

A cross-sectional study exploring the relationship between Oxidative Balance Scoreand and 10-Year Atherosclerotic Cardiovascular Disease Risk based on the National Health and Nutrition Examination Survey (2011-2020).

Rumeng Wang

China Academy of Chinese Medical Sciences

kun gao

China Academy of Chinese Medical Sciences

lianhua wang

China Academy of Chinese Medical Sciences

Lizhi Li

China Academy of Chinese Medical Sciences

mei Xue

m17812051089@163.com

China Academy of Chinese Medical Sciences

Research Article

Keywords: Atherosclerotic cardiovascular disease, Oxidative Balance Score, dietary OBS, lifestyle OBS, NHANES, 3

Posted Date: December 20th, 2023

DOI: https://doi.org/10.21203/rs.3.rs-3733893/v1

License: (a) This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

The association between the Oxidative Balance Score (OBS) and the 10-year risk of atherosclerotic cardiovascular disease (ASCVD) remains unclear. The objective of our study was to investigate the relationship between OBS and 10-year ASCVD risk.

Methods

A total of 4,955 participants were included from the National Health and Nutrition Examination Surveys (NHANES). OBS was calculated based on nutritional and lifestyle factors. Multivariable logistic regression analysis was conducted to explore the association between OBS and 10-year ASCVD risk. Linear relationships were examined using restricted cubic spline methods. Stratified analyses were performed to assess the robustness of the results.

Results

We included 4,955 participants, and the results of the multivariable logistic regression analysis showed that, when compared to the first quartile, individuals in the highest quartile of total OBS were negatively associated with 10-year ASCVD risk (OR: 0.59, 95% CI: 0.42-0.83, P = 0.002), with a significant trend (P = 0.003). Lifestyle OBS and dietary OBS were also significantly negatively associated with 10-year ASCVD risk (OR: 0.18, 95% CI: 0.12-0.27; OR: 0.78, 95% CI: 0.63-0.98, P = 0.03), with significant decreasing trends (P for trend < 0.05). Stratified analyses revealed that race and hypertension were effect modifiers in the relationship between OBS and 10-year ASCVD risk (P for interaction < 0.05).

Conclusion

Our study demonstrates a negative linear relationship between higher OBS and 10-year ASCVD risk, suggesting the crucial importance of antioxidant-rich dietary and lifestyle choices in reducing ASCVD risk.

1. Introduction

Atherosclerotic cardiovascular disease (ASCVD) is a severe health condition that poses a significant global burden in terms of both prevalence and mortality, presenting a significant challenge to public health and placing a substantial economic burden on affected individuals ^[1, 2]. In recent years, researchers have increasingly recognized the close relationship between oxidative stress and ASCVD. Under conditions such as smoking, hypertension, hyperglycemia, and hyperlipidemia, there is an imbalance between the body's oxidative and antioxidative systems, leading to the generation of reactive oxygen species (ROS), including superoxide anions, hydrogen peroxide, hydroxyl radicals, and nitrate ions. High oxidative stress conditions can result in various oxidative modifications of lipoprotein phospholipids, which may contribute to the development of ASCVD and its associated complications ^[3–5]. Approximately 90% of cardiovascular risk can be attributed to modifiable risk factors, with lifestyle-related factors accounting for over half of this risk. These factors include lack of physical activity, BMI, smoking, alcohol consumption, and unhealthy dietary behaviors ^[6, 7], all of which can influence the development of ASCVD. Therefore, lifestyle management forms the cornerstone of primary and secondary prevention of ASCVD. We aim to explore the impact of relevant modifiable lifestyle factors in effectively reducing the incidence and mortality rates of ASCVD.

The Oxidative Balance Score (OBS) is utilized to assess an individual's level of oxidative stress by incorporating nutritional and lifestyle factors from their diet ^[8]. The concept of OBS was initially introduced in a 2002 epidemiological study involving a cohort of Belgian male smokers. This study included vitamin C and beta-carotene as antioxidant components and iron as a pro-oxidant component. In this study, a lower OBS score indicated a higher level of oxidative balance in individuals, and the results demonstrated an association between low OBS and higher mortality rates in the population ^[9]. A substantial body of research indicates that certain antioxidant nutrients, such as vitamin C, carotenoids, flavonoids, as well as pro-oxidant nutrients like iron and saturated fatty acids, along with lifestyle factors like smoking, can influence an individual's oxidative stress levels, subsequently affecting the occurrence and progression of various chronic diseases ^[8, 10–12].

However, the relationship between OBS and a 10-year ASCVD risk remains unclear. Therefore, we conducted a crosssectional study based on the NHANES 2011–2020 dataset to explore and analyze the correlation between OBS and the 10-year ASCVD risk among Americans. This correlation suggests that by modifying dietary and lifestyle habits, it may be possible to reduce the 10-year ASCVD risk. Hence, the findings of this study could have a significant impact on the prevention of ASCVD and the improvement of overall quality of life.

2. Materials and Methods

2.1 Study Population: The population data utilized in this study were derived from the National Health and Nutrition Examination Survey (NHANES) conducted in the United States. NHANES employs a meticulously structured multistage sampling design to gather data from a nationally representative sample of U.S. civilians, with the overarching objective of monitoring the health and nutritional status of both adults and children across the entire United States. Comprehensive details regarding the survey's design and methodologies can be accessed through the NHANES website (Centers for Disease Control and Prevention (CDC), http://cdc.gov/nchs/nhanes). All participants provided written informed consent, and NHANES received ethical approval from the National Center for Health Statistics Ethics Review Board. This study amalgamated data spanning a continuous 10-year period from 2011 to 2020 within the NHANES database. Figure 1 elucidates the process of participant selection.

