The association between low-density lipoprotein cholesterol to apolipoprotein B ratio (LAR) and sarcopenia: a cross-sectional study

Keywords
NHANES, LAR, Cross-sectional study, Sarcopenia

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Material and methods

Data from NHANES cycles 2011-2016 were analyzed between July 2024 and February 2025. Sarcopenia was defined using dual-energy X-ray absorptiometry (DXA)-derived appendicular lean mass (ALM) standardized to body mass index (BMI). Multivariable logistic regression, restricted cubic spline (RCS) regression analysis, subgroup analysis, and interaction tests were applied to evaluate the relationship between LAR and sarcopenia, adjusting for covariates.

Results

A negative correlation between LAR and sarcopenia was observed in 3,235 participants included in the study (OR: 0.399, 95% CI: 0.224-0.712, P = 0.007), which was further confirmed to be non-linear via RCS regression analysis (Pnon-linear = 0.037), with one significant inflection point identified, and participants with LAR ≥ 1.268 demonstrated a significantly reduced risk of sarcopenia. Subgroup analyses and interaction tests indicated that the association between LAR and sarcopenia remained consistent across different subgroups and was not modified by other covariates.

Conclusions

Elevated LAR is significantly associated with lower sarcopenia risk, suggesting its potential role as a biomarker for muscle health. Further studies are needed to elucidate underlying mechanisms and validate these findings prospectively.

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Keywords: LAR; Sarcopenia; NHANES; Cross-sectional study

Introduction

Sarcopenia, a progressive and systemic syndrome characterized by the loss of skeletal muscle mass, strength, and function, has emerged as a critical public health challenge in aging populations. It is strongly associated with adverse clinical outcomes, including increased fracture risk, diminished quality of life, mobility impairment, and elevated mortality [1,2]. While traditionally linked to aging, its prevalence is rising among younger individuals, likely driven by modern sedentary lifestyles and metabolic dysregulation [3,4]. The pathophysiology of sarcopenia is multifactorial, encompassing hormonal imbalances, chronic inflammation, oxidative stress, and dysregulation in lipid metabolism [5,6]. Among these factors, dyslipidemia-a hallmark of metabolic syndrome-has gained attention for its potential role in accelerating muscle deterioration, though the mechanisms remain incompletely understood [7,8].

Conventional lipid markers, such as low-density lipoprotein cholesterol (LDL-C), have been implicated in sarcopenia risk. Elevated LDL-C levels correlate with reduced muscle strength and mass, possibly due to lipid accumulation in muscle tissue, which promotes lipotoxicity, mitochondrial dysfunction, and insulin resistance [9,10]. However, LDL-C alone fails to capture the atherogenic heterogeneity of lipoprotein particles, which varies in size, density, and apolipoprotein composition. The LDL-C/apolipoprotein B (ApoB) ratio (LAR) has emerged as a superior indicator of cardiovascular risk, reflecting the cholesterol content per atherogenic particle [11,12].

ApoB, a structural component of very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), and LDL particles, provides a direct measure of circulating atherogenic particle count. A lower LAR signifies smaller, denser LDL particles with heightened atherogenic potential, whereas a higher ratio suggests larger, cholesterol-enriched particles [13,14]. Beyond cardiovascular disease, this ratio may also offer insights into metabolic disturbances influencing muscle homeostasis. For instance, dysfunctional lipid metabolism could exacerbate sarcopenia through pathways such as oxidative stress, inflammation, and impaired myocyte repair [15,16]. Despite its clinical relevance, the relationship between LAR and sarcopenia remains unexplored, representing a critical gap in understanding how lipid particle characteristics intersect with muscle health.

Recent advancements in lipid biomarkers have highlighted the utility of novel ratios, such as the non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR), in predicting sarcopenia risk. Yang and Zhong [17] demonstrated that elevated NHHR independently correlates with sarcopenia prevalence, underscoring the importance of balancing atherogenic and antiatherogenic lipid fractions. Similarly, the triglyceride to high-density lipoprotein cholesterol (HDL-C) ratio (TG/HDL-C) has been linked to muscle loss, suggesting that lipid ratios capturing systemic metabolic dysregulation may serve as robust predictors of sarcopenia [18,19]. Building on these findings, the LAR-a marker of lipoprotein quality-warrants investigation as a potential biomarker for sarcopenia. Its ability to

differentiate between cholesterol-rich and particle-dense LDL subsets could elucidate distinct pathways through which dyslipidemia contributes to muscle atrophy. For example, smaller LDL particles may exhibit greater propensity for infiltration into muscle tissue, inducing endoplasmic reticulum stress and activating proteolytic pathways, while cholesterol overload may impair mitochondrial β-oxidation, reducing energy availability for muscle maintenance [20,21].

