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Composite dietary antioxidant index of antioxidant vitamins and sarcopenia risk: insights from the UK biobank and NHANES cohorts

HuiMin Liu^{1†}, YuDi Xu^{1†}, QingSheng Li¹, LingFei Yang¹, Xuan Yang¹, KaiXin Wang¹, Zhe Gong¹, Qiang Zhang^{2*} and YanJie Jia^{1*}

Abstract

Background The composite dietary antioxidant index (CDAI), reflecting total dietary intake of antioxidant vitamins, may indicate overall antioxidant capacity. This study examined its association with the risk of probable sarcopenia, defined by handgrip strength, in older adults.

Methods Participants aged over 60 from the UK Biobank ($N=22,921$) and National Health and Nutrition Evaluation Surveys (NHANES) 2011–2014 ($N=2,641$) cohorts were categorized into probable sarcopenia and non-probable sarcopenia groups. Multivariable logistic regression models assessed the associations between CDAI (both continuous and quartile) and its components (vitamin A, vitamin C, vitamin E, and carotene) with probable sarcopenia risk in cohorts, with sex subgroup and sensitivity analyses to validate results.

Results The median (interquartile range) of CDAI was -0.39 ($-1.88, 1.45$) in the UK Biobank and -0.57 ($-1.60, 0.84$) in NHANES, respectively. A higher CDAI was significantly associated with a lower risk of probable sarcopenia in both cohorts. Specifically, each one-unit increase in CDAI was associated with a 2% decrease in the odds of probable sarcopenia in the UK Biobank ($OR=0.98$, 95% $CI=0.97-0.998$, $p=0.027$) and a 13.5% decrease in NHANES ($OR=0.865$, 95% $CI=0.75-0.997$, $p=0.045$), after full adjustment under the Sarcopenia Definition and Outcomes Consortium (SDOC) criteria. In quartile analyses, the risk of probable sarcopenia tended to decrease across higher CDAI quartiles, although the dose–response trend was not strictly linear. In the UK Biobank, multivariable-adjusted odds ratios (95% CI s) across increasing CDAI quartiles were: Q1 (reference), Q2=0.87 (0.78–0.97), Q3=0.91 (0.81–1.01), and Q4=0.86 (0.77–0.96). In NHANES, the trend was more pronounced: Q1 (reference), Q2=0.47 (0.24–0.94), Q3=0.39 (0.19–0.82), and Q4=0.46 (0.22–0.95). Additionally, higher dietary intake of carotene, one of the key antioxidant components, was independently associated with a lower risk of probable sarcopenia in both cohorts. Subgroup analyses indicated an inverse association between CDAI and probable sarcopenia risk in females across both cohorts, whereas no significant association was observed in males. Sensitivity analyses confirmed the robustness of these findings.

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Conclusions Increased dietary intake of antioxidant vitamins may reduce the risk of probable sarcopenia in older adults, emphasizing the need for targeted prevention strategies. Further research on underlying mechanisms and sex differences is warranted.

Keywords Antioxidant vitamins, Sarcopenia, Epidemiology

Background

Sarcopenia, characterized by the progressive loss of skeletal muscle mass, strength, and function [1], represents a significant public health concern, particularly in aging populations [2]. As the global population ages, the prevalence of sarcopenia continues to rise, leading to increased morbidity, disability, and healthcare burden [3–5]. The development of sarcopenia is likely multifactorial. Probable contributors include age-related changes in physiology, sedentary lifestyle, dietary factors, and acute illness [6]. Identifying modifiable risk factors for sarcopenia is crucial for developing effective preventive strategies and interventions to mitigate its impact on individual health and healthcare systems.

Antioxidant vitamins are crucial for scavenging reactive oxygen species and attenuating oxidative stress, which are implicated in age-related muscle decline and dysfunction [7–9]. The dietary consumption of antioxidant vitamins, including vitamins A, C, E, and carotene, has been extensively studied for its potential in preventing sarcopenia [10–12]. Findings from the Framingham cohort study, comprising individuals aged 33 to 80 ($n = 2452$), revealed no significant correlations between hand grip strength (HGS) and the intake of vitamins C, E, or carotene [10]. Conversely, the UK Biobank cohort study, conducted among individuals aged over 60 ($n = 68,002$), reported significant positive associations between HGS and dietary intake of vitamins A, C, E, and carotene in women. However, in men, significant positive associations were observed only for vitamin A and carotene, with no association found for vitamin C and vitamin E [11]. Similarly, results from the National Health and Nutrition Evaluation Surveys (NHANES) cohort study, involving individuals aged over 20 ($n = 6019$), showed a significant positive correlation between HGS and vitamin E intake, while no significant associations were found with vitamins A, C, or carotene [12]. Furthermore, comprehensive assessments of the composite dietary antioxidant index (CDAI) in relation to HGS suggested associations with both continuous and categorical CDAI variables [12]. Despite indications of some association between dietary antioxidant vitamins and HGS, the findings are inconsistent, and none of the studies examined the association between the total antioxidant capacity of antioxidant vitamins and the risk of sarcopenia. CDAI, representing the cumulative antioxidant capacity

of foods, offers a comprehensive approach to evaluating dietary antioxidant intake and its potential health implications [13]. Therefore, assessing the association between CDAI and risk of sarcopenia could inform future trials testing whether higher intakes of these antioxidants reduce the risk or slow the progression of sarcopenia.

The Sarcopenia Definition and Outcomes Consortium (SDOC) [14] found that lean mass was not associated with incident adverse health-related outcomes in community-dwelling older adults, regardless of adjustment for body size. The SDOC analyses identified HGS, either absolute or scaled to measures of body size, as a significant discriminator of slowness, and could predict falls, self-reported mobility limitation, hip fractures, and mortality in community-dwelling older adults. Additionally, given the inherent difficulty in measuring muscle mass, the European Working Group on Sarcopenia in Older People (EWGSOP) recommended low HGS as a diagnostic criterion for screening probable sarcopenia [4]. Therefore, the primary outcome variable in this study is probable sarcopenia diagnosed based on HGS.