2.2 Oxidative Balance Score (Exposure)

The OBS consists of two main categories: 16 dietary OBS and four lifestyle OBS, encompassing five pro-oxidants and 15 antioxidants. The overall OBS is computed by summing the scores for each variable, where a higher OBS signifies a greater exposure to antioxidants ^[13]. Dietary intake data were gathered through 24-hour dietary recall interviews conducted in mobile examination centers (24HR). Participants were asked to recall and describe the types and amounts of foods and beverages consumed in the 24 hours preceding the interview. The assessment of dietary nutrient intake was based on the University of Texas Food Intake Analysis System and the United States Department of Agriculture Nutrient Database. However, it is essential to note that the estimation of dietary nutrients did not encompass those derived from dietary supplements or medications.

Lifestyle factors associated with OBS include alcohol consumption, smoking, BMI, and physical activity. Alcohol information was derived from the 24-hour dietary recall interview, primarily to determine the average quantity of any alcoholic beverage consumed by participants over the past 12 months. Cotinine, which is a metabolite of nicotine with a longer half-life than nicotine, was employed to assess active smoking status. It also served as an indicator of the extent of exposure to environmental tobacco smoke. BMI is calculated as the weight quotient (in kilograms) divided by the square of height (in meters). Physical activity data were obtained through household interviews and computed as metabolic equivalents, taking into account the frequency and duration of each type of physical activity per week.

2.3 ASCVD Risk Assessment and Definition

The Pooled Cohort Equation model, implemented by the ACC/AHA in 2013, is used to assess the 10-year risk of ASCVD. This model takes into account characteristics such as race, gender, age, systolic blood pressure(SBP), total cholesterol(TC), low density lipoprotein-cholesterol(LDL-C), triglyceride(TG), diabetes, and smoking status ^[14]. In this study, a risk score of 7.5% was used as the threshold for defining the 10-year ASCVD risk. Participants with a risk score \geq 7.5% were categorized as having an elevated 10-year ASCVD risk, while those with a score < 7.5% were classified as low-risk individuals.

2.4 Covariates

In our study, the following variables were considered as covariates: age, gender, race/ethnicity, education level, household income-to-poverty ratio, hypertension status, diabetes status, and biochemical indicators, including triglycerides, total cholesterol, high-density lipoprotein, and low-density lipoprotein. Race was categorized as White, African American, and other races, including multiracial. Education levels included three categories: less than high school, high school graduation, and college or higher. Marital status included divorced/separated/widowed, married/cohabiting, and never married. Hypertension and diabetes were assessed through measurements, medication usage, and self-reported diagnosis.

2.5 Statistical Analysis

For continuous variables, if they followed a normal distribution, they were presented as mean ± standard deviation (Mean ± SD) and compared using the t-test. If the distribution was skewed, they were represented by the median (quartiles) and compared using the Mann-Whitney U test. Categorical variables were expressed as counts (%) and compared using the Rao-Scott chi-square test to compare baseline characteristics between the 10-year ASCVD low-risk and high-risk groups.

In this study, logistic regression models were employed to determine the association between OBS and the 10-year ASCVD risk. Results were presented as odds ratios (OR) and 95% confidence intervals (CI). In the multivariate models, Model 1 adjusted for age, gender, and race. In Model 2, further adjustments were made for education level, marital status, household income-to-poverty ratio, BMI, and waist circumference. Model 3 involved comprehensive adjustments, building upon Model 2 by including SBP, diastolic blood pressure(DBP), TC, LDL-C, TG, high density lipoprotein-cholesterol(HDL-C), white blood cell(WBC), diabetes, and hypertension. Notably, since both OBS and the 10-year ASCVD model included smoking, we decided not to include smoking in the covariates in the multivariate regression models to reduce potential multicollinearity issues. Restricted Cubic Splines (RCS) were used to explore the linear relationship between OBS and the 10-year ASCVD. Additionally, we performed subgroup analyses with multivariate logistic regression models for covariates (age, gender, race, hypertension, and diabetes). All statistical analyses were conducted using R 4.1.2, with statistical significance defined as P < 0.05.

3. Results

3.1 Baseline Characteristics

A total of 4,955 participants were included in this study, comprising 2,532 males and 2,423 females, with an average age of 58 (48–65) years. The participant selection process is depicted in Fig. 1. The baseline levels of OBS, dietary OBS, and lifestyle OBS were 21.0 (15.0, 26.0), 17.0 (11.0, 22.0), and 4.0 (3.0, 5.0), respectively. Table 1 presents the baseline characteristics of the two groups, demonstrating significant differences in baseline data between the groups with 10-year ASCVD < 7.5% and 10-year ASCVD \geq 7.5%, including age, gender, race, education level, marital status, income, BMI,

waist circumference, SBP, DBP, TC, LDL-C, TG, HDL-C, WBC, OBS, dietary OBS, lifestyle OBS, and the presence of hypertension and diabetes (P < 0.05).