This study leverages data from National Health and Nutrition Examination Survey (NHANES), a nationally representative, cross-sectional survey of the non-institutionalized US population, to explore the association between LAR and sarcopenia in U.S. adults. We hypothesize that a lower LAR, indicative of atherogenic lipoprotein profiles, is associated with higher sarcopenia risk.

Material and methods

Ethical considerations

This study involving human participants, biological materials, and associated data was conducted in compliance with the Declaration of Helsinki. The protocol received ethical approval from the National Center for Health Statistics (NCHS) Institutional Review Board. Written informed consent was obtained from all participants prior to their inclusion in the research.

Study population

This cross-sectional study utilized data from the nationally representative NHANES conducted between 2011 and 2016. The study protocol was approved by the Ethics Review Board of the National Center for Health Statistics, and all participants provided written informed consent. Initially, the NHANES 2011-2016 included 29,902 participants. However, several exclusions were applied: individuals under 20 years of age (n = 12,854), those with incomplete LAR data (n = 9,903), participants lacking dual-energy X-ray absorptiometry (DXA) data (n = 3,731), and those with missing covariate information (n = 179). Individuals under 20 years of age were excluded as the core adult data on muscle health and fasting lipid profiles in NHANES are primarily collected for participants aged 20 and above. After these exclusions, the final analytical sample comprised 3,235 participants (see **Figure 1**). The detailed baseline characteristics of the included participants are presented in **Table 1** of the Results section.

Assessment of LAR (Exposure)

LAR was determined based on the lipid profiles of the participants. All subjects were required to provide fasting blood samples following a standardized protocol. ApoB concentrations were measured using immunonephelometry. LDL-C was calculated using the Friedewald formula: LDL-C = total cholesterol – HDL-C – triglycerides/5. The assays were performed using a Roche Cobas 6000 or Roche Modular P Chemistry Analyzer.

Sarcopenia assessment (Outcome)

In this study, sarcopenia was defined as the primary outcome. It was evaluated by measuring appendicular lean mass (ALM), which is the combined muscle mass of the four limbs [22]. ALM was assessed using DXA, a method that offers a comprehensive and precise evaluation of muscle mass and composition, in accordance with NHANES protocols. Participants who were taller than 192.5 cm, weighed more than 136.4 kg, or were pregnant were excluded due to equipment limitations for DXA scanning. The sarcopenia index was calculated by normalizing ALM to body mass index (BMI), resulting in an ALM/BMI ratio. Sarcopenia was identified using sex-specific thresholds for the sarcopenia index, with values <0.789 for men and <0.512 for women indicating the presence of the condition [22].

Potential covariates

Covariates were selected based on previous literature [17] and theoretical considerations regarding their potential association with both LAR levels and sarcopenia prevalence. Demographic variables included gender (male, female), age groups (20-39 years, ≥ 40 years), race/ethnicity (Mexican American, non-Hispanic White, non-Hispanic Black, Other Hispanic, other races), and education level (less than 9th grade, 9-11th grade, high school graduate, some college or Associate's degree, and college graduate or above). Marital status included categories: never married,

143 married, living with partner, separated, divorced, and widowed. The poverty income
144 ratio (PIR) was divided into two groups: below 1.3 and 1.3 or above. Anthropometric
145 measurements included BMI (classified as normal weight, overweight, or obese).
146 Smoking status was categorized as current, past, or never, and alcohol consumption
147 was dichotomized based on having at least 12 drinks per year. Age was categorized
148 into 20-39 and ⩾40 years to examine potential differences in muscle mass and
149 metabolic profiles between young adulthood and middle/older age.
150
151 **Statistical analyses**
152 Participants were categorized based on the presence or absence of sarcopenia.
153 Continuous variables were expressed as medians with interquartile ranges (IQR) and
154 categorical variables as frequencies with percentages. To compare continuous
155 variables, Student's t-test was used for normally distributed data, while the Mann-
156 Whitney U test was applied for non-normally distributed data. Chi-square tests
157 assessed categorical differences between groups.
158 Multivariate logistic regression was used to evaluate the independent relationship
159 between LAR and sarcopenia, with odds ratio (OR) and 95% confidence intervals (CI)
160 reported. For linear trends, NHHR was divided into quartiles, with the lowest quartile
161 as the reference group. Three models were applied: Model 1 (unadjusted), Model 2
162 (adjusted for age and gender), and Model 3 (adjusted for race, education, marital
163 status, smoking, and alcohol use). Participants were also stratified into two groups

