RESEARCH ARTICLE

Relationship between dietary protein intake and prevalence of sarcopenia in older adults

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Abstract

Objective: To assess the relationship between sarcopenia and dietary protein use among adults.

Method: The retrospective, cross-sectional study was conducted after approval from the ethics review committee of Sichuan Province Orthopaedic Hospital, China, and comprised prospective data of American adults aged at least 20 years who had participated in the National Health and Nutrition Examination Survey between 2015 and 2018. The survey had complete information on protein intake and sarcopenia status. It was analysed from June to September 2023, and was made public in September 2023. Baseline characteristics were examined, and confounding factors were determined. Data was analysed using R software version 4.2.2.

Results: Of the 5,222 participants with mean age 39.9±11.5 years, 2,708(51.9%) were females and 2.514(48.1%) were males. Sarcopenia was diagnosed in 524(10%) subjects. Those with dietary protein intake ≤56.7g/day had higher odds of sarcopenia compared to the rest (p<0.05).

Conclusion: Consuming enough protein in the diet may help shield adults from developing sarcopenia.

Keywords: Dietary proteins, Skeletal, Elderly, Cross-sectional studies, Correlation. (JPMA 76: 58; 2026)

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Introduction

As skeletal muscle mass, strength and function gradually decline, a common and crippling disease, known as sarcopenia, develops, especially in older adults.[1,2] It is linked to higher chances of fractures, falls, physical impairment, and death.[3,4] Given the aging population worldwide, understanding the modifiable factors that influence sarcopenia is of utmost importance for public health.

Protein consumption, among other dietary parameters, has been proposed as a meaningful influence in preventing and managing sarcopenia.[5] Protein is crucial for the synthesis and maintenance of muscle protein and insufficient protein consumption has been linked to muscle loss and a reduction in function.[6] Multiple research has examined the correlation between protein consumption and sarcopenia, although the results have been inconclusive. Several studies have shown strong links between increased protein consumption and a decreased likelihood of developing sarcopenia. A comprehensive analysis[7] revealed that increased consumption of protein was linked to a reduced likelihood of sarcopenia in elderly individuals. A three-year follow-up research[8] showed that a greater consumption of protein was linked to a decreased incidence of sarcopenia. However, other studies have failed to observe consistent associations between protein intake and sarcopenia. Xu et al.[9] discovered an adverse relationship between female muscle mass and dietary protein consumption. Similarly, there was no discernible correlation between the occurrence of sarcopenia and protein consumption in a long-term research by Mao et al.[10]

The current study was planned to examine the relationship between the incidence of sarcopenia and daily protein intake among adult individuals.

Materials and Methods

The retrospective, cross-sectional study was conducted after approval from the ethics review committee of Sichuan Province Orthopaedic Hospital, China, and comprised prospective data from American adults aged at least 20 years who participated in the National Health and Nutrition Examination Survey (NHANES) between 2015 and 2018. The data in the original NHANES, undertaken by the National Center for Health Statistics of the Centres for Disease Control and Prevention (CDC),[11] had been collected prospectively through standardised surveys and examinations. Pregnant women and individuals with incomplete dietary information had been excluded.

The current study extracted data electronically from the

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NHANES database. No manual medical record extraction was performed as the entire data had already been collected and anonymised in the NHANES dataset. The survey was representative of the whole country, and had gathered detailed dietary and health data from a wide range of American citizens. Each participant in NHANES was assigned a unique respondent sequence number, which did not disclose any individual participant information. The dataset included demographic, dietary and health-related information, making it suitable for investigating the research question of interest.[12] The dietary data had been collected at baseline during in-person interviews using 24-hour recall methodology. On two separate days, the participants were asked to recall everything they had eaten and drunk over the preceding 24 hours. Trained interviewers had used standardised protocols to collect detailed information on food items and portion sizes. The calculation of protein consumption was derived from the United States Department of Agriculture (USDA) Food and Nutrient Database for Dietary Studies.[13] The current study calculated the mean protein consumption over the two days. If there was a lack of data for the second day, data from the first day was used to complete the missing information.