LDL-C, (mg/dl)	113.0 (89.0, 138.0)	111.0 (85.0, 138.0)	115.0 (93.0, 138.0)	⊠ 0.00′	
Characteristics	Total	10- year risk of ASCVD < 7.5%	10-year risk of ASCVD $≥$ 7.5%	Ρ	
	n = 4955	n = 2699	n = 2256		
Age (years)	58.0 (48.0, 65.0)	50.0 (45.0, 56.0)	66.0 (61.0, 71.0)	< 0.001	
Gender n(%)	2423(48.9)	969(35.9)	1454(54.5)	< 0.00 ²	
Male	2532(51.1)	1730(64.1)	802(35.5)		
Female					
Race n(%)	1972 (39.8)	1032 (38.2)	940 (41.7)	<	
White	1193 (24.1)	627 (23.2)	566 (25.1)	0.001	
African American	1790 (36.1)	1040 (38.5)	750 (33.2)		
Others					
Marital status, n (%)	3190 (64.4)	1755 (65)	1435 (63.6)	< 0.001	
Married/Living with Partner	1313 (26.5)	634 (23.5)	679 (30.1)		
Widowed/Divorced/Separated	452 (9.1)	310 (11.5)	142 (6.3)		
Never married					
Education level, n (%) 929(18.7) 411(15.2) 518(23)					
Did not graduate from high	1085(21.9)	529(19.6)	556(24.6)	0.001	
school	2941(59.4)	1759(65.2)	1182(52.4)		
Graduated from high school					
College education or above					
PIR	2.5(1.3, 4.6)	2.8 (1.3, 4.9)	2.3 (1.2, 4.2)	< 0.00	
BMI, (kg/m2)	28.8 (25.2, 33.5)	28.4 (24.8, 33.4)	29.2 (25.6, 33.6)	0.00	
Waist	100.8 (91.5, 111.7)	98.2(88.8, 108.8)	104.2(94.8, 114.1)	< 0.00	
Hypertension, n (%)	2373 (47.9)	1677 (62.1)	696 (30.9)	<	
No	2582 (52.1)	1022 (37.9)	1560 (69.1)	0.001	
Yes					
Diabetes, n (%)	1341 (27.1)	355 (13.2)	986 (43.7)	< 0.001	
No	3614 (72.9)	2344 (86.8)	1270 (56.3)		
Yes					
SBP, (mmHg)	124.7(114.0,137.0)	119.0(110.0,129.0)	131.0(121.0,144.0)	< 0.00	

LDL-C, (mg/dl)	113.0 (89.0, 138.0)	111.0 (85.0, 138.0)	115.0 (93.0, 138.0)	⊠ 0.001
DBP, (mmHg) DBP	72.3 (65.0, 79.5)	73.0 (66.0, 79.5)	71.0 (63.3, 79.4)	⊠ 0.001
TC, (mg/dl)	192.0 (164.0, 219.0)	188.0 (161.0, 218.0)	194.0 (168.0, 219.0)	⊠ 0.001
TG, (mg/dl)	99.0 (69.0, 144.0)	91.0 (64.0, 131.0)	111.0 (78.0, 155.0)	< 0.001
HDL-C, (mg/dl)	52.0 (43.0, 64.0)	54.0 (46.0, 66.0)	49.0 (41.0, 61.0)	< 0.001
WBC(1000/µ)	6.4 (5.3, 7.8)	6.1 (5.1, 7.6)	6.6 (5.5, 7.9)	< 0.001
OBS	21.0 (15.0, 26.0)	22.0 (16.0, 27.0)	19.0 (14.0, 24.0)	< 0.001
Dietary OBS	17.0 (11.0, 22.0)	18.0 (12.0, 22.0)	15.0 (10.0, 21.0)	< 0.001
Lifestyle OBS	4.0 (3.0, 5.0)	4.0 (3.0, 5.0)	4.0 (3.0, 5.0)	< 0.001

PIR, ratio of family income to poverty; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC:total cholesterol;LDL-C: low density lipoprotein-cholesterol; TG:triglyceride;HDL-C:high density lipoprotein-cholesterol;WBC:white blood cell; OBS: oxidative balance score.

3.2 Association between OBS and 10-year ASCVD Risks

We employed logistic regression modeling to explore the correlation between OBS and 10-year ASCVD risk. Table 2. Initially, when OBS was treated as a continuous variable, and after adjusting for age, gender, race, education level, marital status, PIR, BMI, waist circumference, SBP, DBP, TC, LDL-C, TG, HDL-C, WBC, diabetes, and hypertension, the results revealed a negative correlation between OBS and 10-year ASCVD (OR: 0.97, 95% CI: 0.95–0.99). When OBS was stratified into quartiles, the highest quartile of OBS showed a lower risk of 10-year ASCVD (OR: 0.49, 95% CI: 0.42–0.58, P < 0.01). In the fully adjusted model (Model 3), compared to the first quartile, individuals in the highest quartile of OBS exhibited a negative association with 10-year ASCVD (OR: 0.59, 95% CI: 0.42–0.83, P = 0.002), with a significant trend (P = 0.003).

Similarly, lifestyle OBS demonstrated a significant negative correlation with 10-year ASCVD risk. After adjusting for all confounding factors, individuals in the second, third, and fourth quartiles had a significantly lower risk of 10-year ASCVD compared to the lowest reference quartile, with a significant trend. Similarly, we observed a significant negative association between lifestyle OBS and 10-year ASCVD risk. After adjusting for all confounding factors, individuals in the second, third, and fourth quartiles of lifestyle OBS had a 10-year ASCVD risk of (OR: 0.59, 95% CI: 0.39–0.88, P = 0.01; OR: 0.4, 95% CI: 0.26–0.61, P < 0.001; OR: 0.18, 95% CI: 0.12–0.28, P < 0.001), with a significant trend. Similarly, participants in the highest quartile of dietary OBS had a lower 10-year ASCVD risk (OR: 0.78, 95% CI: 0.63–0.98, P = 0.03), with a significant decreasing trend (P for trend P = 0.005).