based on initial LAR values above or below 1.2 to further investigate the relationship between LAR and sarcopenia [23]. Non-linear relationships between LAR and sarcopenia risk were evaluated using restricted cubic spline (RCS) regression. Subgroup analyses and interaction tests further explored variations across subgroups. Statistical analyses were performed with R statistical software (version 4.3.3; R Foundation for Statistical Computing). The primary R packages used in our analysis include: survey, haven, tidyverse, gtsummary, arsenal, and rms. A two-tailed *P*-value threshold of 0.05 was adopted for determining statistical significance throughout the study.

Results

Baseline characteristics

This study comprised 3,235 participants, of whom 1,574 were male (48.66%), and the median age was 39 (29, 49) years. A total number of 91.68% (2,966) and 8.32% (269) of the participants were distributed in the normal group and sarcopenia group severally (**Table 1**). The median LAR value was significantly lower ($P < 0.001$) in patients with sarcopenia (1.23; IQR: 1.13-1.31) compared to those without sarcopenia (1.27; IQR: 1.17-1.37). Besides, non-sarcopenia participants were more likely to have low education levels, low annual family income, be a current drinker and smoker, have high BMI, and so on. However, there was no significant statistical difference in gender between people with and without sarcopenia.

Multivariable logistic regression analysis

As shown in **Table 2**, weighted logistic regression analysis was utilized to explore the association between LAR and sarcopenia. When LAR was analyzed as a continuous variable, each unit increase in LAR was associated with a reduction in the OR by 79.1%, 78.3%, and 60.1% decrease in OR for the occurrence of sarcopenia from model 1 to model 3, respectively.

When LAR were categorized into two groups based on whether their baseline LAR was above or below 1.2, individuals in the higher LAR exhibited a 34.6%, 34.6% and 21.1% decreased risk of sarcopenia incidence from model 1 to model 3, respectively. After LAR was transformed into a categorical variable by quartiles, individuals in the highest quartile (Q4) exhibited a lower risk of sarcopenia incidence in a fully adjusted model (OR: 0. 560, 95% CI: 0.408-0.771, $P < 0.001$) compared to those in the lowest quartile (Q1).

Analysis of RCS regression

In restricted cubic spline regression, after the adjustment of potential covariates, a horizontally flipped J-shaped and significant non-linear relationship between LAR and sarcopenia ($P_{\text{non-linear}} = 0.037$) was detected (**Figure 2**), suggesting a threshold effect at higher LAR levels. Then, we conducted a segmented regression analysis, categorizing LAR into two groups based on the inflection point (≤ 1.268 vs. > 1.268).

For individuals with LAR ≤ 1.268, the adjusted OR was 1.030 (95% CI: 0.221-4.799; P = 0.969), indicating no significant association with sarcopenia risk in this range. In contrast, LAR >1.268 was strongly associated with reduced risk (OR = 0.039, 95% CI: 0.003-0.555; P = 0.018) (**Table 3**).

Subgroup analyses

The subgroup analyses were conducted in order to scrutinize the reliability and robustness of the relationship between LAR and sarcopenia across different subgroups. The aim was to uncover potential disparities in the association between LAR and sarcopenia risk within specific demographic contexts, including gender, age, PIR, education level, smoking status, alcohol consumption, BMI, marital status, and race. Besides, interaction tests revealed that such an association between LAR and sarcopenia was not modified by other covariates (all P for interaction > 0.05) (**Figure 3**).

Discussion

This cross-sectional study of 3,235 U.S. adults revealed a significant association between elevated LAR and decreased sarcopenia risk. Participants in the highest quartile of LAR exhibited a decreased risk of sarcopenia compared to the lowest quartile. The RCS analysis further demonstrated a non-linear relationship. These findings are consistent with emerging evidence associating dysregulation of lipid

metabolism with the deterioration of muscle health [5,7].

The observed association between LAR and sarcopenia likely reflects the interplay between atherogenic lipoproteins and skeletal muscle pathophysiology. Smaller, denser LDL particles-indicated by a lower LAR-are more prone to oxidative modification and endothelial permeation, enabling their infiltration into muscle tissue [20]. These processes accelerate muscle protein degradation, a hallmark of sarcopenia [10]. Excess cholesterol deposition in muscle mitochondria disrupts electron transport chain efficiency, leading to reactive oxygen species (ROS) overproduction and subsequent oxidative damage to myofibers [21].