Dual-Energy X-ray Absorptiometry (DEXA) had been utilised to quantify body composition, including muscle mass and body fat, using a scanner (QDR-4500 Hologic, Bedford, Massachusetts, USA). Only those who met the eligibility requirements, including age 8-60 years, weight <136.4kg and height <192.5cm, were eligible to take the examination. Before the examination, all metallic items — aside from dentures and hearing aids — had been taken out. Appendicular lean mass or appendiceal lean mass (ALM) was measured using dual DEXA and was calculated as the total fat-free lean body mass of the limbs (arms and legs).[14] To diagnose sarcopenia, ALM was divided by body mass index (BMI), following the guidelines set out by the Foundation for the National Institutes of Health (FNIH), with a cut-off value of <0.512kg/m^2 for women and <0.789kg/m^2 for men.[15] For participants with unilateral limb loss, the current study data from the intact limb to fill in the missing values. Data of those who had a bilateral lower limb or bilateral upper limb loss was excluded.

The covariates that were analysed were subjected to acyclic graphs. The variables were adjusted for age, gender, race, physical activity, diabetes, alcohol use, smoking, BMI, and degree of education. The following categories were included in the race and ethnicity classification: Mexican American, non-Hispanic Black, non-Hispanic White, and other races. There were three types of marital status: cohabiting, married and single. Three categories were used to categorise schooling levels: post-high school, high school, and below high school. The poverty income ratio (PIR) split household income into three categories: high (PIR >3.5), low (PIR <1.3) and medium (PIR >1.3-3.5).[16] There were four categories of physical activity: moderate (requiring at least 10 minutes of walking or cycling for transportation in the preceding week), vigorous (requiring at least 10 minutes of activity that led to meaningful sweating or an increase in breathing or heart rate in the preceding week), sedentary, and mild (requiring at least 10 minutes of walking or cycling for transportation in the preceding week). The smoking status had three categories; those who had never smoked (less than 100 cigarettes), those who were current smokers, and former smokers who had smoked over 100 cigarettes but had quit. Alcohol intake was defined as the act of regularly drinking at least one alcoholic beverage every month. Pre-existing diseases, such as hypertension (HTN) and diabetes mellitus (DM), were identified by asking participants whether they had been advised by a healthcare practitioner about these problems in the past. The BMI was determined using standard methodologies relying on the measurements of height and weight.

In the current study, data was analysed using the R statistical software tool version 4.2.2 (http://www.R-project.org, R Foundation, Shanghai, China).[17] The mouse package was used to execute multiple imputations for missing covariate data, which entailed producing several sets of imputed data. From these imputed datasets, one set was randomly selected for subsequent analysis. This approach helped address the issue of missing data, and led to more reliable and robust results. During the process of data analysis, the daily consumption of protein was split into four groups based on quartiles. While continuous variables were reported either as mean ± standard deviation (SD) or as median with interquartile range (IQR), depending on data distribution. Categorical variables were presented as frequencies and percentages. One-way analysis of variance (ANOVA) was used to evaluate disparities across groups for variables that adhered to a normal distribution. Kruskal-Wallis test was used for variables exhibiting skewness, while chi-square test was employed for categorical data. Logistic regression models were used to determine the relationship between protein intake and the incidence of sarcopenia. Calculations were conducted to ascertain the odds ratios (ORs) and their corresponding 95% confidence intervals (CIs). Sociodemographic factors, such as age, race/ethnicity, gender and marital status, were included in Model 1. Model 2 was also modified for variables chosen based on a directed acyclic graph, which included education, physical activity and DM. Model 3 included extensive adjustments, including sociodemographic factors, educational background, physical activity levels, DM status, alcohol intake, smoking habits, and BMI, Stratified analyses were conducted by including interaction terms between protein intake quartiles and each stratification variable (age group, gender, BMI category, physical activity level, and DM status) in the fully adjusted Model 3. The statistical significance of interaction terms was assessed using likelihood ratio tests, comparing models with and without the interaction terms. Additionally, the linear relationship between protein consumption and sarcopenia was evaluated using restricted cubic spline (RCS) regression to analyse the dose-response curve in the models. There were four knots that fell into the protein consumption; 5th, 65th, 35th and 95th percentiles. In addition, several subgroups were analysed to evaluate the correlation between protein intake and sarcopenia. These subgroups included different age ranges (40-60 years and 20-40 years), gender, BMI (<25kg/m^2 versus 25kg/m^2 or more), levels of physical activity (sedentary and mild vs. moderate and vigorous), and presence of DM (yes or no). The study used likelihood ratio tests to assess the relationship between dietary protein intake and subgroups. Two-tailed $p<0.05$was considered statistically significant.