Multivariable-adjust ORs and 95% CI of the VAI and LAP quartiles associated with elevated 10-year ASCVD risk. **3st Ouartile** 0.7 (0.59 0.72 (0.6 0.93 0.49 0.95 0.649 < < ~ 0.82) 0.001 ~ 0.85) 0.001 (0.77~ (0.76~ **1.13**) **1.18**) Ρ Ρ Ρ Non-Ρ Model 1 Model 2 Model 3 value value value Adjusted value OR (95% OR (95% OR (95% OR (95% Cls) Cls) Cls) Cls) OBS(continuous) 0.96 0.95 0.97 0.97(0.95 0.001 < < < (0.95~ 0.001 (0.94~ 0.001 (0.96~ 0.001 ~ 0.99 0.97) Ò.97) Ò.99) **OBS**(quartile) **1st Quartile** 1.0 (ref) 1.0 (ref) 1.0 (ref) 1.0 (ref) 0.766 2st Quartile 0.88 0.114 0.89 0.363 0.96 0.88 0.43 (0.75~ (0.68~ $(0.73 \sim$ (0.63~ 1.03) 1.15) 1.26) 1.22) **3st Quartile** 0.72 0.78 (0.6 0.07 0.94 0.672 0.92 0.614 < (0.61~ 0.001 ~ 1.02) (0.71~ (0.66~ 0.84) 1.24) 1.28) 0.49 0.43 0.56 4st Quartile 0.59 0.002 < < < 0.001 0.001 (0.42~ (0.42~ (0.33~ 0.001 (0.43~ Ò.58) Ò.56) Ò.74) Ò.83) P for trend 0.003 < < < 0.001 0.001 0.001 OBS.Lifestyle(continuous) OBS.Lifestyle(quartile) 0.87 0.63(0.59 0.69(0.65 0.64(0.58 < < < < 0.001 (0.84~ 0.001 0.001 0.001 ~ 0.67) ~ 0.75 ~ 0.71) Ò.9) **1st Ouartile** 1.0 (ref) 1.0 (ref) 1.0 (ref) 1.0 (ref) 0.034 0.6 (0.44 0.001 0.67 (0.5 0.012 0.59 0.01 2st Ouartile 0.82 (0.39~ (0.68~ ~ 0.81) ~ 0.92) Ò.98) Ò.88) **3st Quartile** 0.62 0.35 0.46 0.4 (0.26 < < < < (0.51~ 0.001 (0.26~ 0.001 (0.33~ 0.001 ~ 0.61) 0.001 Ò.75) Ò.48) Ò.63) 0.57 0.27 (0.2 4st Quartile 0.18 0.18(0.12 < < < < (0.48~ 0.001 (0.13~ 0.001 0.001 0.001 ~ 0.36) ~ 0.28) 0.24) 0.67) P for trend < < < < 0.001 0.001 0.001 0.001 0.96(0.95 0.97(0.96 0.98(0.97 OBS.dietary(continuous) < 0.98 0.001 < < 0.001 0.001 ~ 0.97) 0.001 ~ 0.98) $(0.97 \sim$ ~ 0.99 99) OBS.dietary(quartile) 1.0 (ref) **1st Quartile** 1.0 (ref) 1.0 (ref) 1.0 (ref)

Table 2

3st Quartile	0.7 (0.59 ~ 0.82)	< 0.001	0.72 (0.6 ~ 0.85)	< 0.001	0.93 (0.77 ~ 1.13)	0.49	0.95 (0.76 ~ 1.18)	0.649
2st Quartile	0.87 (0.74 ~ 1.02)	0.089	0.72 (0.76 ~ 1.06)	0.193	1.06 (0.88 ~ 1.28)	0.539	1.15 (0.93 ~ 1.42)	0.203
4st Quartile	0.53 (0.45 ~ 0.62)	< 0.001	0.56 (0.47 ~ 0.66)	< 0.001	0.74 (0.61 ~ 0.9)	0.003	0.78(0.63 ~0.98)	0.03
P for trend		< 0.001		< 0.001		0.001		0.00

Model 1 adjust for Age, Gender, Race.

Model 2 adjust for Model 1 + Education level, Marital status, PIR, BMI, Waist.

Model 3 adjust for Model 1 + Model 2 + SBP, DBP, TC, LDL-C, TG, HDL-C, WBC, Diabetes, Hypertension.

PIR, ratio of family income to poverty; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC:total cholesterol;LDL-C: low density lipoprotein-cholesterol; TG:triglyceride;HDL-C:high density lipoprotein-cholesterol;WBC:white blood cell; OBS: oxidative balance score.

3.3 Restricted Cubic Splines (RCS)

RCS were utilized to describe the association between OBS, dietary OBS, lifestyle OBS, OBS.lifestyle, and the 10-year ASCVD risk. Figure 2. The results indicated a negative linear relationship between OBS, dietary OBS, lifestyle OBS, and the 10-year ASCVD risk, suggesting that an increase in OBS, dietary OBS, and lifestyle OBS is associated with a decrease in the 10-year ASCVD risk (P for non-linearity: 0.333; P for non-linearity: 0.563; P for non-linearity: 0.414).

3.4 Subgroup Analysis

We conducted a multivariate stratified analysis of OBS with 10-year ASCVD to assess potential effect modification in the relationship between OBS and elevated 10-year ASCVD risk. Figure 3. Within all strata, we found that race and hypertension were effect modifiers in the relationship between OBS and 10-year ASCVD risk (P for interaction < 0.05). However, no interaction was observed in the subgroups when stratified by age, gender, and diabetes (P for interaction > 0.05).

4. Discussion

In this study, we conducted a cross-sectional investigation involving 4,599 participants from the NHANES database to evaluate the association between OBS and 10-year ASCVD risk. Our study findings indicate that, after adjusting for relevant confounding variables, overall OBS, dietary OBS, and lifestyle OBS are inversely associated with 10-year ASCVD risk, whether considered as continuous or categorical variables. This implies that higher OBS levels are associated with a reduced risk of ASCVD, underscoring the significance of antioxidant dietary patterns and lifestyles in the context of ASCVD. Furthermore, subgroup analyses revealed that race and hypertension act as effect modifiers in the relationship between OBS and 10-year ASCVD risk.