The role of reverse cholesterol transport (RCT) in muscle health warrants attention. HDL-C facilitates RCT by shuttling excess cholesterol from peripheral tissues, including muscle, to the liver for excretion. Reduced HDL-C levels-common in individuals with elevated LAR-may compromise this process, leading to cholesterol accumulation in muscle cells. Preclinical studies suggest that impaired RCT disrupts membrane fluidity and signaling in myocytes, hindering muscle contraction and repair [24]. These mechanisms collectively highlight how LAR serves as a proxy for both lipid toxicity and metabolic inflammation, two key drivers of sarcopenia.

The present study identified a significant non-linear association between the LAR and sarcopenia risk, with a threshold effect observed at higher LAR levels. This finding suggests that the relationship between LAR and sarcopenia is not uniform

248 across the spectrum of LAR values but rather follows a horizontally flipped J-shaped
249 pattern, where only individuals exceeding the inflection point exhibit a clinically
250 meaningful reduction in sarcopenia risk. These results align with emerging evidence
251 highlighting non-linear relationships between metabolic biomarkers and
252 musculoskeletal outcomes. The observed threshold effect may reflect biological
253 mechanisms wherein higher LAR levels signify a predominance of larger, cholesterol-
254 enriched LDL particles over smaller, denser atherogenic particles. Larger LDL
255 particles have been linked to improved metabolic flexibility and reduced systemic
256 inflammation, which could indirectly preserve muscle mass and function by
257 mitigating chronic low-grade inflammation-a known contributor to sarcopenia
258 pathogenesis [1,25]. Furthermore, ApoB, a key component of LAR, serves as a
259 surrogate for lipoprotein particle number. A lower ApoB concentration relative to
260 LDL-C (i.e., higher LAR) may indicate reduced atherogenic burden, potentially
261 creating a metabolic milieu favorable to muscle homeostasis [26].

262 Subgroup analyses revealed nuanced variations in the association between LAR
263 and sarcopenia risk across demographic and lifestyle factors (**Figure 3**). The
264 protective effect of higher LAR was particularly pronounced in males, individuals
265 aged <40 years, those with a PIR <1.3, non-drinkers, and individuals with obesity.
266 Notably, the association remained significant among other races (P = 0.021), though
267 the interaction terms for race, gender, and other covariates were statistically non-
268 significant (P for interaction >0.05), suggesting potential homogeneity in the

threshold effect across subgroups despite differing baseline risks.

The stronger protective association observed in younger adults and socioeconomically disadvantaged groups (lower PIR and education levels) may reflect age- and environment-dependent metabolic adaptations. For instance, younger individuals often exhibit more efficient lipid metabolism, potentially amplifying the benefits of higher LAR on muscle preservation [1]. Similarly, socioeconomic stressors in disadvantaged groups could exacerbate inflammation and oxidative stress, making the anti-inflammatory properties of larger LDL particles (indicated by higher LAR) more critical for mitigating sarcopenia risk. The lack of significant interactions, however, implies that the threshold effect of LAR operates independently of these demographic factors, emphasizing its broad relevance as a biomarker.

Our findings extend prior research on lipid biomarkers and sarcopenia. Yang and Zhong [17] demonstrated that the NHHR independently predicts sarcopenia, emphasizing the balance between atherogenic and antiatherogenic lipids. Similarly, Lin et al. [8] identified the TG/HDL-C as a predictor of muscle loss. While these ratios broadly reflect dyslipidemia, the LAR offers unique insights into lipoprotein particle quality. ApoB quantifies the number of atherogenic particles (VLDL, IDL, LDL), whereas LDL-C measures the cholesterol content within these particles. Thus, a lower LAR signifies a predominance of small, dense LDL particles-each carrying less cholesterol but exhibiting greater oxidative susceptibility and tissue penetrance [11]. Future studies could incorporate more detailed NHANES questionnaire data on

aspects such as dietary patterns and physical activity to provide a more comprehensive understanding of the mechanisms linking LAR to sarcopenia.

Limitations

This study benefits from a large, nationally representative sample, rigorous adjustment for confounders, and standardized sarcopenia assessment using DXA. However, several limitations warrant caution. First, the cross-sectional design precludes causal inference. Reverse causation-where sarcopenia exacerbates dyslipidemia via reduced physical activity and altered energy metabolism-cannot be ruled out. Longitudinal studies were required to further clarify the causal relationships underpinning our findings. Second, residual confounding may persist due to unmeasured factors such as dietary patterns (e.g., saturated fat intake), genetic predispositions (e.g., APOE polymorphisms), or subclinical inflammation. Future studies should aim to account for additional previously unmeasured confounders (e.g., dietary patterns, genetic predispositions) to minimize this residual confounding risk. Third, DXA-derived ALM does not distinguish between muscle and connective tissue, potentially underestimating sarcopenia prevalence in individuals with high intramuscular fat. To achieve a more accurate assessment, future studies must utilize more advanced imaging techniques. Fourth, the study population was restricted to U.S. adults, limiting generalizability to other ethnicities or regions with distinct lifestyle and genetic backgrounds. Future multicenter, multinational studies are needed to

validate our findings and enhance their generalizability to diverse populations.