Results

Of the 19,225 NHANES participants, 5,222(%) met the inclusion criteria (Figure 1). Of them, 2,708(51.9%) were females and 2,514(48.1%) were males. The overall mean age was 39.9±11.5 years. Sarcopenia was diagnosed in 524(10%) subjects. The differences were significant on multiple counts with respect to dietary protein intake (Table 1).

Age, gender, race, marital status, family income, level of schooling, sports participation, the use of alcohol, HTN, DM, BMI, serum albumin, creatinine, C-reactive protein (CRP), and total calorie intake were all linked to sarcopenia (Table 2).

Subsequently, the causal relationships was explored among covariates, excluding demographic variables, using a directed acyclic graph for which education was considered an ancestral variable, and serum albumin, creatinine and total energy intake were potential intermediate variables. After selection, the variables

Table-1: Baseline characteristics by categories of dietary protein (g/d) intake.

Variables	**Total** n (%)	**Q1** (g/d) ≤56.7 n (%)	**Q2** (g/d) 56.8-76.5 n (%)	**Q3** (g/d) 76.6-100.8 n (%)	**Q4** (g/d) ≥100.8 n (%)	***p*-value**
n	5222	1306	1305	1305	1306	
Mean Age (years)	39.9±11.5	40.2±11.9	40.4±11.5	39.6±11.3	39.3±11.2	0.047
Gender						<0.001
Male	2514 (48.1)	352 (27)	507 (38.9)	680 (52.1)	975 (74.7)	
Female	2708 (51.9)	954 (73)	798 (61.1)	625 (47.9)	331 (25.3)	
Race						<0.001
Non-Hispanic White	1598 (30.6)	380 (29.1)	409 (31.3)	409 (31.3)	400 (30.6)	
Non-Hispanic Black	1147 (22.0)	348 (26.6)	293 (22.5)	276 (21.1)	230 (17.6)	
Mexican American	875 (16.8)	167 (12.8)	219 (16.8)	224 (17.2)	265 (20.3)	
Other Race	1602 (30.7)	411 (31.5)	384 (29.4)	396 (30.3)	411 (31.5)	
Marital Status						<0.001
Married or Living with partner	3193 (61.1)	720 (55.1)	803 (61.5)	832 (63.8)	838 (64.2)	
Divorced or Live alone	2029 (38.9)	586 (44.9)	502 (38.5)	473 (36.2)	468 (35.8)	
PIR						<0.001
<1.3	1621 (31.0)	491 (37.6)	404 (31)	355 (27.2)	371 (28.4)	
1.3 - 3.49	2071 (39.7)	504 (38.6)	506 (38.8)	528 (40.5)	533 (40.8)	
>3.5	1530 (29.3)	311 (23.8)	395 (30.3)	422 (32.3)	402 (30.8)	
Education						<0.001
Less Than High School	946 (18.1)	263 (20.1)	226 (17.3)	217 (16.6)	240 (18.4)	
High School	1169 (22.4)	325 (24.9)	287 (22)	256 (19.6)	301 (23)	
More than High School	3107 (59.5)	718 (55)	792 (60.7)	832 (63.8)	765 (58.6)	
Smoke						<0.001
Never	3220 (61.7)	815 (62.4)	831 (63.7)	807 (61.8)	767 (58.7)	
Former	889 (17.0)	171 (13.1)	220 (16.9)	227 (17.4)	271 (20.8)	
Current	1113 (21.3)	320 (24.5)	254 (19.5)	271 (20.8)	268 (20.5)	
Alcohol						<0.001
No	990 (19.0)	307 (23.5)	284 (21.8)	215 (16.5)	184 (14.1)	
Yes	4232 (81.0)	999 (76.5)	1021 (78.2)	1090 (83.5)	1122 (85.9)	
Activity						<0.001
Sedentary	1986 (38.0)	547 (41.9)	523 (40.1)	486 (37.2)	430 (32.9)	
Mild	658 (12.6)	185 (14.2)	175 (13.4)	156 (12)	142 (10.9)	
Moderate	1164 (22.3)	274 (21)	303 (23.2)	302 (23.1)	285 (21.8)	
Vigorous	1414 (27.1)	300 (23)	304 (23.3)	361 (27.7)	449 (34.4)	
Hypertension						0.135
No	3934 (75.3)	956 (73.2)	978 (74.9)	1000 (76.6)	1000 (76.6)	
Yes	1288 (24.7)	350 (26.8)	327 (25.1)	305 (23.4)	306 (23.4)	
Diabetes						0.071
No	4698 (90.0)	1159 (88.7)	1163 (89.1)	1181 (90.5)	1195 (91.5)	
Yes	524 (10.0)	147 (11.3)	142 (10.9)	124 (9.5)	111 (8.5)	
Mean BMI (kg/m^2)	29.5±7.1	29.7±7.4	29.5±7.1	29.6±7.2	29.2±6.8	0.202
Mean TP (g/L)	72.1±4.3	72.2±4.5	71.7±4.3	72.2±4.2	72.3±4.2	0.001
Mean ALB (g/L)	42.4±3.6	41.7±3.6	42.1±3.6	42.7±3.5	43.2±3.5	<0.001
Mean BUN (mmol/L)	4.8±1.6	4.3±1.6	4.6±1.4	4.9±1.4	5.3±1.8	<0.001
Mean Cr (umol/L)	74.4±34.7	70.4±23.8	72.3±35.8	74.2±28.4	80.8±45.9	<0.001
CRP (mg/L),Median (IQR)	1.8 (0.8, 4.4)	2.4 (0.9, 5.3)	2.0 (0.8, 4.6)	1.8 (0.7, 4.1)	1.4 (0.6, 3.3)	<0.001
Energy (kcal/d) Median (IQR)	1969.0 (1506.1, 2551.8)	1297.0 (1007.1, 1612.2)	1803.5 (1524.0, 2093.0)	2173.0 (1827.0, 2533.5)	2892.5 (2421.1, 3452.8)	<0.001
Sarcopenia						<0.001
No	4698 (90.0)	1142 (87.4)	1177 (90.2)	1172 (89.8)	1207 (92.4)	
Yes	524 (10.0)	164 (12.6)	128 (9.8)	133 (10.2)	99 (7.6)	