The objective of this study is to explore the relationship between dietary CDAI and the likelihood of probable sarcopenia (diagnosed based on HGS) in individuals aged over 60, utilizing data from both the UK Biobank and NHANES cohorts. Through a comprehensive assessment of the collective impact of antioxidant vitamins on the risk of probable sarcopenia, we aim to enhance the existing knowledge regarding the role of dietary antioxidants in safeguarding muscle health and mitigating the onset of sarcopenia.

Methods

Study population

The UK Biobank (<http://www.ukbiobank.ac.uk>) is a large general population cohort study with a wide range of data collected, an ongoing multicenter study comprising approximately 500,000 individuals aged 38–73 years between 2006 and 2010. Ethical approval was obtained from the North-west Multicenter Research Ethics Committee, and all participants provided written informed consent. Clinical and biochemical information were collected using touchscreen questionnaires, physical examinations, sample collection, and electronic health records. Additional details about the UK Biobank project can be found in other published work

[15]. Although the UK Biobank is a prospective cohort, only cross-sectional data were used in the present study. Among the 68,128 participants with data on both muscle strength and antioxidant vitamins in the UK Biobank, 28,926 samples were aged 60 years or older. After excluding 6,005 participants with missing data for at least one variable, the final analysis cohort comprised 22,921 participants.

To validate the findings observed in the UK Biobank cohort, we conducted an analysis using data from the NHANES (<http://www.cdc.gov/nchs/nhanes.htm>). The NHANES data utilized in this study were collected during the 2011–2012 and 2013–2014 survey cycles. NHANES is conducted by the National Center for Health Statistics (NCHS) and approved by the ethics review boards of both the NCHS and the Centers for Disease Control (CDC). This cross-sectional survey involved in-home interviews and physical examinations. Participants provided written consent, and all interviews and examinations were conducted by trained technicians following standardized protocols outlined in the NHANES operation manuals. Among the 13,569 participants with data on both muscle strength and antioxidant vitamins in the NHANES, 2760 samples were aged 60 years or older. After excluding 119 participants with missing data for at least one variable, the final analysis cohort comprised 2641 participants.

The flowchart illustrating the participant selection process is presented in Fig. 1.

Primary exposure—CDAI

In the UK Biobank, dietary information was collected through a touch screen questionnaire administered at baseline [16], which recorded the habitual consumption frequency of major foods and food categories over the past year. Additionally, a web-based 24-h dietary assessment tool called the Oxford WebQ [17] was utilized to evaluate the average dietary intake of over 200,000 participants. The Oxford WebQ calculated nutrient intakes by multiplying the serving size of each food by the number of servings consumed and then by the quantity of each nutrient contained in those food servings, as referenced from the UK Nutrient Databank Food Composition Tables [18]. Multiple dietary assessments were conducted to capture seasonal variations in dietary intake and to provide estimates of habitual intake during baseline and follow-up visits from 2009 to 2012. Each dietary variable was averaged from five measurements taken during the follow-up period.

During the NHANES data survey, trained interviewers collected dietary data through two separate 24-h dietary recall interviews, assessing the average intake from both recalls. The first recall was conducted in person at a mobile examination center, while the second interview took place 3–10 days later via telephone consultation. An average of two 24-h dietary intake records was used to represent the usual dietary intake status of participants, as using a minimum of two non-consecutive days of dietary data provides a more accurate estimate [19]. Total energy and nutrient intakes were calculated based

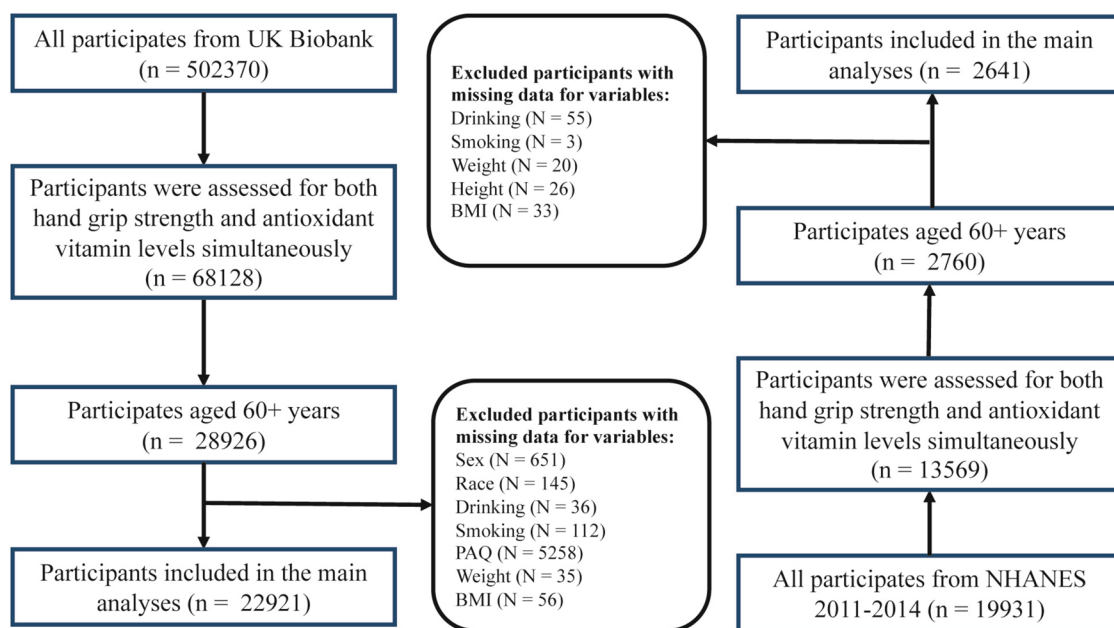


Fig. 1 Flowchart for the selection of participants

on the collected dietary information using the United States Department of Agriculture (USDA) Survey Nutrient Database [20, 21].