Conclusion

This study demonstrates a non-linear, threshold-dependent relationship between LAR and sarcopenia risk, with higher LAR significantly reducing sarcopenia risk. Subgroup analyses confirmed consistent associations across demographics, suggesting LAR's potential as a biomarker. Prospective studies are warranted to validate this inflection point and elucidate underlying mechanisms linking lipid metabolism to muscle health.

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Competing interests

The authors declare that they have no financial or personal relationships that may have inappropriately influenced them in writing this article.

Authors' contributions

X.Y. and Z.Z. conceived of the presented idea. X.Y. developed the theory and

performed the computations. X.Y. verified the analytical methods. Z.Z. supervised the findings of this work. All authors contributed to the article, discussed the results, and approved the final version for submission and publication.

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Data availability

Data will be made available on request. For requesting data, please write to the corresponding author.

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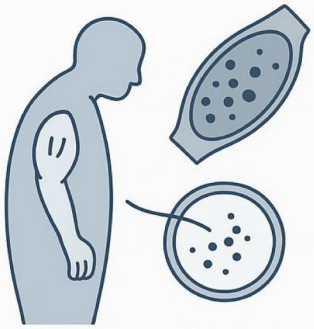
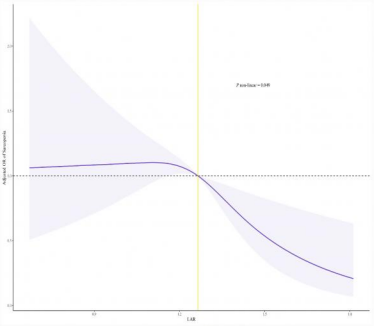
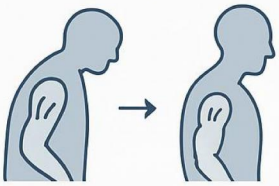
Sarcopenia



low LAR →→ small, dense LDL particles
muscle lipid infiltration
→ muscle loss

3,235 adults;
DXA-assessed ALM/BMI;
adjusted logistic &
spline regression





Higher LAR (> 1.268)
→ lower sarcopenia risk
(OR = 0.399, P = 0.007)

Suggests LAR as a potential biomarker for muscle health

Cross-sectional analysis
(NHANES 2011–2016)

Table 1. Baseline characteristics of the study population

Characteristics	Overall	Without sarcopenia	With sarcopenia	*P* value
	N = 3235 (100%)	N = 2966 (91.68%)	N = 269 (8.32%)	
Age group				**<0.001**
< 40	1661 (51.34%)	1568 (52.87%)	93 (34.57%)	
≥ 40	1574 (48.66%)	1398 (47.13%)	176 (65.43%)	
Gender				0.400
Female	1607 (49.68%)	1466 (49.43%)	141 (52.42%)	
Male	1628 (50.32%)	1500 (50.57%)	128 (47.58%)	
Race				**<0.001**
Mexican American	454 (14.03%)	373 (12.58%)	81 (30.11%)	
Other Hispanic	344 (10.63%)	303 (10.22%)	41 (15.24%)	
Non-Hispanic White	1239 (38.30%)	1149 (38.74%)	90 (33.46%)	
Non-Hispanic Black	648 (20.03%)	632 (21.31%)	16 (5.95%)	
Other/multiracial	550 (17.00%)	509 (17.16%)	41 (15.24%)	
BMI group				**<0.001**
Normal (< 25)	1061 (32.80%)	1034 (34.86%)	27 (10.04%)	
Overweight (25 to < 30)	1037 (32.06%)	969 (32.67%)	68 (25.28%)	
Obese (≥ 30)	1137 (35.15%)	963 (32.47%)	174 (64.68%)	
Drink group				**<0.001**