PIR: Poverty income ratio, BMI: Body mass index, TP: Total protein, ALB: Albumin, BUN: Blood urea nitrogen, Cr: Creatinine, CRP: C-reactive protein, IQR: Interquartile range; Data represents baseline characteristics collected at the time of initial National Health and Nutrition Examination Survey) NHANES) survey between 2015 and 2018.

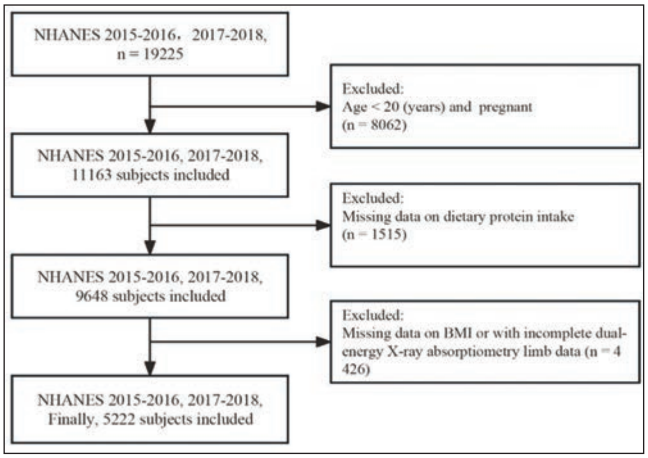

Figure-1: The study flow diagram.

NHANES: National Health and Nutrition Examination Survey, BMI: Body mass index

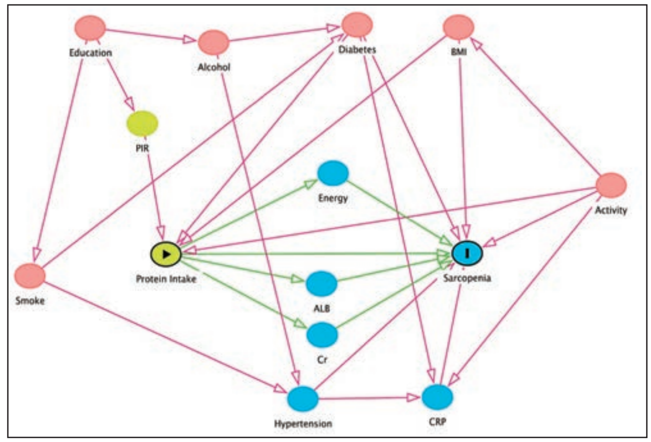

Figure-2: Relationships among studied variables. Blue lines represent confounders, red lines indicate intermediate variables, and green lines show stratification variables.

