)	
Yes	4533 (73.17)	1174 (77.03)	1151 (75.37)	1122 (69.51)	1086 (66.30)	
Migraine,n (%)						0.01
No	4966 (80.16)	1205 (76.43)	1188 (76.45)	1258 (79.76)	1315 (82.20)	
Yes	1229 (19.84)	338 (23.57)	310 (23.55)	303 (20.24)	278 (17.80)	

PIR: poverty to income ratio;*OBS*:oxidative balance score; *CRP*, C-reactive protein; *SII*, systemic immune inflammation index; *HDL-C*, high density lipoprotein cholesterol; *LDL-C*, low density lipoprotein cholesterol; *CVD*, cardiovascular disease; *CKD*, chronic kidney disease.
Mean (S.E) for continuous variables: P-value was calculated by weighted linear regression model; *n (%)* for categorical variables: P-value was calculated by weighted x^2 test.
n: Unweighted number of observations in data set; *N*: Weighted number of observations in data set.

Cardiovascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia, cardiovascular disease (CVD), and chronic kidney disease (CKD), were also considered. Hypertension was defined by at least three abnormal blood pressure readings (systolic ≥140 mmHg or diastolic ≥90 mmHg), use of antihypertensive medication, or a self-reported or physician-confirmed diagnosis. Diabetes mellitus diagnosis criteria included a medical history, HbA1c > 6.5%, fasting glucose ≥7.0 mmol/L, random glucose ≥11.1 mmol/L, 2-h OGTT ≥11.1 mmol/L, or use of diabetes medication or insulin [21]. Hyperlipidemia was identified if total cholesterol ≥5.18 mmol/L, triglycerides ≥150 mg/dL, HDL cholesterol <1.04 mmol/L in men and <1.30 mmol/L in women, LDL cholesterol ≥3.37 mmol/L, or if cholesterol-lowering medications were used. CKD was defined as an eGFR below 60 mL/min/1.73 m^2 or a urinary albumin-to-creatinine ratio >30 mg/g, calculated using the CKD Epidemiology Collaboration creatinine equation [22]. CVD encompassed conditions like congestive heart failure, coronary artery disease, myocardial infarction, angina pectoris, and stroke [21].

2.5. Statistical analyses

NHANES analysis guide's complex sampling weight calculations ensured appropriate weighting of the sample data. Categorical data were expressed as frequency with weighted percentage, and continuous variables as means with standard errors (SE).

Continuous variables were analyzed using t-tests, and categorical variables with chi-square tests for baseline characteristics. Multivariate logistic regression models generated odds ratios (ORs) and 95% confidence intervals (CIs) for the association between OBS and severe headache or migraine risk, adjusting for potential confounders. OBS variables were divided into quartiles and treated as continuous variables, with the lowest quartile as the reference. The basic model included no covariate adjustments. Model 1 adjusted for age, gender, and race. Model 2 added adjustments for education, marital status, PIR, and total calorie intake. Model 3 included all Model 2 adjustments plus CKD, CVD, diabetes, hypertension, and hyperlipidemia.

Restricted cubic spline analysis with a spline smoothing function assessed a dose-response relationship between OBS and migraine risk. Subgroup analyses considered age, gender, CKD, CVD, hypertension, diabetes, and hyperlipidemia. For sensitivity analyses, data on CVD (n = 525) and abnormal energy intake (n = 238 for males with intake <800 or >4200 kcal/day; n = 100 for females with intake <500 or >3500 kcal/day) were excluded due to potential migraine associations. Statistical analyses were conducted using R version 4.2.2, with statistical significance set at a two-sided P-value of 0.05.

3. Results

3.1. Baseline characteristics

This study analyzed 6195 NHANES participants who completed the pain questionnaire and provided accurate 24-h dietary recall. Baseline characteristics, delineated in Table 2, were stratified by migraine prevalence. Migraines affected 19.84% (n = 1229) of the sample, with a higher incidence in females (751, 61.88%) compared to males (478, 38.12%). The average age of participants was 40.32

Table 4
Weighted Multiple logistic regression analysis models showing the associations between OBS and migraine.