Characteristics	Overall	Without sarcopenia	With sarcopenia	*P* value
	N = 3235 (100%)	N = 2966 (91.68%)	N = 269 (8.32%)	
No	770 (23.80%)	683 (23.03%)	87 (32.34%)	
Yes	2465 (76.20%)	2283 (76.97%)	182 (67.66%)	
Smoke group				**0.010**
Never	1916 (59.23%)	1755 (59.17%)	161 (59.85%)	
Past	563 (17.40%)	502 (16.93%)	61 (22.68%)	
Current	756 (23.37%)	709 (23.90%)	47 (17.47%)	
Education				**<0.001**
Less Than 9th Grade	170 (5.26%)	130 (4.38%)	40 (14.87%)	
9-11th Grade	402 (12.43%)	365 (12.31%)	37 (13.75%)	
High School Grad/GED	698 (21.58%)	637 (21.48%)	61 (22.68%)	
Some College or AA degree	1020 (31.53%)	938 (31.63%)	82 (30.48%)	
College Graduate or above	945 (29.21%)	896 (30.21%)	49 (18.22%)	
PIR group				**<0.001**
≥ 1.3	2142 (66.21%)	1991 (67.13%)	151 (56.13%)	
< 1.3	1093 (33.79%)	975 (32.87%)	118 (43.87%)	
Marital Status				**0.036**
Married	1561 (48.25%)	1411 (47.57%)	150 (55.76%)	
Widowed	49 (1.51%)	44 (1.48%)	5 (1.86%)	

Characteristics	Overall	Without sarcopenia	With sarcopenia	*P* value
	N = 3235 (100%)	N = 2966 (91.68%)	N = 269 (8.32%)	
Divorced	282 (8.72%)	255 (8.60%)	27 (10.04%)	
Separated	112 (3.46%)	100 (3.37%)	12 (4.46%)	
Never married	880 (27.20%)	825 (27.82%)	55 (20.45%)	
Living with partner	351 (10.85%)	331 (11.16%)	20 (7.43%)	
Age (years)	39 (29,49)	38 (29, 48)	45 (35, 54)	**<0.001**
BMI (kg/m^2)	27.6 (23.7, 32.0)	27.2 (23.5, 31.4)	32.4 (28.1, 37.6)	**<0.001**
PIR	2.11 (1.03, 4.05)	2.17 (1.05, 4.10)	1.55 (0.88, 3.32)	**0.002**
Waist circumference (cm)	95.4 (84.5, 106.5)	94.5 (83.7, 105.3)	105.1 (94.1, 118.7)	**<0.001**
TG (mg/dL)	93 (64, 141)	91 (63, 137)	123 (88, 167)	**<0.001**
TC (mg/dL)	186 (162, 212)	185 (161, 212)	197 (170, 223)	**<0.001**
LDL-C (mg/dL)	111 (89,135)	110 (88, 134)	117 (98, 144)	**<0.001**
ApoB (mg/dL)	88 (72, 106)	87 (71, 105)	98 (82, 117)	**<0.001**
LAR	1.26 (1.16, 1.36)	1.27 (1.17, 1.37)	1.23 (1.13, 1.31)	**<0.001**
LAR group				**<0.001**
< 1.2	1066 (33%)	953 (32%)	113 (42%)	
≥ 1.2	2169 (67%)	2013 (68%)	156 (58%)	

Note: BMI: body mass index; LAR: LDL-C/ApoB ratio; LDL-C: low density lipoprotein cholesterol; PIR: poverty-to-income ratio; TC: total cholesterol; TG: triglyceride.

Table 2. Association between the prevalence of sarcopenia and the LAR as continuous and categorical variables

Characteristic	Model 1†			Model 2‡			Model 3§		
	OR	**95% CI**	***P* value**	**OR**	**95% CI**	***P* value**	**OR**	**95% CI**	***P* value**
LAR	0.209	0.124, 0.351	**<0.001**	0.217	0.128, 0.368	**<0.001**	0.399	0.224, 0.712	**0.007**
LAR group									
< 1.2	—	—		—	—		—	—	
≥ 1.2	0.654	0.522, 0.818	**<0.001**	0.654	0.520, 0.822	**<0.001**	0.789	0.632, 0.986	**0.038**
LAR Quartile									
Q1	—	—		—	—		—	—	
Q2	0.865	0.637, 1.176	0.348	0.881	0.648, 1.199	0.413	0.971	0.690, 1.367	0.863
Q3	0.734	0.545, 0.987	**0.041**	0.743	0.554, 0.997	**0.048**	0.935	0.686, 1.275	0.664
Q4	0.406	0.303, 0.544	**<0.001**	0.406	0.297, 0.554	**<0.001**	0.560	0.408, 0.771	**<0.001**

Note: OR: Odds Ratio; CI: Confidence Interval.