PIR: Poverty income ratio, BMI: Body mass index, ALB: Albumin, Cr: Creatinine, CRP: C-reactive protein.

Table-2: Association of covariates with sarcopenia risk.

Variable	OR (95% CI)	*p*-value	Variable	OR (95% CI)	*p*-value
Age (years)	1.04 (1.03~1.04)	<0.001	**Alcohol,** n (%)		
Gender, n (%)		0.045	No	1(reference)	
Male	1(reference)		Yes	0.56 (0.46~0.69)	<0.001
Female	0.83 (0.69~1)		**Activity,** n (%)		
Race, n (%)			Sedentary	1(reference)	
Non-Hispanic White	1(reference)		Mild	0.77 (0.57~1.04)	0.086
Non-Hispanic Black	0.26 (0.17~0.41)	<0.001	Moderate	0.74 (0.58~0.95)	0.018
Mexican American	3.77 (2.96~4.81)	<0.001	Vigorous	0.79 (0.63~0.99)	0.041
Other Race	1.51 (1.18~1.93)	0.001	**Hypertension,** n (%)		
Marital, n (%)			No	1(reference)	
Married or Living with partner	1(reference)		Yes	1.49 (1.23~1.82)	<0.001
Divorced or Live alone	0.82 (0.68~0.99)	0.042	**Diabetes,** n (%)		
PIR, n (%)			No	1(reference)	
<1.3	1(reference)		Yes	2.56 (2.02~3.24)	<0.001
1.3 - 3.49	0.7 (0.57~0.86)	0.001	**BMI** (kg/m^2)	1.11 (1.09~1.12)	<0.001
>3.5	0.43 (0.33~0.55)	<0.001	**TP** (g/L)	1 (0.98~1.02)	0.895
Education, n (%)			**ALB** (g/L)	0.91 (0.89~0.94)	<0.001
Less Than High School	1(reference)		**BUN** (mmol/L),	1.05 (0.99~1.1)	0.077
High School	0.51 (0.4~0.66)	<0.001	**Cr** (umol/L)	0.99 (0.98~0.99)	<0.001
More than High School	0.3 (0.24~0.37)	<0.001	**CRP** (mg/L)	1.03 (1.02~1.04)	<0.001
Smoke, n (%)			**Energy** (kcal/L)	1 (1~1)	<0.001
Never	1(reference)		**Protein intake** (g/L)	1 (0.99~1)	0.001
Former	1.03 (0.81~1.31)	0.808			
Current	0.79 (0.63~1.01)	0.06			

OR: Odds ratios, CI: Confidence interval, PIR: Poverty income ratio, BMI: Body mass index, TP: Total protein, ALB: Albumin, BUN: Blood urea nitrogen, Cr: Creatinine, CRP: C-reactive protein.

included in the model were demographic characteristics, education level, physical exercise, DM, smoking, alcohol consumption and BMI (Figure 2).

After removing any possible confounding variables, dietary protein consumption and sarcopenia were shown to be inversely correlated when analysed using quartiles (Table 3). The restricted cubic spline plots for each of the four models demonstrated a negative linear relationship between protein intake and sarcopenia (Figure 3). In the directed acyclic graph analysis, serum albumin, creatinine and total energy intake were identified as potential intermediate variables. When these variables were added to Model 3, the odds for protein intake quartiles moved towards the null (Q2: 0.68, 95%CI: 0.51-0.90; Q3: 0.71, 95%CI: 0.54-0.93; Q4: 0.42, 95%CI: 0.31-0.58). However, given their potential intermediary role in the causal pathway, these variables were not included in the final model to avoid over-adjustment.

There was no significant interactions in any of the subgroups (Figure 4).

Table-3: Association between dietary protein intake and sarcopenia.