	Crude model		Model 1		Model 2		Model 3	
	OR (95%CI)	P value	OR (95%CI)	P value	OR (95%CI)	P value	OR (95%CI)	P value
OBS	0.98 (0.97, 0.99)	<0.0001	0.98 (0.97, 0.99)	<0.0001	0.98 (0.97, 0.99)	0.0001	0.98 (0.97, 0.99)	0.0001
OBS quartiles								
Q1	Ref.		Ref.		Ref.		Ref.	
Q2	1.00 (0.77, 1.30)	0.9912	1.02 (0.78, 1.33)	0.9099	1.00 (0.76, 1.32)	0.9999	1.00 (0.76, 1.31)	0.9912
Q3	0.82 (0.67, 1.01)	0.0645	0.82 (0.67, 1.02)	0.0829	0.78 (0.62, 0.98)	0.0432	0.79 (0.63, 0.99)	0.0481
Q4	0.70 (0.60, 0.83)	0.0001	0.69 (0.58, 0.81)	0.0001	0.67 (0.54, 0.83)	0.0008	0.68 (0.55, 0.84)	0.0015
p for trend		<0.0001		<0.0001		0.0002		0.0003
Dietary OBS	0.98 (0.97, 0.99)	<0.0001	0.98 (0.97, 0.99)	<0.0001	0.97 (0.96, 0.98)	0.0001	0.97 (0.96, 0.99)	0.0002
Dietary OBS quartiles								
Q1	Ref.		Ref.		Ref.		Ref.	
Q2	1.01 (0.79, 1.27)	0.9646	1.01 (0.80, 1.28)	0.9243	0.99 (0.77, 1.27)	0.9288	0.99 (0.78, 1.27)	0.9484
Q3	0.83 (0.70, 0.99)	0.0449	0.82 (0.69, 0.99)	0.0474	0.76 (0.62, 0.93)	0.0134	0.76 (0.62, 0.93)	0.0124
Q4	0.72 (0.61, 0.85)	0.0003	0.70 (0.59, 0.83)	0.0002	0.63 (0.50, 0.80)	0.0006	0.64 (0.51, 0.81)	0.0009
p for trend		<0.0001		<0.0001		0.0001		0.0001
Lifestyle OBS	0.91 (0.86, 0.95)	0.0004	0.91 (0.87, 0.96)	0.0015	0.95 (0.90, 1.00)	0.0613	0.96 (0.91, 1.01)	0.1557
Lifestyle OBS quartiles								
Q1	Ref.		Ref.		Ref.		Ref.	
Q2	0.83 (0.66, 1.04)	0.1101	0.83 (0.66, 1.05)	0.1379	0.89 (0.71, 1.13)	0.3477	0.91 (0.72, 1.15)	0.4323
Q3	0.78 (0.60, 1.02)	0.081	0.83 (0.63, 1.10)	0.2046	0.95 (0.72, 1.26)	0.7456	0.98 (0.73, 1.31)	0.8976
Q4	0.60 (0.45, 0.80)	0.0013	0.63 (0.46, 0.85)	0.0044	0.77 (0.57, 1.04)	0.0946	0.81 (0.59, 1.11)	0.1968
p for trend		0.0013		0.0046		0.1189		0.2463

Crude model: Unadjusted model.
Model 1: Adjusted for age, sex, race.
Model 2: Additionally adjusted for marital status, education, poverty-income ratio, and total energy intake.
Model 3: Additionally, adjusted for hypertension, diabetes, hyperlipidemia, cardiovascular disease, and chronic kidney disease.

± 0.38 years, and the mean total energy intake was 2278.69 ± 14.53 kcal/day. Significant differences in age, gender, education, CKD, PIR, and OBS were observed between individuals with and without migraine. Migraineurs had lower scores in dietary OBS, lifestyle OBS, and total OBS compared to non-migraineurs. Additionally, migraine prevalence was higher among women, non-Hispanic whites, individuals with higher education and income levels, and those with a higher incidence of CKD. Table 3 further details participant characteristics by OBS quartiles.