The CDAI was originally developed by Wright et al. [13], and is calculated as the sum of standardized intakes of individual antioxidant vitamins and other nutrients with antioxidant properties. In this present study, we modified the original CDAI to include only four key dietary antioxidants: vitamin A, vitamin C, vitamin E, and carotene, as expressed in Formula (1). In the formula, x_i represents the daily intake of antioxidants; u_i represents the mean of x_i across the entire antioxidant spectrum; and s_i denotes the standard deviation of u_i .

$$\text{CDAIAV} = \sum_{i=1}^4 \frac{x_i - u_i}{s_i} \quad (1)$$

In present study, vitamin A refers to retinol equivalents (in both UK Biobank and NHANES); carotene is defined as the sum of alpha-carotene and beta-carotene (in both UK Biobank and NHANES); and vitamin E refers to total vitamin E in the UK Biobank and alpha-tocopherol in NHANES. Intake estimates exclude contributions from dietary supplements, medications, or plain drinking water. The CDAI is analyzed both as a continuous variable and by dividing it into quartiles (Q1, Q2, Q3, Q4) for further comparison.

Primary outcome – probable sarcopenia

In the UK Biobank cohort, HGS was measured using a Jamar J00105 hydraulic hand dynamometer. Each participant had one measurement recorded for each hand. During the test, participants sat upright with their forearms at a 90-degree angle on armrests, gripping the dynamometer handle set to a specific position. They were instructed to grip the handle as forcefully as possible for about 3 s. The maximum grip strength was recorded in whole kilogram-force units. In the NHANES cohort, HGS was measured using a Takei TTK5401 dynamometer by trained examiners, with adjustments made for hand size. Each hand was tested three times, with a 60-s rest between measurements. Participants stood during the HGS measurement unless they had physical limitations. In present study, those unable to complete testing for both hands were excluded. The highest value from all measurements was recorded as the final HGS value in both databases.

Due to differences in the definition of sarcopenia, which remains a source of debate [14], we utilized different probable sarcopenia definitions. Two different criteria for probable sarcopenia were used: (1) the SDOC criteria (applied in both the UK Biobank and NHANES cohorts) classified probable sarcopenia in older adults

if any of the following conditions were met: hand grip strength (HGS) < 35.5 kg for men and < 20 kg for women; HGS divided by body mass index (BMI) < 1.05 for men and < 0.79 for women; or HGS divided by body weight < 0.45 for men and < 0.34 for women. (2) The EWGSOP definition classified probable sarcopenia with HGS < 27 kg for men and < 16 kg for women (in the UK Biobank), while the FNIH definition classified it with HGS < 26 kg for men and < 16 kg for women (in NHANES). Height and weight data were obtained during physical examinations, and BMI was calculated as weight divided by the square of height. Given that our study population is composed of older adults and that HGS is closely related to height and weight, we chose to fully adopt the SDOC criteria as the primary classification for probable sarcopenia in this present study.

Covariates

In both UK Biobank and NHANES cohorts, social demographic and lifestyle factors were collected via questionnaire. Sociodemographic factors included age, sex (male and female), and race (white, black, Asian, and other). Lifestyle factors included drinking status (yes and no), smoking status (yes and no), physical activity (yes and no), multivitamin supplementation (yes and no), total protein intake, and total energy intake obtained from questionnaires. In the UK Biobank cohort, smoking status through the question “Do you smoke tobacco now?”; drinking status collected through the question “Do you drink alcohol now?”; physical activity indicates whether a person met the 2017 UK Physical activity guidelines of 150 min of moderate activity per week or 75 min of vigorous activity [22]. In the NHANES cohorts, smoking status collected through the question “Do you now smoke cigarettes?”; drinking status collected through the question “Had at least 12 alcohol drinks/1 year?”; physical activity collected through the question “Does your sport’s/work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking or carrying light loads for at least 10 min continuously?” or “Do your sport’s/work involve vigorous-intensity activity that causes large increases in breathing or heart rate carrying or lifting heavy loads, digging or construction work for at least 10 min continuously?”. Additionally, in the UK Biobank cohort, potential risk factors for probable sarcopenia such as type 2 diabetes, multiple sclerosis, myasthenia gravis, Parkinson’s disease, osteoporosis, arthritis, and fracture were collected.

Statistical analysis

In both UK Biobank and NHANES cohorts, participants were stratified into two groups depending on the presence or absence of probable sarcopenia. Continuous

variables were presented as medians with interquartile ranges (IQR) for non-normally distributed data, and compared using Mann–Whitney U tests. Categorical variables were reported as counts and percentages, and comparisons were conducted using chi-square tests. Multivariable logistic regression models were employed to examine the relationships between CDAI (both continuous and category) and its components (vitamin A, vitamin C, vitamin E, and carotene) with probable sarcopenia risk (in both UK Biobank and NHANES cohorts). Models 1 and 2 were utilized, with model 1 adjusted for age, sex, and race, and model 2 further added control covariates such as smoking status, drinking status, physical activity, multivitamin supplement, total protein intake, and total energy intake. The primary findings were derived from model 2. Additionally, we observed an interaction between sex and CDAI, along with its components, in both models. Sex subgroup analyses were conducted after adjusting for covariates in the multivariable logistic regression models in both UK Biobank and NHANES cohorts. In the sensitivity analysis, we conducted an exclusion of individuals diagnosed with sarcopenia-related diseases (including type 2 diabetes, multiple sclerosis, myasthenia gravis, Parkinson's disease, osteoporosis, arthritis, and fracture), as well as participants who were taking multivitamin supplements. Subsequently, we reexamined the associations between CDAI and the risk of probable sarcopenia exclusively within the UK Biobank cohort. A significance threshold of two-sided $p < 0.05$ was applied, and all analyses were performed using R (4.3.3).