Protein intake	n (%)	Crude		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
Q1	164 (12.6)	1(Ref)		1(Ref)		1(Ref)		1(Ref)	
Q2 vs Q1	128 (9.8)	0.76 (0.59~0.97)	0.026	0.61 (0.47~0.8)	<0.001	0.65 (0.5~0.84)	0.001	0.61 (0.46~0.81)	0.001
Q3 vs Q1	133 (10.2)	0.79 (0.62~1.01)	0.057	0.63 (0.48~0.82)	0.001	0.69 (0.53~0.9)	0.007	0.63 (0.48~0.84)	0.002
Q4 vs Q1	99 (7.6)	0.57 (0.44~0.74)	<0.001	0.37 (0.28~0.5)	<0.001	0.4 (0.3~0.54)	<0.001	0.36 (0.26~0.49)	<0.001
p-value for Trend			<0.001		<0.001		<0.001		<0.001

OR: Odds ratio, CI: Crude: Unadjusted for confounders, Model-1: Adjusted for age, gender, race and marital status, Model-2: Adjusted for model-1 + education level, physical activity status and diabetes, Model-3: Adjusted for model-2 + smoking status, alcohol consumption, body mass index (BMI).

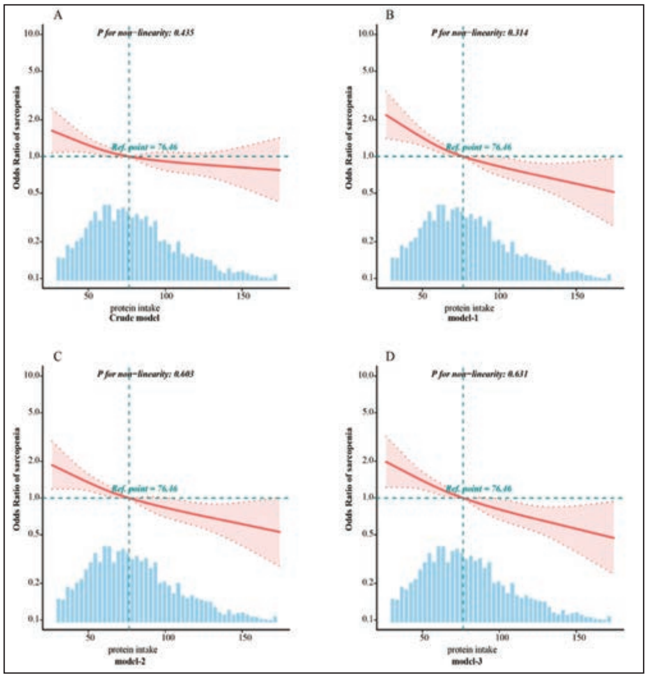

Figure-3: Association between dietary protein intake and sarcopenia odds ratio (OR), with all models showing significant linear associations (p<0.05 for non-linearity). A: Crude model, unadjusted for confounders. B: Model-1, adjusted for age, gender, race and marital status. C: Model-2, adjusted for model-1 + education level, physical activity status and diabetes. D: Model-3, adjusted for model-2 + smoking status, alcohol consumption, body mass index (BMI).

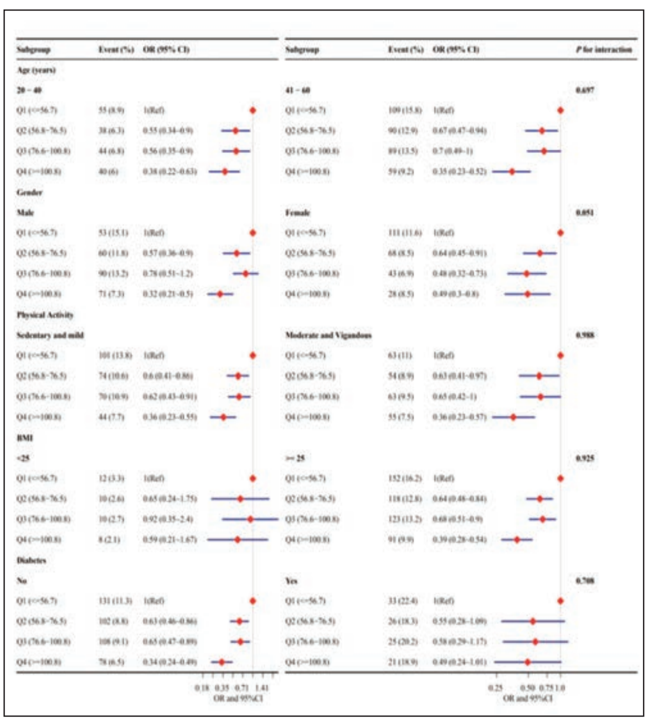

Figure-4: Relationship between dietary protein intake and ratio of sarcopenia in various subgroups.
OR: Odds ratio, BMI: Body mass index.