†Crude model: unadjusted.

‡Partial-adjusted model: adjusted for gender and age.

§Fully-adjusted model: adjusted for gender, age, race, PIR, education, marital status, smoking, drinking and BMI.

Table 3. Segment analysis

LAR	OR	95% CI	*P* value
≤ 1.268	1.030	0.221, 4.799	0.969
> 1.268	0.039	0.003, 0.555	**0.018**

Note: OR: Odds Ratio; CI: Confidence Interval.

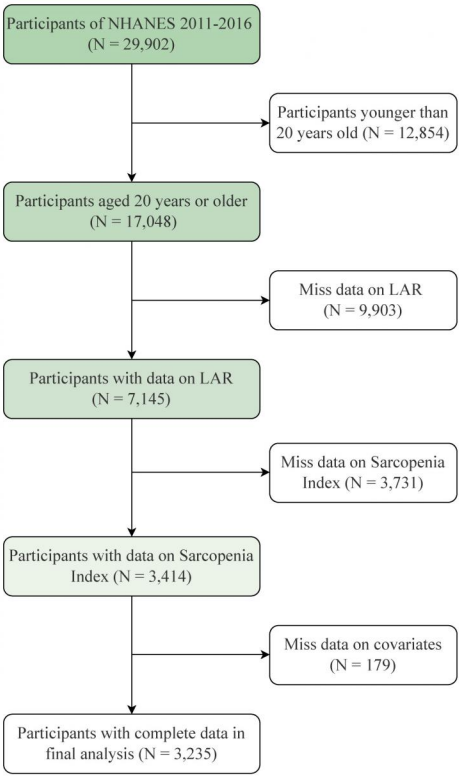


Figure 1: Participant selection flowchart from the 2011-2016 NHANES cycles.

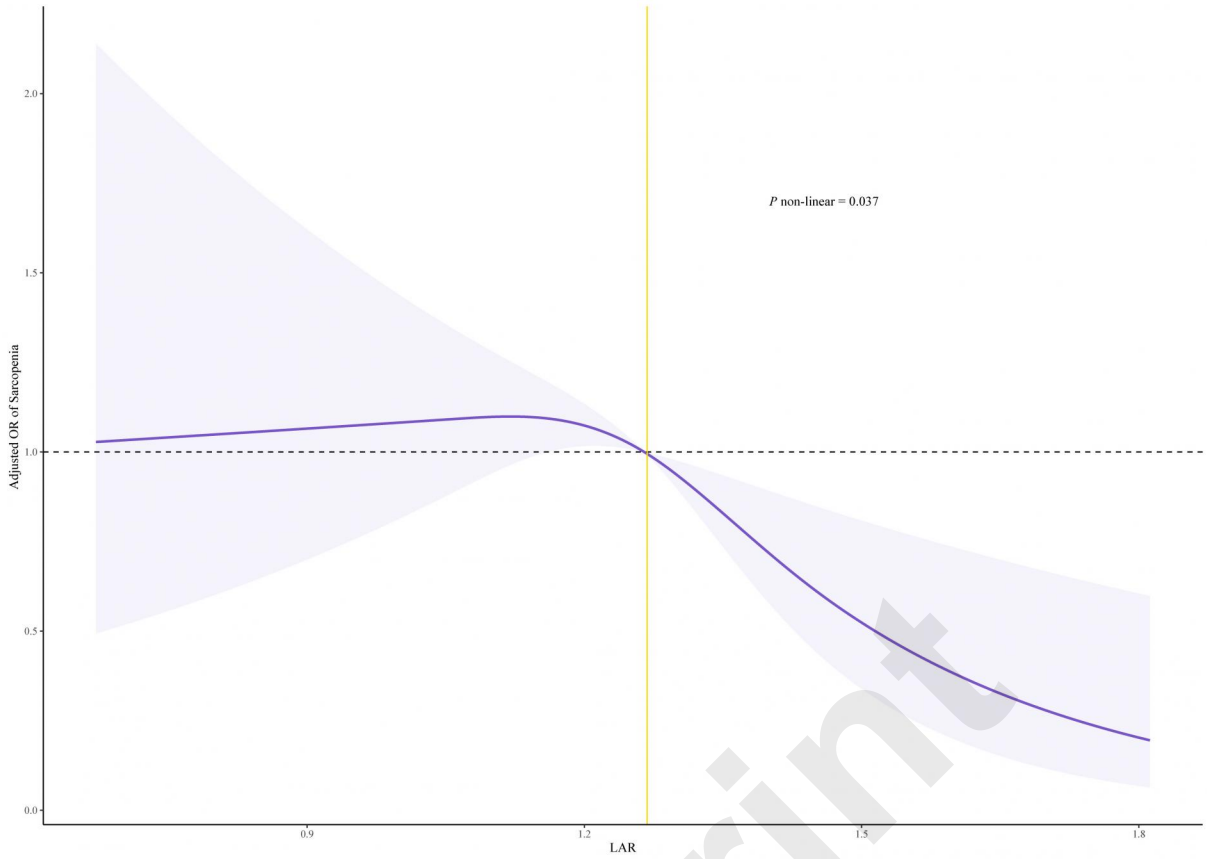
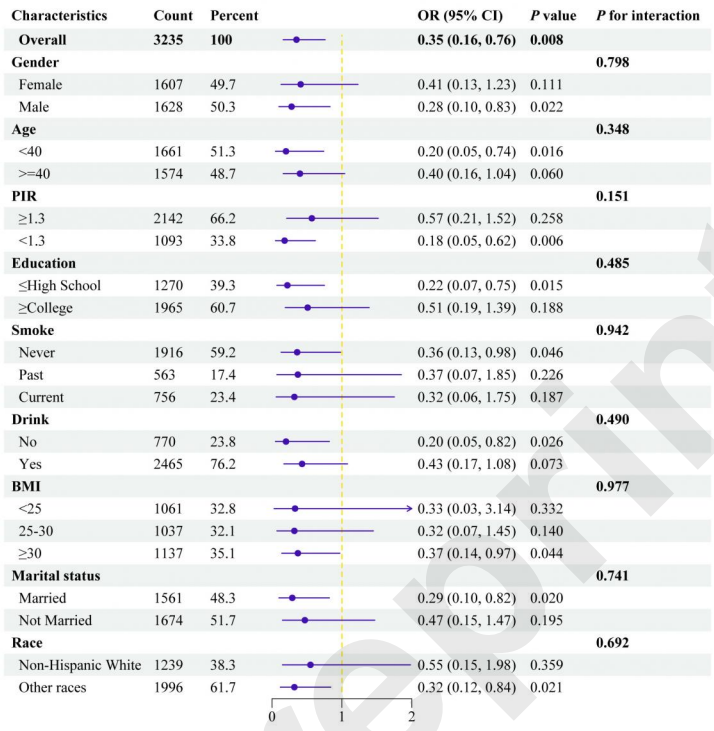