Results

Baseline characteristics

In the UK Biobank cohort, out of 22,921 participants, 52% were identified as probable sarcopenia patients based on the SDOC criteria (Table 1). Probable sarcopenia patients had a higher mean age compared to non-probable sarcopenia individuals (median 64.0 vs. median 63.0 years) and a higher proportion among females (52% vs. 48%). According to the EWGSOP criteria (eTable 1), 6.8% of participants were identified as probable sarcopenia patients. Similar patterns were observed regarding age and sex distribution. In the NHANES cohort, 51.9% of participants were classified as probable sarcopenia patients based on the SDOC criteria (Table 2), with those individuals having a significantly higher median age compared to non-sarcopenic individuals ($p < 0.001$). Notably, there was no sex difference in the prevalence of probable sarcopenia ($p = 0.881$). Similarly, using the FNIH criteria (eTable 2), 5.4% of participants were identified as probable sarcopenia patients, exhibiting a higher median age compared to non-sarcopenic individuals ($p < 0.001$), with

no significant difference in prevalence between males and females ($p = 0.444$).

Associations between CDAI and probable sarcopenia risk

The median (interquartile range) of CDAI was -0.39 ($-1.88, 1.45$) in the UK Biobank and -0.57 ($-1.60, 0.84$) in NHANES, respectively. Under the SDOC criteria (Table 3), a higher CDAI was significantly associated with a lower risk of probable sarcopenia in both the UK Biobank (Model 2: OR = 0.98, 95% CI: 0.97–0.998, $p = 0.027$) and NHANES (Model 2: OR = 0.87, 95% CI: 0.75–0.997, $p = 0.045$) cohorts. In quartile analyses, the risk of probable sarcopenia tended to decrease across higher CDAI quartiles, although the dose–response trend was not strictly linear. The multivariable-adjusted ORs (95% CIs) across increasing CDAI quartiles were 1 (reference), 0.87 (0.78–0.97), 0.91 (0.81–1.01), and 0.86 (0.77–0.96) in the UK Biobank cohort; and 1 (reference), 0.47 (0.24–0.94), 0.39 (0.19–0.82), and 0.46 (0.22–0.95) in the NHANES cohort. With regard to individual antioxidant vitamins, higher intakes of vitamin E and carotene were associated with a lower risk of probable sarcopenia in both datasets (eTable 3).

Under the EWGSOP/FNIH criteria (Table 3), a significant inverse association was observed only for the highest CDAI quartile (Q4) in the UK Biobank cohort. For individual vitamins, only higher carotene intake showed a consistent association with reduced probable sarcopenia risk in both cohorts (eTable 4).

All models were adjusted for potential confounders, including age, sex, race/ethnicity, smoking, alcohol consumption, physical activity, multivitamin use, total protein intake, and total energy intake. Taken together, these findings suggest that higher dietary intake of antioxidant vitamins, particularly vitamin E and carotene, may be beneficial in reducing the risk of probable sarcopenia.

Subgroup analysis

We conducted sex-stratified analyses to further explore the associations between CDAI and the risk of probable sarcopenia in both cohorts (Table 4). Notably, no significant interaction was observed between sex and CDAI (all P -interaction > 0.05 , Table 3). When stratified by sex, significant inverse associations between both continuous and categorical CDAI and probable sarcopenia were observed in females under the SDOC criteria in the UK Biobank cohort, but not under the EWGSOP criteria. No associations were detected in males, regardless of the criteria applied. Similar patterns were found in the NHANES cohort, where significant associations were observed in females but not in males. These findings suggest that the association may be more pronounced in females, although the absence of a significant sex and

Table 1 Characteristics of the participants between probable sarcopenia and non-probable sarcopenia (defined by SDOC) in UK Biobank