Discussion

The current, using NHANES 2015-2018 data, looked at the relationship between the prevalence of sarcopenia and daily protein consumption. According to the findings, there was a correlation between a lower incidence of sarcopenia and a greater protein consumption. This correlation persisted even after possible confounding variables were taken into account, such as age, gender, DM status, level of physical activity, and education. Subgroup analysis did not reveal any meaningful effect changes, suggesting that the association between sarcopenia and protein intake was consistent across a range of health-related and demographic variables.

The current results align with earlier studies that have shown the advantageous impact of increased protein consumption on both muscle mass and functionality. Higher protein consumption has been linked to a lower incidence of sarcopenia.[5,18,19] All these results lend credence to the idea that a sufficient protein diet is essential for both managing and preventing sarcopenia. It is crucial to recognise, nonetheless, that some studies have not shown any conclusive evidence linking protein consumption to sarcopenia.[9,10] The inconsistencies in the literature may be attributed to various factors, including differences in study populations, methodologies and definitions of sarcopenia. Additionally, there might be variations in the optimal protein intake thresholds for different populations, such as older adults with or without underlying health conditions.

The complicated and multifaceted processes that underlie the correlation between sarcopenia and protein consumption. Maintaining muscle mass and function requires muscle protein synthesis, which is mostly dependent on protein.[20] Sufficient consumption of protein supplies the amino acids required for the synthesis of muscle protein, promoting muscle development and repair.[21] Sarcopenia and muscle loss may ensue from an imbalance between the production and breakdown of muscle proteins caused by inadequate protein intake.[22] In addition to its direct effects on muscle protein synthesis, protein intake may also influence sarcopenia through other pathways. It has been displayed that protein increases muscle anabolism by triggering insulin-like growth factor-1 (IGF-1) signalling and the mammalian target of rapamycin (mTOR) pathway, which are two crucial regulators of muscle protein production.[23] Moreover, protein intake may improve physical performance and functional outcomes, which are important components of sarcopenia assessment.

The current results have significance for public health initiatives and clinical practice. Promoting adequate protein intake among older adults may be a valuable strategy for preventing and managing sarcopenia. Healthcare professionals should consider assessing the dietary protein intake of their older patients, and providing appropriate recommendations to ensure they meet their protein requirements. Furthermore, public health initiatives should focus on raising awareness about the importance of protein intake in maintaining muscle health and preventing sarcopenia among older adults.

The current study has limitations, like its cross-sectional design which limited the inference of causal relationships. Besides, 24-hour dietary recall interviews had been used to measure protein consumption, which is subject to both random errors (day-to-day variability in food intake) and systematic errors (underreporting of certain foods, portion size mis-estimation). Compared to dietary records and food frequency questionnaires, 24-hour recalls typically show higher within-individual variability, but lower between-individual variability, which may attenuate observed associations. The method may not adequately reflect long-term dietary habits and could lead to non-differential misclassification of exposure. Further, DEXA scanning is only applicable to participants aged <60, which means that high-risk elderly populations were not included in the analysis. These factors collectively affect the external validity of the results, making it difficult to derive clear clinical recommendations. Future research should design prospective cohort studies targeting specific elderly populations (≥60 years old) and use more accurate dietary assessment methods to validate the current findings. Moreover, the age range of the sample is too broad. Due to the fact that sarcopenia mainly occurs in the elderly population, the presence of young adults in the sample may have led to biased results. Participants in the 20-40 age group may not yet exhibit significant symptoms of sarcopenia, which may dilute the true association between protein intake and sarcopenia. Finally, data collection methods based on online interview recall may have introduced recall bias.

Conclusion

There was a correlation between higher dietary protein intake and lower incidence of sarcopenia. As part of a comprehensive strategy for treating and preventing sarcopenia, emphasising adequate protein intake is appropriate.

Disclaimer: None.

Conflict of Interest: None.

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Author Contribution:

DY: Design, preparation and final approval.

BL: Collected, analysed clinical data and final approval.

BW & MW: Data acquisition, analysis, interpretation, drafting and final approval.