3.2. Association between OBS and migraine

Multivariate logistic regression, detailed in Table 4, established a relationship between migraine and OBS. A 2% reduction in migraine prevalence was associated with every 1-unit increase in total OBS across all models (OR = 0.98, 95% CI: 0.97–0.99, p = 0.0001). This indicates a consistent negative correlation between OBS and migraine frequency. Additionally, when OBS was categorized, individuals in the highest quartile exhibited a 32% lower migraine risk compared to the lowest quartile in the fully adjusted model (OR = 0.68, 95% CI: 0.55–0.84, p = 0.0015).

Logistic regression modeling assessed the impact of dietary and lifestyle OBS on migraine. Both were considered preventive factors, though lifestyle OBS did not achieve statistical significance. A pronounced dose-response relationship was observed for both overall OBS and dietary OBS (P for trend = 0.0001).

3.3. Subgroup analyses

Subgroup analyses focused on the relationship between dietary OBS, lifestyle OBS, and migraine. These analyses accounted for cardiovascular disease (CVD), chronic kidney disease (CKD), hyperlipidemia, hypertension, and diabetes mellitus (Fig. 2). Results were consistent across subgroups, confirming the stable association between OBS and migraine risk.

No significant interactions were found between lifestyle OBS, dietary OBS, and the mentioned covariates (P for interaction >0.05). However, noteworthy findings emerged in specific subgroups. In the diabetes subgroup, individuals with diabetes showed a heightened response to lifestyle OBS compared to non-diabetics (P = 0.0245). In contrast, the CKD subgroup revealed a stronger response to dietary OBS in individuals without CKD compared to those with CKD (P < 0.0001).

3.4. RCS analysis

Fig. 3 depicts the non-linear relationships between dietary OBS, lifestyle OBS, and migraine. Utilizing Restricted Cubic Spline (RCS) analysis with Model 3 adjustments, we examined the association between OBS and migraine. RCS analysis revealed a non-linear correlation between dietary OBS and migraine (p-value for nonlinearity: 0.0258). Specifically, the risk of migraine decreased rapidly with an increase in dietary OBS, particularly when it exceeded 12 (Fig. 3A). Beyond a dietary OBS of 20, the decline in migraine risk lessened with further increases in dietary OBS. However, no non-linear associations were found between lifestyle OBS, overall OBS, and migraine (P-values for nonlinearity: 0.6087 and 0.1548, respectively).