Variable	Overall, N= 22,921	Non-probable sarcopenia, N= 10,992	Probable sarcopenia, N= 11,929	p-value
Age, median (IQR), years	64.0 (62.0, 66.0)	63.0 (62.0, 66.0)	64.0 (62.0, 66.0)	< 0.001
Sex, n (%)				< 0.001
Female	11,467.0 (50.0)	5,258.0 (47.8)	6,209.0 (52.0)	
Male	11,454.0 (50.0)	5,734.0 (52.2)	5,720.0 (48.0)	
Race, n (%)				< 0.001
White	22,213.0 (96.9)	10,737.0 (97.7)	11,476.0 (96.2)	
Asian	354.0 (1.5)	106.0 (1.0)	248.0 (2.1)	
Black	174.0 (0.8)	70.0 (0.6)	104.0 (0.9)	
Mixed	180.0 (0.8)	79.0 (0.7)	101.0 (0.8)	
Smoking status, n (%)				0.214
no	21,531.0 (93.9)	10,303.0 (93.7)	11,228.0 (94.1)	
yes	1,390.0 (6.1)	689.0 (6.3)	701.0 (5.9)	
Alcohol status, n (%)				< 0.001
no	1,603.0 (7.0)	620.0 (5.6)	983.0 (8.2)	
yes	21,318.0 (93.0)	10,372.0 (94.4)	10,946.0 (91.8)	
Physical activity, n (%)				< 0.001
no	9,566.0 (41.7)	4,140.0 (37.7)	5,426.0 (45.5)	
yes	13,355.0 (58.3)	6,852.0 (62.3)	6,503.0 (54.5)	
Multivitamin supplement, n (%)				0.03
no	18,341.0 (80.0)	8,730.0 (79.4)	9,611.0 (80.6)	
yes	4,580.0 (20.0)	2,262.0 (20.6)	2,318.0 (19.4)	
Total protein intake, median (IQR), g	78.73 (62.70, 96.56)	78.88 (63.10, 96.37)	78.53 (62.34, 96.79)	0.364
Total energy intake, median (IQR), kJ	8,348.74 (6,754.21, 10,253.90)	8,398.07 (6,823.19, 10,284.03)	8,306.92 (6,681.88, 10,233.50)	0.002
Vitamin A, median (IQR), ug	282.52 (170.02, 433.96)	281.50 (167.68, 433.14)	283.95 (171.92, 434.88)	0.277
Q1	108.6 (70, 140.56)	109.41 (70.27, 140.4)	108.14 (69.72, 140.79)	0.617
Q2	224.89 (198.48, 253.15)	224.14 (197.9, 252.48)	225.87 (199.15, 253.9)	0.233
Q3	350.69 (313.47, 388.52)	351.19 (313.46, 389.92)	350.51 (313.5, 387.59)	0.725
Q4	562.57 (490.55, 679.85)	562.53 (491.25, 676.85)	562.81 (490.34, 682.92)	0.856
Vitamin C, median (IQR), mg	134.31 (74.93, 213.73)	137.87 (77.59, 217.36)	130.95 (72.60, 210.28)	< 0.001
Q1	45.08 (27.46, 60.63)	46.82 (28.03, 61.39)	43.65 (26.97, 59.72)	0.003
Q2	103.33 (88.67, 118.2)	104.01 (88.81, 118.46)	102.69 (88.43, 117.96)	0.186
Q3	169.37 (151.2, 189.3)	169.14 (150.49, 188.92)	169.47 (151.61, 189.88)	0.264
Q4	292.24 (247.38, 365.33)	292.68 (247.75, 365.32)	291.81 (246.94, 365.31)	0.804
Vitamin E, median (IQR), mg	8.15 (5.58, 11.50)	8.29 (5.69, 11.67)	8.03 (5.47, 11.33)	< 0.001
Q1	4.14 (3.15, 4.91)	4.2 (3.24, 4.93)	4.1 (3.06, 4.89)	0.002
Q2	6.85 (6.23, 7.47)	6.87 (6.25, 7.49)	6.83 (6.22, 7.46)	0.12
Q3	9.59 (8.86, 10.47)	9.6 (8.84, 10.47)	9.59 (8.88, 10.47)	0.649
Q4	14.62 (12.83, 17.58)	14.54 (12.84, 17.47)	14.72 (12.82, 17.73)	0.314
Carotene, median (IQR), ug	2,532.46 (845.30, 4,944.22)	2,611.26 (907.11, 4,996.69)	2,459.98 (795.06, 4,893.42)	< 0.001
Q1	402.29 (243.61, 594.59)	413.7 (249.12, 602.47)	391.87 (240.81, 588.27)	0.084
Q2	1498.29 (1157.48, 1998.87)	1502.61 (1162.77, 1999.22)	1494.26 (1147.81, 1997.27)	0.681
Q3	3772.86 (3026.95, 4374.52)	3777.09 (3030.24, 4393.7)	3759.91 (3024.13, 4351.69)	0.389
Q4	6916.29 (5812.63, 9021.36)	6881.49 (5793.36, 8897.08)	6969.88 (5837.93, 9144.94)	0.069
CDAI, median (IQR)	−0.39 (−1.88, 1.45)	−0.43 (−1.95, 1.39)	−0.33 (−1.81, 1.50)	< 0.001
Q1	−2.79 (−3.4, −2.29)	−2.78 (−3.4, −2.29)	−2.8 (−3.41, −2.3)	0.313
Q2	−1.12 (−1.49, −0.75)	−1.12 (−1.49, −0.75)	−1.12 (−1.5, −0.75)	0.596
Q3	0.42 (0.02, 0.9)	0.44 (0.02, 0.91)	0.41 (0.02, 0.89)	0.293
Q4	2.99 (2.11, 4.4)	2.99 (2.1, 4.41)	3 (2.11, 4.39)	0.649

Probable sarcopenia was diagnosed if any of the following criteria were met: HGS < 35.5 kg for men and < 20 kg for women; HGS divided by body mass index (BMI) < 1.05 for men and < 0.79 for women; HGS divided by body weight < 0.45 for men and < 0.34 for women. N, number; IQR: Interquartile Range (25%, 75%); CDAI, composite dietary antioxidant index; SDOC, Sarcopenia Definition and Outcomes Consortium; HGS: handgrip strength

Table 2 Characteristics of the participants between probable sarcopenia and non-probable sarcopenia (defined by SDOC) in NHANES