Cardiovascular disease (CVD) is a significant threat to human life and health. Data shows that CVD is the leading cause of death in the global population aged 35–70, accounting for 40% of all deaths ^[14, 15]. Atherosclerotic cardiovascular disease (ASCVD) was first introduced by the ACC/AHA in 2013, encompassing ischemic or endothelial dysfunction-inflammatory diseases caused by atherosclerosis, such as coronary heart disease, ischemic stroke, and diabetes-

related atherosclerotic vascular diseases ^[16]. Risk assessment forms the basis for primary prevention of ASCVD, as identifying the presence and severity of atherosclerotic lesions before clinical symptoms appear is essential for managing and treating risk factors and preventing further disease progression. The premise of early prevention of cardiovascular disease is the proper assessment of cardiovascular disease risk. The most commonly used and extensively validated risk assessment models internationally and domestically are the Framingham 10-year risk score ^[17] and the Pooled Cohort Equation for ASCVD risk calculation, developed by the ACC/AHA. The Framingham 10-year risk score was developed based on studies in the Caucasian population, limiting its applicability to specific ethnic groups and assessing only coronary heart disease risk. In contrast, the Pooled Cohort Equation assesses the 10-year fatal or non-fatal ASCVD risk, including ischemic heart disease, non-fatal myocardial infarction, coronary heart disease, and fatal and non-fatal stroke. This scoring system was developed based on extensive, multi-ethnic data and draws from large-scale community-based population studies. Previous research has confirmed the significant value of the 10vear ASCVD risk score in estimating cardiovascular disease risk ^[18]. The 10-year ASCVD risk score applies to adults aged 40-79, and according to the guidelines for risk management, a score below 7.5% indicates low risk, while a score equal to or above 7.5% indicates high risk. Individuals at high risk must choose appropriate intervention measures based on their medical history and lipid profile. Assessing an individual's 10-year ASCVD risk can guide preventive interventions matched to the individual, maximizing the expected benefits of prevention while minimizing the potential harm of over-treatment.

Oxidative stress (OS) is a series of adaptive responses in the body resulting from the imbalance between reactive oxygen species (ROS) and the antioxidant system ^[19]. ROS readily reacts with small molecules such as low-density lipoprotein (LDL) to form oxidized low-density lipoprotein (ox-LDL) ^[20]. Driven by chemotactic factors (e.g., matrix-derived factors, macrophage chemoattractants), ox-LDL, as a source of OS, is directed toward the heart and blood vessels. Under the action of adhesion factors (e.g., plasminogen activator inhibitor), it adheres to the vascular wall, leading to arterial hardening and narrowing, ultimately resulting in the development of fatal cardiovascular diseases such as hypertension, atherosclerosis, and coronary heart disease ^[21–23]. In recent years, changes in dietary habits and lifestyles have led to the body remaining in a state of continuous oxidative stress. As ROS acts as a critical signaling factor in the progression of inflammatory diseases, its overexpression can disrupt the balance between ROS generation and the antioxidant system, leading to immune activation, insulin resistance, endothelial cell damage, and the release of inflammatory mediators ^[24].

Diet plays a crucial role in regulating systemic inflammation and oxidative stress. Poor dietary habits can increase systemic inflammation and oxidative stress, leading to weight gain enlargement of fat cells, promoting the progression of atherosclerosis, and ultimately resulting in fatalities among patients with atherosclerotic vascular diseases ^[25–27]. Research has found that dietary antioxidant components primarily include vitamins A, C, and E carotenoids, which have antioxidative, antiproliferative, anti-inflammatory, and cholesterol-lowering properties ^[28]. They also contain compounds such as retinol, vitamin D, zinc, calcium, flavonoids, and total catechins, but there is relatively limited research on these components. As for dietary pro-oxidants, details typically consist of iron, unsaturated fatty acids, and fatty acids ^[8]. Therefore, a balanced dietary pattern, such as the Mediterranean diet, which emphasizes the consumption of vegetables, fruits, fish, whole grains, legumes, and olive oil, offers the body an optimal balance to maintain antioxidative capacity, reducing the risk of cardiovascular diseases ^[29–31]. Several studies have shown that an antioxidant-rich dietary pattern can lower the risk of ASCVD ^[32–34], which aligns with our research findings.

Modifying a single factor in one's lifestyle can influence the risk of cardiovascular diseases, and multiple lifestyle changes can have a synergistic effect. In addition to dietary components, the impact of lifestyle on ASCVD cannot be underestimated. An increasing body of research indicates a connection between sedentary behavior and the incidence

and mortality rates of cardiovascular diseases in adults ^[35, 36]. Maintaining a healthy body weight is a critical step in preventing cardiovascular diseases. Studies have shown that overweight and obesity significantly increase the risk of cardiovascular disease incidence and mortality ^[37, 38]. Therefore, weight control is an effective strategy for reducing the risk of cardiovascular diseases. Engaging in regular, long-term exercise not only effectively manages body weight but also reduces the release of inflammatory factors (e.g., IL-6/IL-10/TNF-a), protecting endothelial cells and thereby lowering the risk of cardiovascular diseases ^[39]. However, a study involving nearly 1.5 million individuals ^[40, 41] found that vigorous exercise in areas with severe air pollution could increase cardiovascular diseases through physical activity. Smoking is a significant risk factor for ASCVD ^[42], given the diversity and complexity of compounds in tobacco smoke. Research on the association between representative compounds in tobacco smoke, such as nicotine and its metabolites, and ASCVD risk is currently limited. Cotinine is a metabolite of nicotine ^[43] with a longer half-life, which allows for a better assessment of nicotine exposure. The dangers of passive smoking are equally important. Brief exposure to secondhand smoke can increase platelet activity, damage endothelial cells, promote inflammation and oxidative stress responses, increase endothelial permeability, and, consequently, raise the risk of acute myocardial infarction ^[44].