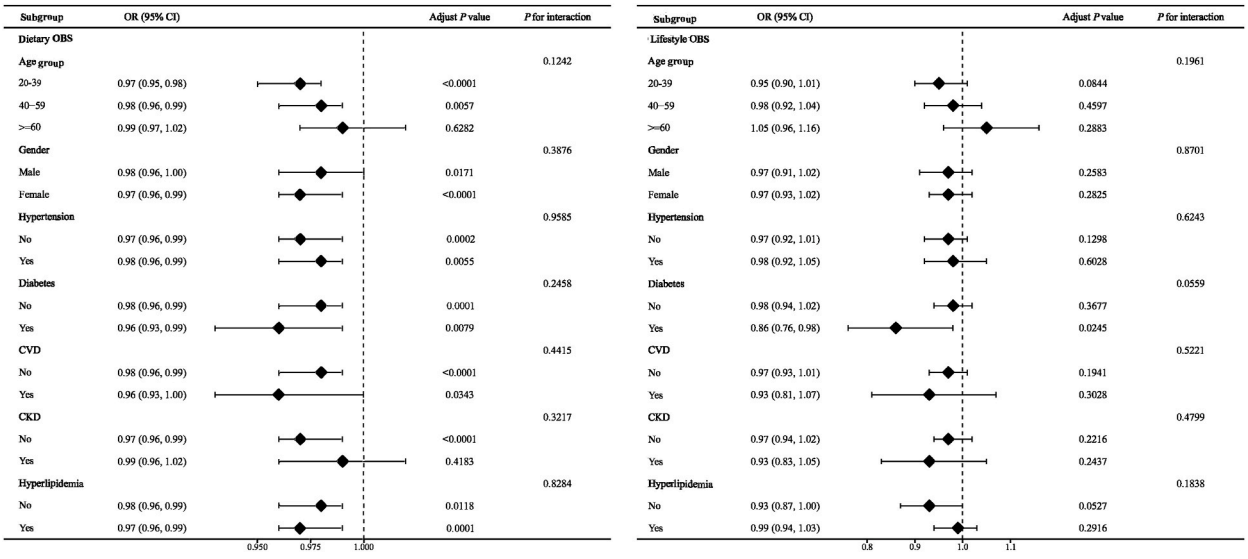

Fig. 2. Data are all adjusted by age,gender, hypertension,diabetes, hyperlipidemia, CVD, and CKD.CVD, cardiovascular disease; CKD, chronic kidney disease.

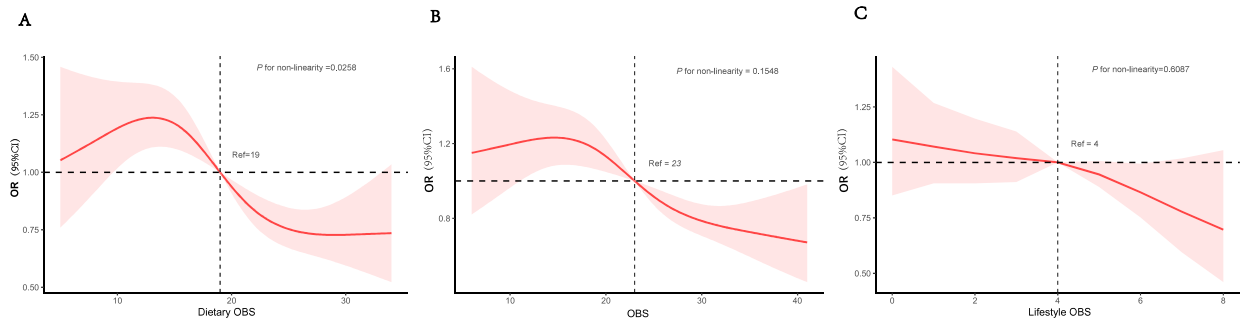

Fig. 3. Dose-response relationships between migraine and dietary OBS(A), total OBS(B),lifestyle OBS(C).Non-linear associations between migraine and dietary OBS were found (P = 0.0258).

3.5. Sensitivity analyses

Sensitivity analyses affirmed the robustness of our findings. Excluding cardiovascular disease (CVD) and abnormal calorie intake did not significantly alter the results (Supplementary Tables 5–6).

4. Discussion

This study sought to explore the relationship between the Oxidative Balance Score (OBS) and migraine prevalence in individuals aged 20 and older in the United States, yielding several significant insights. Notable differences were observed in total OBS, dietary OBS, and lifestyle OBS between participants with and without migraine. A marked inverse correlation was identified between dietary OBS and migraine prevalence. This association maintained its stability, even after adjusting for potential confounding factors.

Subgroup analyses revealed that females and individuals under 60 years showed a heightened response to dietary OBS, although these interactions were not statistically significant. RCS analysis supported this, illustrating a non-linear relationship between dietary OBS and migraine risk. For instance, migraine risk decreased substantially as dietary OBS increased, especially beyond 12. The risk then gradually plateaued when dietary OBS surpassed 20.

In conclusion, our findings suggest that increasing dietary OBS levels could act as a preventive approach against migraine. This contributes valuable understanding to the complex interplay between oxidative balance, dietary habits, and migraine risk among US adults.