Variable	Overall, N = 2,641	Non-probable sarcopenia, N = 1,271	Probable sarcopenia, N = 1,370	p-value
Age, median (IQR), years	69.0 (63.0, 76.0)	66.0 (63.0, 72.0)	72.0 (65.0, 79.8)	< 0.001
Sex, n (%)				0.881
Female	1,338.0 (50.7)	642.0 (50.5)	696.0 (50.8)	
Male	1,303.0 (49.3)	629.0 (49.5)	674.0 (49.2)	
Race, n (%)				< 0.001
White	1,291.0 (48.9)	558.0 (43.9)	733.0 (53.5)	
Asian	194.0 (7.3)	118.0 (9.3)	76.0 (5.5)	
Black	628.0 (23.8)	348.0 (27.4)	280.0 (20.4)	
Mixed	528.0 (20.0)	247.0 (19.4)	281.0 (20.5)	
Smoking status, n (%)				0.007
no	1,300.0 (49.2)	591.0 (46.5)	709.0 (51.8)	
yes	1,341.0 (50.8)	680.0 (53.5)	661.0 (48.2)	
Alcohol status, n (%)				< 0.001
no	851.0 (32.2)	356.0 (28.0)	495.0 (36.1)	
yes	1,790.0 (67.8)	915.0 (72.0)	875.0 (63.9)	
Physical activity, n (%)				< 0.001
no	1,821.0 (69.0)	815.0 (64.1)	1,006.0 (73.4)	
yes	820.0 (31.0)	456.0 (35.9)	364.0 (26.6)	
Multivitamin supplement, n (%)				0.994
no	2,589.0 (98.0)	1,246.0 (98.0)	1,343.0 (98.0)	
yes	52.0 (2.0)	25.0 (2.0)	27.0 (2.0)	
Total protein intake, median (IQR), g	65.99 (48.13, 89.47)	67.93 (49.60, 91.17)	64.00 (46.72, 87.82)	0.011
Total energy intake, median (IQR), kJ	7,213.39 (5,347.28, 9,489.54)	7,351.46 (5,476.99, 9,556.49)	7,035.56 (5,200.84, 9,258.37)	0.01
Vitamin A, median (IQR), ug	494.00 (275.00, 803.00)	487.00 (276.00, 784.50)	500.50 (275.00, 807.00)	0.63
Q1	160 (94, 221.75)	109.41 (70.27, 140.4)	108.14 (69.72, 140.79)	0.172
Q2	382 (327, 437)	224.14 (197.9, 252.48)	225.87 (199.15, 253.9)	0.632
Q3	619.5 (549, 705)	351.19 (313.46, 389.92)	350.5 (313.5, 387.59)	0.343
Q4	1111 (928.25, 1450.75)	562.53 (491.25, 676.85)	562.81 (490.34, 682.92)	0.537
Vitamin C, median (IQR), mg	57.40 (23.30, 115.70)	57.00 (23.60, 118.35)	57.45 (22.83, 113.18)	0.247
Q1	11.45 (5.08, 17.63)	46.82 (28.03, 61.39)	43.65 (26.97, 59.72)	0.044
Q2	36.5 (30.9, 45.5)	104.01 (88.81, 118.46)	102.69 (88.43, 117.96)	0.199
Q3	81.75 (68.1, 96.8)	169.14 (150.49, 188.92)	169.47 (151.61, 189.88)	0.021
Q4	169.15 (138.65, 222.08)	292.68 (247.75, 365.32)	291.81 (246.94, 365.31)	0.047
Vitamin E, median (IQR), mg	6.62 (4.19, 10.06)	6.88 (4.47, 10.45)	6.33 (3.90, 9.71)	< 0.001
Q1	2.85 (2.09, 3.59)	4.2 (3.24, 4.93)	4.1 (3.06, 4.89)	0.203
Q2	5.36 (4.75, 6.03)	6.87 (6.25, 7.49)	6.83 (6.22, 7.46)	0.652
Q3	8.1 (7.36, 9.01)	9.6 (8.84, 10.47)	9.59 (8.88, 10.47)	0.376
Q4	13.47 (11.34, 17.38)	14.54 (12.84, 17.47)	14.72 (12.82, 17.73)	0.751
Carotene, median (IQR), ug	976.00 (324.00, 3,045.00)	1,068.00 (360.00, 3,469.00)	900.50 (306.00, 2,753.00)	0.006
Q1	146 (75, 232)	413.7 (249.12, 602.47)	391.87 (240.81, 588.27)	0.974
Q2	575 (439.25, 750.75)	1502.61 (1162.77, 1999.22)	1494.26 (1147.81, 1997.27)	0.468
Q3	1718.5 (1279.5, 2310.5)	3777.09 (3030.24, 4393.7)	3759.91 (3024.13, 4351.69)	0.55
Q4	6255 (4423, 10,448)	6881.49 (5793.36, 8897.08)	6969.88 (5837.93, 9144.94)	0.884
CDAI, median (IQR)	-0.57 (-1.60, 0.84)	-0.51 (-1.53, 1.05)	-0.64 (-1.63, 0.70)	0.028
Q1	-2.17 (-2.48, -1.89)	-2.78 (-3.4, -2.29)	-2.8 (-3.41, -2.3)	0.678
Q2	-1.08 (-1.33, -0.84)	-1.12 (-1.49, -0.75)	-1.12 (-1.5, -0.75)	0.769
Q3	0.02 (-0.3, 0.4)	0.44 (0.02, 0.91)	0.41 (0.02, 0.89)	0.432
Q4	2.14 (1.43, 3.65)	2.99 (2.1, 4.41)	3 (2.11, 4.39)	0.773

Probable sarcopenia was diagnosed if any of the following criteria were met: HGS < 35.5 kg for men and < 20 kg for women; HGS divided by body mass index (BMI) < 1.05 for men and < 0.79 for women; HGS divided by body weight < 0.45 for men and < 0.34 for women. N, number; IQR: Interquartile Range (25%, 75%); CDAI, composite dietary antioxidant index; SDOC, Sarcopenia Definition and Outcomes Consortium; NHANES, National Health and Nutrition Evaluation Surveys; HGS: handgrip strength

Table 3 Associations between CDAI and probable sarcopenia risk in both UK Biobank and NHANES cohorts

UK Biobank				NHANES			
Model 1		Model 2		Model 1		Model 2	
OR (95% CI)	p	p for interaction	OR (95% CI)	p	p for interaction	OR (95% CI)	p for interaction
Probable sarcopenia was diagnosed by SDOC							
CDAI	0.99 (0.97, 1)	0.043	0.667				
Q1	1 (reference)		0.98 (0.97, 1)	0.027	0.637	0.87 (0.76, 0.99)	0.034
Q2	0.87 (0.78, 0.96)	0.008	0.87 (0.78, 0.97)	0.011	0.34	1 (reference)	0.605
Q3	0.91 (0.82, 1.01)	0.072	0.91 (0.81, 1.01)	0.078	0.583	0.47 (0.24, 0.92)	0.026
Q4	0.87 (0.79, 0.97)	0.011	0.86 (0.77, 0.96)	0.01	0.852	0.42 (0.21, 0.84)	0.014
Probable sarcopenia was diagnosed by EWGSOP/FNIH							
CDAI	0.98 (0.96, 1.01)	0.238	0.966			0.47 (0.24, 0.91)	0.025
Q1	1 (reference)		0.99 (0.96, 1.02)	0.515	0.902	0.97 (0.93, 1.01)	0.146
Q2	0.86 (0.7, 1.04)	0.122	0.87 (0.71, 1.07)	0.18	0.777	1 (reference)	0.25
Q3	1.01 (0.83, 1.21)	0.958	1.03 (0.85, 1.25)	0.771	0.241	0.94 (0.67, 1.31)	0.704
Q4	0.8 (0.66, 0.98)	0.028	0.81 (0.65, 1.01)	0.063	0.883	0.89 (0.64, 1.24)	0.5