Figure 2: The restricted cubic spline (RCS) regression analysis of LAR with sarcopenia risk.



Characteristics	Count	Percent	OR (95% CI)	*P* value	*P* for interaction
Overall	**3235**	**100**	**0.35 (0.16, 0.76)**	**0.008**	
Gender					**0.798**
Female	1607	49.7	0.41 (0.13, 1.23)	0.111	
Male	1628	50.3	0.28 (0.10, 0.83)	0.022	
Age					**0.348**
<40	1661	51.3	0.20 (0.05, 0.74)	0.016	
>=40	1574	48.7	0.40 (0.16, 1.04)	0.060	
PIR					**0.151**
≥1.3	2142	66.2	0.57 (0.21, 1.52)	0.258	
<1.3	1093	33.8	0.18 (0.05, 0.62)	0.006	
Education					**0.485**
≤High School	1270	39.3	0.22 (0.07, 0.75)	0.015	
≥College	1965	60.7	0.51 (0.19, 1.39)	0.188	
Smoke					**0.942**
Never	1916	59.2	0.36 (0.13, 0.98)	0.046	
Past	563	17.4	0.37 (0.07, 1.85)	0.226	
Current	756	23.4	0.32 (0.06, 1.75)	0.187	
Drink					**0.490**
No	770	23.8	0.20 (0.05, 0.82)	0.026	
Yes	2465	76.2	0.43 (0.17, 1.08)	0.073	
BMI					**0.977**
<25	1061	32.8	0.33 (0.03, 3.14)	0.332	
25-30	1037	32.1	0.32 (0.07, 1.45)	0.140	
≥30	1137	35.1	0.37 (0.14, 0.97)	0.044	
Marital status					**0.741**
Married	1561	48.3	0.29 (0.10, 0.82)	0.020	
Not Married	1674	51.7	0.47 (0.15, 1.47)	0.195	
Race					**0.692**
Non-Hispanic White	1239	38.3	0.55 (0.15, 1.98)	0.359	
Other races	1996	61.7	0.32 (0.12, 0.84)	0.021	

Figure 3: Stratified analyses exploring LAR-sarcopenia associations across subgroups.