The Oxidative Balance Score (OBS) is an aggregate measure combining various dietary and lifestyle elements, indicative of an individual's antioxidant level [23]. Aligning with previous research, many dietary components of the OBS have been shown to have protective effects against migraine. Notable examples include folate [27], iron [28], zinc [29], calcium [30], magnesium [30,24], selenium, niacin [25], riboflavin [31], and dietary fiber [32]. A cross-sectional analysis of the NHANES dataset linked an increase in daily riboflavin intake, from 2.07 to 2.87 mg, with reduced migraine occurrence [31]. Another study identified an L-shaped relationship between dietary niacin intake and migraine risk [25], noting that migraine risk ceased to decline after reaching a daily intake of 21.0 mg of niacin. Additionally, one clinical trial found that adherence to a Mediterranean diet lessened the frequency, duration, and intensity of migraines [33]. These findings collectively suggest that strategic consumption of specific antioxidant dietary components could potentially lower migraine incidence by enhancing antioxidant defense mechanisms.

In the realm of lifestyle factors, research has demonstrated mixed results regarding the association between alcohol consumption, smoking, body mass index (BMI), physical activity, and migraine incidence. For instance, a Danish cross-sectional study found that migraine sufferers were more likely to smoke, engage in less physical activity, have lower educational levels, and be underweight [34]. This correlation may be attributed to severe headaches limiting migraineurs' ability to focus on academic endeavors, engage in physical activities, or affect their appetite due to associated symptoms like nausea [34]. Similarly, data from the Spanish National Health Survey (SNHS) indicated a higher prevalence of migraines in women aged 31 to 50, correlating with lower income, inadequate sleep, deteriorating health, depression, and comorbidities such as chronic neck pain and asthma [35]. However, several studies have failed to establish a link between migraine and levels of physical activity, smoking, or alcohol and coffee consumption [36].

Although no statistically significant association was found between lifestyle OBS and migraine in our study, insights from prior research suggest that adopting healthy lifestyle habits—such as regular meal schedules, adequate hydration, consistent exercise (at least three days a week), and sufficient sleep (7–9 h)—can act as preventive measures against migraine attacks [37]. These findings highlight the crucial role of lifestyle choices in managing and potentially reducing migraine occurrences.

Migraine pathophysiology involves several mechanisms, including cortical inhibition, neurogenic inflammation, and intracranial vasoconstrictive dysfunction [38]. Emerging research points to oxidative stress as a contributing factor in migraine onset and progression. Clinical studies [38–40] have identified significantly elevated plasma levels of oxidative stress markers such as 4-hydroxy-2--nonenal (HNE) [39], malondialdehyde (MDA) [39,40], and 2-hydroxy-8′-deoxyguanosine (8-OHdG) [41] in migraine patients. MDA, a commonly used oxidative stress marker, and HNE are key indicators of lipid peroxidation [42]. ROS can cause oxidative damage to DNA bases, detectable through the oxidation product 8-OHdG, and lead to oxidative stress, mitochondrial dysfunction, and metabolic

impairment [43]. These findings indicate that migraine sufferers may experience elevated oxidative stress levels during attacks, supporting the theory that oxidative stress is involved in migraine pathogenesis.

Studies have also demonstrated that TRPA1 channels in the sensory nerve endings of the trigeminal nerve are activated by reactive oxygen species (ROS) and reactive nitrogen species (RNS) [44]. Transient Receptor Potential Ankyrin 1 (TRPA1) channels [45], sensitive to oxidative, nitrative, and electrophilic stimuli, play a pivotal role in various pain syndromes, including migraines [9]. TRPA1 channel activation triggers oxidative and nitrative stress responses, leading to the release of calcitonin gene-related peptide (CGRP) [9]. This release causes neurogenic inflammation of the meninges and vasodilation, culminating in migraine attacks [9]. Notably, substances that activate TRPA1 channels, like ammonium chloride and formalin [26], can induce migraine or cluster headache attacks. Conversely, some analgesic and antimigraine medications block or desensitize TRPA1 channels. These findings suggest that many migraine triggers and exacerbating factors may be linked to oxidative stress.