Odds ratios (ORs) and 95% confidence intervals (CIs) for probable sarcopenia based on the CDAI are shown. Model 1 was adjusted for age, gender, and race. Model 2 includes further adjustments for smoking status, alcohol consumption, physical activity, multivitamin use, total protein intake, and total energy intake. The 'p' is derived from logistic regression, and 'p' for interaction tests the interaction between sex and CDAI. Abbreviations: CDAI, Composite Dietary Antioxidant Index; NHANES, National Health and Nutrition Examination Survey; SDOC, Sarcopenia Definition and Outcomes Consortium; EWGSOP, European Working Group on Sarcopenia in Older People; FNIH, Foundation for the National Institutes of Health

Table 4 Sex subgroup analysis of relationship between CDAI and probable sarcopenia risk

	UK Biobank				NHANES			
	Female		Male		Female		Male	
	OR (95% CI)	<i>p</i>	OR (95% CI)	<i>p</i>	OR (95% CI)	<i>p</i>	OR (95% CI)	<i>p</i>
Probable sarcopenia was diagnosed by SDOC								
CDAI (continues)	0.97 (0.96, 0.99)	0.001	0.99 (0.97, 1.01)	0.168	0.94 (0.89, 0.99)	0.025	0.91 (0.79, 1.05)	0.2
Q1	1 (reference)		1 (reference)		1 (reference)		1 (reference)	
Q2	0.84 (0.76, 0.94)	0.002	0.97 (0.87, 1.07)	0.523	1.06 (0.76, 1.46)	0.743	0.52 (0.26, 1.04)	0.063
Q3	0.88 (0.79, 0.98)	0.023	0.9 (0.81, 1.01)	0.072	0.9 (0.64, 1.27)	0.557	0.53 (0.25, 1.13)	0.101
Q4	0.81 (0.72, 0.92)	0.001	0.9 (0.8, 1.02)	0.101	0.68 (0.48, 0.98)	0.037	0.74 (0.35, 1.56)	0.431
Probable sarcopenia was diagnosed by EWGSOP/FNIH								
CDAI (continues)	1 (0.97, 1.03)	0.925	0.98 (0.95, 1.02)	0.384	1.03 (0.91, 1.16)	0.635	0.97 (0.93, 1.02)	0.213
Q1	1 (reference)		1 (reference)		1 (reference)		1 (reference)	
Q2	0.88 (0.72, 1.08)	0.211	0.84 (0.68, 1.05)	0.129	1.04 (0.5, 2.19)	0.916	0.82 (0.59, 1.15)	0.254
Q3	1.06 (0.87, 1.3)	0.571	0.85 (0.67, 1.07)	0.156	0.79 (0.36, 1.75)	0.563	0.89 (0.63, 1.26)	0.514
Q4	0.88 (0.69, 1.11)	0.265	0.79 (0.61, 1.02)	0.073	0.84 (0.37, 1.92)	0.677	0.76 (0.52, 1.1)	0.146

Sex subgroup analysis was adjusted for age, race, smoking status, alcohol consumption, physical activity, multivitamin use, total protein intake, and total energy intake. Abbreviations: OR: Odds ratio, CI: confidence intervals; CDAI, Composite Dietary Antioxidant Index; NHANES, National Health and Nutrition Examination Survey; SDOC, Sarcopenia Definition and Outcomes Consortium; EWGSOP, European Working Group on Sarcopenia in Older People; FNIH, Foundation for the National Institutes of Health

CDAI interaction limits definitive conclusions regarding sex-specific differences.

Sensitivity analysis

Sensitivity analysis conducted in the UK Biobank cohort by excluding participants with diseases possibly related to sarcopenia (*n* = 1916 excluded) and participants with multivitamin supplement (*n* = 6047 excluded) yielded consistent results with the primary analysis (eTable 5).

Discussion

Based on data from the UK Biobank and the NHANES cohorts of individuals aged 60 and above, our study revealed that increased intake of antioxidant vitamins in the diet was linked to a lower risk of probable sarcopenia, even after accounting for pertinent covariates. This association was particularly pronounced in females. Additionally, sensitivity analyses demonstrated the robustness of our primary findings.

According to the SDOC criteria, our study identified an inverse relationship between the CDAI (based on Vitamin A, C, E, and carotene) and the risk of probable sarcopenia in older populations from both the UK and the US. Although prior research had not examined the association between CDAI and sarcopenia risk, a recent study in the NHANES cohort (aged 20 and above) reported that a comprehensive assessment of dietary antioxidants and grip strength showed associations with both continuous and categorical CDAI (based on Vitamin A, C, E, carotene, selenium, and zinc) [12]. That study suggested

that increased intake of dietary antioxidant vitamins was associated with enhanced muscle strength, indirectly supporting our conclusions.

In a subsequent sex-stratified analysis, this previous study found a significant association between CDAI and grip strength in males but not in females [12]. In contrast, our research observed that the association between CDAI and probable sarcopenia risk varies by sex, showing an inverse relationship in females but no significant relationship in males across both the UK Biobank and NHANES cohorts, after adjusting for relevant covariates. These discrepancies could be attributed to differences in exposure and outcome variables between the two studies. The previous study [12] examined the association between an antioxidant index of six dietary antioxidants (Vitamin A, C, E, carotene, selenium, and zinc) and grip strength, whereas our study focused on the association between an antioxidant index of four dietary antioxidant vitamins (Vitamin A, C, E, and carotene) and the risk of probable sarcopenia. The selection of different exposure and outcome variables may be a key reason for the varying results. Additionally, the age differences between the study populations may have contributed to the divergent findings. The previous study [12] primarily targeted individuals aged 20 and older, while our study focused on older adults aged 60 and above. In this present study, although sex-stratified analyses revealed that the inverse association between CDAI and probable sarcopenia was significant in females but not in males, we did not detect any statistically significant interaction between sex and

CDAI. This suggests that the observed differences may not represent a true sex-specific effect but rather reflect variations in effect sizes or other unmeasured factors. Additionally, the number of probable sarcopenia cases was comparable between males and females, indicating that the lack of association in males is unlikely due to insufficient statistical power. Further studies are warranted to clarify whether the role of dietary antioxidants in sarcopenia risk differs by sex.