Through further subgroup analysis, we found that the negative correlation between OBS and 10-year ASCVD risk was generally more significant in the white population and those without hypertension, with interactions being present. It is well-known that compared to the black population, white individuals have a lower prevalence of cardiovascular risk factors (such as hypertension, dyslipidemia, smoking, and physical inactivity), suggesting a lower incidence of adverse cardiovascular events in white populations ^[45]. However, the recent inclusion of race in the PCE equation for assessing ASCVD risk has been questioned due to biological differences, which may ultimately lead to treatment decisions based solely on racial disparities. Thus, it is recommended to develop race-free CVD risk prediction algorithms ^[46]. In individuals without hypertension, the negative correlation between OBS and 10-year ASCVD risk is more pronounced, indicating that antioxidant-rich diets and healthy lifestyles confer incredible benefits in participants without hypertension. Studies have shown that antioxidant-rich diets, including carotenoids, vitamin E, and magnesium supplements, are negatively associated with hypertension ^[47-49].

This is the first study to explore the relationship between OBS and 10-year ASCVD risk. The results indicate a negative association between both overall OBS and dietary OBS and lifestyle OBS with ASCVD risk, which could contribute to the development of cardiovascular disease prevention strategies in clinical practice. However, our study has several limitations. First, due to the cross-sectional design, it is impossible to establish causality in the relationship between OBS and 10-year ASCVD risk. Therefore, extensive prospective studies are needed to elucidate the causal relationship between these factors. Second, using self-reported 24-hour dietary recall to obtain dietary data may introduce recall bias. Additionally, despite adjusting for some potential covariates, we cannot eliminate the influence of other potential confounding factors.

5. Conclusion

In summary, our study revealed a negative association between OBS and the initial 10-year ASCVD risk. This finding suggests that antioxidant-rich diets and lifestyles potentially influence cardiovascular disease.

Declarations

Authors' contributions

RMW and KG contributed equally to this work. LZL and MX conceptualized this research aim, planned the analyses and guided the literature review. LHW extracted the data from the NHANES database. XG and SFH participated in processing and analyzing the data. RMW wrote the first draft of the paper. MX revised and commented on the draft and overall responsibility. All the authors have read and approved the final manuscript.

Funding

This work was supported by the Major Research Project of Scientific and Technological Innovation Project of Chinese Academy of Chinese Medicine Sciences (No.CI2021A00913) and the National Natural Science Foundation of China (grant number 81973686).

Availability of data and materials

Publicly available datasets were analyzed in this study. This data can be found here: www.cdc.gov/nchs/nhanes/.

Ethics approval and consent to participate

The studies involving humans were approved by the National Center for Health Statistics Ethics review Committee. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

Competing interests

The authors declare that they have no competing interests.

References

- Sacco RL, Roth GA, Reddy KS, et al. The Heart of 25 by 25: Achieving the Goal of Reducing Global and Regional Premature Deaths From Cardiovascular Diseases and Stroke: A Modeling Study From the American Heart Association and World Heart Federation[J]. Circulation. 2016;133(23):e674–90.
- 2. Zheng WC, Chan W, Dart A et al. Novel therapeutic targets and emerging treatments for atherosclerotic cardiovascular disease[J]. Eur Heart J Cardiovasc Pharmacother, 2023.
- 3. Petrucci G, Rizzi A, Hatem D et al. Role of Oxidative Stress in the Pathogenesis of Atherothrombotic Diseases[J]. Antioxidants (Basel), 2022,11(7).
- 4. Pantazi D, Tellis C, Tselepis AD. Oxidized phospholipids and lipoprotein-associated phospholipase A(2) (Lp-PLA(2)) in atherosclerotic cardiovascular disease: An update[J]. BioFactors. 2022;48(6):1257–70.
- 5. Stocker R, Keaney JJ. Role of oxidative modifications in atherosclerosis[J]. Physiol Rev. 2004;84(4):1381–478.
- 6. Kirkpatrick CF, Maki KC. Dietary Influences on Atherosclerotic Cardiovascular Disease Risk[J]. Curr Atheroscler Rep. 2021;23(10):62.
- 7. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study[J]. Lancet. 2004;364(9438):937–52.
- 8. Hernández-Ruiz Á, García-Villanova B, Guerra-Hernández E et al. A Review of A Priori Defined Oxidative Balance Scores Relative to Their Components and Impact on Health Outcomes[J]. Nutrients, 2019,11(4).
- 9. Van Hoydonck PG, Temme EH, Schouten EG. A dietary oxidative balance score of vitamin C, beta-carotene and iron intakes and mortality risk in male smoking Belgians[J]. J Nutr. 2002;132(4):756–61.
- 10. Omidifar N, Moghadami M, Mousavi SM et al. Trends in Natural Nutrients for Oxidative Stress and Cell Senescence[J]. Oxid Med Cell Longev, 2021,2021:7501424.