Furthermore, it has been hypothesized that migraine constitutes a neuroprotective response to oxidative stress within the brain [46]. During a migraine attack, various physiological processes are triggered, involving increased plasma albumin extravasation, elevated synthesis of endothelial nitric oxide, heightened levels of platelet activating factor (PAF) levels [47], and rising concentrations of brain-derived neurotrophic factor (BDNF) [48], calcitonin gene-related peptide (CGRP) [49], and serotonin in the serum [50]. These changes lead to a cascade that suppresses the production of reactive oxygen species (ROS) and enhances antioxidant defenses. This response encompasses upregulation of antioxidant enzymes, promotion of mitochondrial biogenesis, increased adenosine triphosphate (ATP) production, inhibition of neuronal apoptosis, release of neurotrophic factors, activation of neuroprotection and repair signaling pathways, and reduced cerebral energy demands [51,46]. Thus, this network of processes plays a vital role in countering oxidative stress, contributing significantly to neuroprotection and repair.

This study offers several notable advantages. First, it is the inaugural investigation into the relationship between the Oxidative Balance Score (OBS) and migraine risk, shedding light on the inverse correlation between OBS influenced by diet and lifestyle, and migraine risk. Previous research primarily focused on individual dietary components' impact on migraine risk, overlooking the comprehensive interplay between lifestyle and dietary factors. Second, the study utilizes data from the nationally representative NHANES, enhancing its external validity and generalizability.

5. Limitations

However, acknowledging the potential limitations of our research is essential. The NHANES Pain Questionnaire relies on self-reported severe headache or migraine conditions to define migraine. This approach could not be cross validated against the International Classification of Headache Disorders (ICHD) diagnostic standards, raising concerns about the accuracy of migraine diagnoses. Additionally, the OBS does not account for factors such as mood, sleep patterns, environmental conditions, and other known migraine triggers. This omission might introduce unaccounted-for confounders into the analysis. The lack of specific migraine data hindered our ability to establish correlations between OBS and various migraine parameters, including type, attack frequency, and duration. Moreover, the absence of headache scoring precluded the use of the Visual Analog Scale for assessing headache severity. This study also excluded dietary supplements (apart from dietary iron), which could affect oxidative stress score calculations. Furthermore, the lack of data on preventive migraine medications might introduce some confounding bias. Lastly, the impact of lifestyle-induced OBS on migraine risk remains uncertain. Despite these limitations, our study offers valuable insights by establishing a significant association between OBS and migraine in a substantial population-based prospective cohort study. Conducting further large-scale prospective studies is crucial to reinforce and validate our findings.

6. Conclusion

This study revealed a non-linear relationship between dietary Oxidative Balance Score (OBS) and migraine occurrence in a cohort of 6195 adults from the National Health and Nutrition Examination Survey (NHANES). Notably, a significant reduction in migraine risk was observed with an increase in dietary OBS, particularly beyond the threshold of 12. Beyond a dietary OBS of 20, the decline in migraine risk gradually plateaued. These findings underscore the potential benefits of a moderate intake of antioxidant-rich foods in the management of migraines.

Ethics approval

The work presented in this manuscript is not considered human subjects research because it used only de-identified, publicly available data from the National Health and Nutrition Examination Survey and is therefore not subject to IRB review.

The date on which the research proposal was approved by the ethical committee: Protocol #98-12.

Data availability statement

Data associated with this study has been deposited into a publicly available repository: the US National Health and Nutrition Examination Survey (NHANES), https://www.cdc.gov/nchs/nhanes/index.htm.

CRediT authorship contribution statement

Xinxin Liu: Writing – review & editing, Writing – original draft, Investigation, Formal analysis, Data curation. **Ran Liu:** Writing – review & editing, Investigation, Data curation. **Wenbin Liu:** Writing – review & editing, Data curation. **Hua Rong:** Writing – review & editing, Visualization. **Haoyou Xu:** Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e27426.

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