In our present study, under the SDOC criteria, dietary intake of vitamins E and carotene were associated with a reduced risk of probable sarcopenia in both UK and US population aged over 60. Under the EWGSOP/FNIH criteria, dietary intake of carotene was associated with a reduced risk of probable sarcopenia in both the UK and US populations aged over 60. Overall, our present study highlighted the association of dietary carotene supplements with the risk of sarcopenia in older population, as previously described [23, 24]. Previous studies have presented conflicting evidence regarding the association between antioxidant vitamins, including vitamins A, C, E, and carotene, and sarcopenia. A cross-sectional study involving 1172 individuals aged 50 to 85 from NHANES 2001–2002 suggested a positive association between serum α -carotene and muscle strength in older adults [23]. Similarly, a study of 801 subjects aged 70–84 in the Korean older population found that higher intakes of carotene were linked to a lower prevalence of sarcopenia [24]. However, findings from the Framingham study, which included individuals aged 33 to 80, did not show significant associations between grip strength and α -carotene or β -carotene [10]. Furthermore, in the NHANES cohort study involving individuals aged over 20, grip strength was significantly positively correlated with vitamin E intake, while no significant associations were found with vitamins A, C, or carotenoids [12]. These discrepancies in results may stem from differences in sample ages, definitions of outcomes and exposures, as well as variations in adjusted covariates. Nevertheless, the consistent findings of the two studies conducted in older populations [23, 24] indicate a positive association between carotene and muscle strength, as well as a lower prevalence of sarcopenia, which align with the results of our study.

Antioxidant vitamin plays a crucial role in mitigating oxidative stress, which is a key factor in the development and progression of sarcopenia [9]. Oxidative stress, characterized by the overproduction of ROS, leads to damage to macromolecules (such as: lipids, nucleic acids, and proteins), ultimately impairing muscle health and function [7, 25, 26]. Mitochondria, particularly in skeletal muscle cells, are the main source of ROS, and their accumulation can result in oxidative DNA damage,

contributing to muscle cell disorders and decreased muscle strength [9, 27, 28]. Studies have demonstrated that antioxidants (such as carotene) can effectively regulate oxidative stress levels, thereby improving muscle strength and overall skeletal muscle health [29]. Older adults with lower serum/plasma carotenoid levels have been found to face an increased risk of experiencing elevated IL-6 levels over time [30, 31]. Elevated IL-6 and C-reactive protein concentrations have been associated with a higher risk of muscle strength loss. Additionally, serum protein carbonyls, indicative of oxidative protein damage from oxidative stress, may independently forecast reduced grip strength, slower walking speed, and progression to severe walking disability in older females [32, 33]. Therefore, dietary intake of carotene and other antioxidants represents a potential strategy to mitigate oxidative stress and preserve muscle function, ultimately enhancing the quality of life in aging individuals [34].

This study enrolled a significant number of male and female participants from two community-based cohorts. We utilized CDAI as a measure of the collective potential of antioxidant vitamins derived from the diet. Furthermore, we extensively investigated its association with the risk of probable sarcopenia under different criteria. However, several limitations need to be acknowledged in this study. Firstly, the evaluation metrics employed primarily relied on subjective questionnaires rather than objective biochemical measurements from blood samples. Secondly, although efforts were made to control for social confounders that could impact the relationship between CDAI and probable sarcopenia risk, not all potential influencing variables could be included due to limitations in data sources and existing theoretical frameworks. Such as, in the sensitivity analysis excluding individuals who took multivitamin supplements, we were unable to entirely exclude the potential influence of carotene supplements due to lack of data on carotene supplement users. This could have impacted the study results. Thirdly, another limitation of this study lies in the differences in the assessment of lifestyle and sociodemographic variables between the UK Biobank and NHANES cohorts. Although similar categories of confounding factors were included in both datasets — such as smoking, alcohol consumption, and physical activity — the specific questions and definitions used to collect these variables differed. For instance, alcohol intake in NHANES was based on whether participants had consumed at least 12 alcoholic drinks in any one year, which may reflect historical rather than current use, whereas UK Biobank assessed current alcohol consumption. Likewise, physical activity was evaluated based on national guidelines in the UK Biobank but using self-reported occupational or sport-related activity questions in NHANES. These differences

may introduce heterogeneity in the interpretation of lifestyle effects and limit the direct comparability of associations across cohorts. Finally, although sex-stratified analyses revealed a significant inverse association between CDAI and probable sarcopenia in females but not in males, no significant interaction between sex and CDAI was observed. This implies that the observed differences may not reflect true sex-specific effects. The biological plausibility and potential mechanisms underlying this pattern remain to be elucidated and should be further examined in future studies.

Conclusions

In conclusion, our study observed that increased dietary intake of antioxidant vitamins was linked to a lower risk of probable sarcopenia, particularly in females. Further research exploring the underlying mechanisms and sex differences in the association between antioxidant vitamins intake and probable sarcopenia risk is warranted to better inform preventive strategies for sarcopenia.

Abbreviations

BMI	Body mass index
CDAI	Composite dietary antioxidant index
CDC	Centers for Disease Control
EWGSOP	European Working Group on Sarcopenia in Older People
FNIH	Foundation for the National Institutes of Health
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Evaluation Surveys
HGS	Hand grip strength
IQR	Interquartile Range
OR	Odds ratio
CI	Confidence intervals
SDOC	Sarcopenia Definition and Outcomes Consortium

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12986-025-00945-w>.

Supplementary Material 1.

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Authors' contributions

H.M.L. and Y.D.X.: analysis and interpretation of data, and drafted the work. Q.S.L., L.F.Y., X.Y., K.X.W., and Z.G.: substantively revised the manuscript. Q.Z. and J.Y.J. substantial contributions to the conception, design of the work, the acquisition of data. All authors read and approved the final version.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Ethical approval of UK Biobank was obtained from the North-west Multicenter Research Ethics Committee. Ethical approval of NHANES was obtained from the National Center for Health Statistics and the Centers for Disease Control. Written informed consent has been obtained from the patient(s) to publish this paper.

Consent to publication

Not applicable.

Competing interests

The authors declare no competing interests.

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