- 11. Puntarulo S. Iron, oxidative stress and human health[J]. Mol Aspects Med. 2005;26(4-5):299-312.
- 12. Salem AA, Trares K, Kohl M et al. Long-term effects of smoking on serum concentrations of oxidative stress biomarkers: Results of a large, population-based cohort study[J]. Environ Res, 2022,204(Pt A):111923.
- 13. Zhang W, Peng SF, Chen L, et al. Association between the Oxidative Balance Score and Telomere Length from the National Health and Nutrition Examination Survey 1999–2002[J]. Oxid Med Cell Longev. 2022;2022:1345071.
- Dagenais GR, Leong DP, Rangarajan S, et al. Variations in common diseases, hospital admissions, and deaths in middle-aged adults in 21 countries from five continents (PURE): a prospective cohort study[J]. Lancet. 2020;395(10226):785–94.
- 15. Roth GA, Mensah GA, Johnson CO, et al. Global Burden of Cardiovascular Diseases and Risk Factors, 1990–2019: Update From the GBD 2019 Study[J]. J Am Coll Cardiol. 2020;76(25):2982–3021.
- 16. Goff DJ, Lloyd-Jones DM, Bennett G et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines[J]. J Am Coll Cardiol, 2014,63(25 Pt B):2935–59.
- 17. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories[J]. Circulation. 1998;97(18):1837–47.
- 18. Ridker PM, Cook NR. Statins: new American guidelines for prevention of cardiovascular disease[J]. Lancet. 2013;382(9907):1762–5.
- 19. Pu Y, Cheng CK, Zhang H, et al. Molecular mechanisms and therapeutic perspectives of peroxisome proliferatoractivated receptor α agonists in cardiovascular health and disease[J]. Med Res Rev. 2023;43(6):2086–114.
- 20. Gao S, Zhao D, Wang M, et al. Association Between Circulating Oxidized LDL and Atherosclerotic Cardiovascular Disease: A Meta-analysis of Observational Studies[J]. Can J Cardiol. 2017;33(12):1624–32.
- 21. Libby P. The changing landscape of atherosclerosis[J]. Nature. 2021;592(7855):524–33.
- 22. Correction to. Oxidized Phospholipids Promote NETosis and Arterial Thrombosis in LNK(SH2B3). Deficiency[J] Circulation. 2022;146(4):e13.
- 23. Marchio P, Guerra-Ojeda S, Vila JM et al. Targeting Early Atherosclerosis: A Focus on Oxidative Stress and Inflammation[J]. Oxid Med Cell Longev, 2019,2019:8563845.
- 24. Niki E. Oxidant-specific biomarkers of oxidative stress. Association with atherosclerosis and implication for antioxidant effects[J]. Free Radic Biol Med. 2018;120:425–40.
- 25. Bondonno NP, Lewis JR, Blekkenhorst LC, et al. Dietary inflammatory index in relation to sub-clinical atherosclerosis and atherosclerotic vascular disease mortality in older women[J]. Br J Nutr. 2017;117(11):1577–86.
- 26. Wu L, Noyan AM, Facci M, et al. Dietary approach to attenuate oxidative stress, hypertension, and inflammation in the cardiovascular system[J]. Proc Natl Acad Sci U S A. 2004;101(18):7094–9.
- 27. Incalza MA, D'Oria R, Natalicchio A, et al. Oxidative stress and reactive oxygen species in endothelial dysfunction associated with cardiovascular and metabolic diseases[J]. Vascul Pharmacol. 2018;100:1–19.
- 28. Pérez-Jiménez J, Neveu V, Vos F, et al. Identification of the 100 richest dietary sources of polyphenols: an application of the Phenol-Explorer database[J]. Eur J Clin Nutr. 2010;64(Suppl 3):112–S120.
- 29. Azzini E, Polito A, Fumagalli A, et al. Mediterranean Diet Effect: an Italian picture[J]. Nutr J. 2011;10:125.
- 30. Trujillo-Mayol I, Guerra-Valle M, Casas-Forero N, et al. Western Dietary Pattern Antioxidant Intakes and Oxidative Stress: Importance During the SARS-CoV-2/COVID-19 Pandemic[J]. Adv Nutr. 2021;12(3):670–81.
- 31. Abbasalizad FM, Najafi M. Empirically developed dietary inflammatory potential (EDIP) in patients candidate for coronary artery bypass grafting surgery (CABG): Association with metabolic parameters, dietary antioxidant quality score and dietary phytochemical index[J]. PLoS ONE. 2018;13(12):e208711.

- 32. Zhang J, Lu X, Wu R, et al. Associations between composite dietary antioxidant index and estimated 10-year atherosclerotic cardiovascular disease risk among U.S. adults[J]. Front Nutr. 2023;10:1214875.
- 33. Liu C, Lai W, Zhao M et al. Association between the Composite Dietary Antioxidant Index and Atherosclerotic Cardiovascular Disease in Postmenopausal Women: A Cross-Sectional Study of NHANES Data, 2013–2018[J]. Antioxidants (Basel), 2023,12(9).
- 34. Charitha KS, Sikand G, Agarwala A. Optimizing Dietary Patterns and Lifestyle to Reduce Atherosclerotic Cardiovascular Risk Among South Asian Individuals[J]. Am J Cardiol. 2023;203:113–21.
- 35. Young DR, Hivert MF, Alhassan S, et al. Sedentary Behavior and Cardiovascular Morbidity and Mortality: A Science Advisory From the American Heart Association[J]. Circulation. 2016;134(13):e262–79.
- 36. Liang ZD, Zhang M, Wang CZ, et al. Association between sedentary behavior, physical activity, and cardiovascular disease-related outcomes in adults-A meta-analysis and systematic review[J]. Front Public Health. 2022;10:1018460.
- 37. Dwivedi AK, Dubey P, Cistola DP, et al. Association Between Obesity and Cardiovascular Outcomes: Updated Evidence from Meta-analysis Studies[J]. Curr Cardiol Rep. 2020;22(4):25.
- 38. Iso H, Cui R, Takamoto I, et al. Risk Classification for Metabolic Syndrome and the Incidence of Cardiovascular Disease in Japan With Low Prevalence of Obesity: A Pooled Analysis of 10 Prospective Cohort Studies[J]. J Am Heart Assoc. 2021;10(23):e20760.
- 39. Pedersen BK. Anti-inflammatory effects of exercise: role in diabetes and cardiovascular disease[J]. Eur J Clin Invest. 2017;47(8):600–11.
- 40. Kim SR, Choi S, Keum N, et al. Combined Effects of Physical Activity and Air Pollution on Cardiovascular Disease: A Population-Based Study[J]. J Am Heart Assoc. 2020;9(11):e13611.
- 41. Kim SR, Choi S, Kim K, et al. Association of the combined effects of air pollution and changes in physical activity with cardiovascular disease in young adults[J]. Eur Heart J. 2021;42(25):2487–97.
- 42. Lechner K, von Schacky C, McKenzie AL, et al. Lifestyle factors and high-risk atherosclerosis: Pathways and mechanisms beyond traditional risk factors[J]. Eur J Prev Cardiol. 2020;27(4):394–406.
- 43. Kermah D, Shaheen M, Pan D, et al. Association between secondhand smoke and obesity and glucose abnormalities: data from the National Health and Nutrition Examination Survey (NHANES 1999–2010)[J]. BMJ Open Diabetes Res Care. 2017;5(1):e324.
- 44. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking[J]. Circulation. 2005;111(20):2684–98.
- 45. Kunutsor SK, Khunti K, Seidu S. Racithnic and regional differences in the effect of sodium-glucose co-transporter 2 inhibitors and glucagon-like peptide 1 receptor agonists on cardiovascular and renal outcomes: a systematic review and meta-analysis of cardiovascular outcome trials[J]. J R Soc Med, 2023:323481322.
- 46. Vasan RS, Rao S, van den Heuvel E. Race as a Component of Cardiovascular Disease Risk Prediction Algorithms[J]. Curr Cardiol Rep; 2023.
- 47. Wu M, Si J, Liu Y, et al. Association between composite dietary antioxidant index and hypertension: insights from NHANES[J]. Clin Exp Hypertens. 2023;45(1):2233712.
- 48. Li Z, Chen J, Zhang D. Association between dietary carotenoid intakes and hypertension in adults: National Health and Nutrition Examination Survey 2007–2014[J]. J Hypertens. 2019;37(12):2371–9.
- 49. Zhang Y, Yang S, Wu Q, et al. Dietary vitamin E intake and new-onset hypertension[J]. Hypertens Res. 2023;46(5):1267–75.

Figures

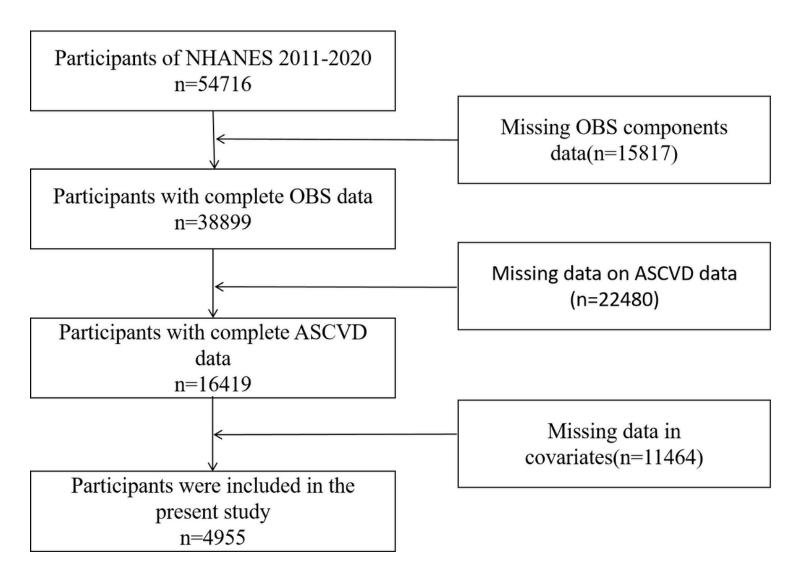


Figure 1

Flowchart of study population screening.

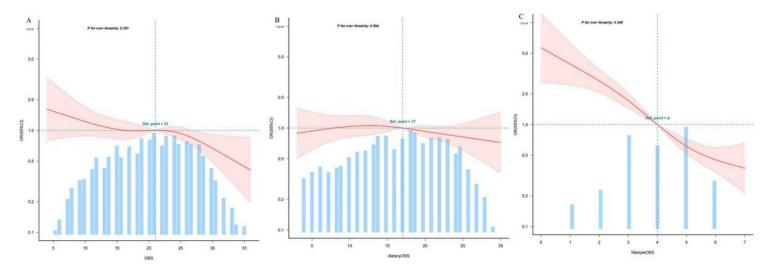


Figure 2

(A)RCS analysis of the association between OBS and elevated 10-year ASCVD risk.

(B)RCS analysis of the association between lifestyle OBS and elevated 10-year ASCVD risk.

Subgroup	Total	Event(%)		OR(95%CI)	P for interaction
Age			- 195		
<65	3576	973 (27.2)		0.99 (0.97~1)	0.518
=65	1379	1283 (93)	•••	0.97 (0.93~1,0)	
Gender					
Female	2532	802 (31.7)		0.97 (0.95~1)	0.85
Male	2423	1454 (60)		0.98 (0.95~1)	
Race					
White	1972	940 (47.7)	 -	0.94 (0.91~0.97)	0.003
African American	1193	566 (47.4)		1.01 (0.97~1.04)	
Others	1790	750 (41.9)		0.99 (0.96~1.02)	
Hypertension					
No	2373	696 (29.3)	••••	0.95 (0.92~0.97)	<0.001
Yes	2582	1560 (60.4)	F	1 (0.97~1.03)	
Diabetes					
No	3614	1270 (35.1)		0.97 (0.95~0.99)	0.965
Yes	1341	986 (73.5)	F	0.98 (0.94~1.01)	

(C)RCS analysis of the association between dietary OBS and elevated 10-year ASCVD risk.

Figure 3

Stratified analyses between OBS and 10-year risk of ASCVD using logistic regression. Each subgroup was adjusted for all other variables except the grouping factor itself.