

Association between Blood Lipid Levels and Risk of Sarcopenic Obesity in Chinese Middle-Aged and Elderly Adults: A Cohort Study Postprint

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Abstract

Background With the increasingly severe problem of population aging, the incidence and impact of geriatric syndromes have gradually received widespread attention, and sarcopenic obesity (SO), as one of them, has also become a hot topic in recent research. Studies have shown that blood lipid levels may be an important influencing factor for SO; however, current research on the correlation between blood lipid components and SO has not yet reached consistent conclusions.

Objective To explore the association between blood lipid levels and the risk of incident SO among middle-aged and elderly Chinese adults.

Methods Based on data from the China Health and Retirement Longitudinal Study (CHARLS) from 2011 to 2015, a cohort study was conducted including middle-aged and elderly adults without SO at baseline in 2011. Baseline total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) in the study population were treated as exposure factors, and incident SO in 2013 and 2015 was treated as the outcome event. SO was defined using dual criteria for both sarcopenia and obesity, with sarcopenia determined based on appendicular skeletal muscle mass adjusted for body weight (ASM/W), and obesity defined as BMI ≥ 25 kg/m². Cox proportional hazards regression models were constructed to analyze the relationship between baseline TC, TG, HDL-C, LDL-C and the risk of incident SO, and restricted cubic spline models were used to examine potential non-linear associations.

Results A total of 5,268 participants were included, with a median age of 58 (52, 64) years and a cumulative follow-up of 20,592 person-years. There were 382 incident cases of SO, with a cumulative incidence of 7.25%, including

5.22% (128/2,451) in males and 9.02% (254/2,817) in females. Results from the fully adjusted Cox proportional hazards regression model showed that compared with the lowest quartile Q1 group for TC, the Q4 group (HR=1.35, 95%CI=1.00~1.82) had a significantly increased risk of incident SO; compared with the TG Q1 group, the Q2 (HR=1.55, 95%CI=1.08~2.21), Q3 (HR=2.07, 95%CI=1.48~2.90), and Q4 (HR=2.53, 95%CI=1.82~3.52) groups had significantly increased risk of incident SO ($P<0.05$); compared with the LDL-C Q1 group, the Q2 (HR=1.38, 95%CI=1.02~1.88) and Q4 (HR=1.44, 95%CI=1.07~1.95) groups also had significantly increased risk of incident SO ($P<0.05$); whereas compared with the HDL-C Q1 group, the Q2 (HR=0.75, 95%CI=0.58~0.96), Q3 (HR=0.54, 95%CI=0.41~0.71), and Q4 (HR=0.43, 95%CI=0.31~0.58) groups had significantly decreased risk of incident SO ($P<0.05$). Restricted cubic spline models indicated that TG levels showed an inverted L-shaped association with incident SO (P for non-linearity <0.001), while TC (P for non-linearity=0.731), HDL-C (P for non-linearity=0.600), and LDL-C (P for non-linearity=0.400) showed linear relationships with the risk of incident SO.

Conclusion TG, TC, and LDL-C are risk factors for incident SO in Chinese middle-aged and elderly populations, while HDL-C has a protective effect, with TG levels showing an inverted L-shaped association with incident SO. Therefore, blood lipid management may be of great significance for the prevention and treatment of SO in Chinese middle-aged and elderly populations.

Full Text

Associations of Lipid Levels and the Risk of Sarcopenic Obesity in Middle-aged and Elderly Chinese: a Cohort Study

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Abstract

Background With population aging intensifying, the incidence and impact of geriatric syndromes have garnered widespread attention, with sarcopenic obesity (SO) emerging as a key research focus. Studies suggest lipid levels may be an important factor influencing SO, yet research on the association between lipid components and SO has yielded inconsistent conclusions. **Objective** To investigate the relationship between blood lipid levels and SO risk among middle-aged

and elderly Chinese. **Methods** Using data from the China Health and Retirement Longitudinal Study (CHARLS) 2011-2015, we conducted a cohort study of middle-aged and elderly individuals without SO at baseline in 2011. Baseline total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) served as exposure variables, while incident SO in 2013 or 2015 was the outcome. SO was defined using dual criteria for sarcopenia and obesity: sarcopenia was determined by weight-adjusted appendicular skeletal muscle mass (ASM/W), and obesity was defined as $BMI \geq 25 \text{ kg/m}^2$. Cox proportional hazards regression models were constructed to analyze the relationship between baseline lipid levels and SO risk, with potential non-linear associations examined using restricted cubic spline models. **Results** Among 5,268 participants (median age 58 [52, 64] years) followed for 20,592 person-years, 382 new SO cases occurred, yielding a cumulative incidence of 7.25% (5.22% in men [128/2,451] and 9.02% in women [254/2,817]). In fully adjusted models, compared with the lowest quartile (Q1), the highest TC quartile (Q4) showed elevated SO risk (HR=1.35, 95%CI=1.00-1.82). For TG, Q2 (HR=1.55, 95%CI=1.08-2.21), Q3 (HR=2.07, 95%CI=1.48-2.90), and Q4 (HR=2.53, 95%CI=1.82-3.52) all demonstrated significantly increased risk. Similarly, LDL-C Q2 (HR=1.38, 95%CI=1.02-1.88) and Q4 (HR=1.44, 95%CI=1.07-1.95) showed elevated risk. Conversely, higher HDL-C quartiles were protective: Q2 (HR=0.75, 95%CI=0.58-0.96), Q3 (HR=0.54, 95%CI=0.41-0.71), and Q4 (HR=0.43, 95%CI=0.31-0.58). Restricted cubic spline analysis revealed an inverted L-shaped association between TG levels and SO risk (P-nonlinear<0.001), while TC (P-nonlinear=0.731), HDL-C (P-nonlinear=0.600), and LDL-C (P-nonlinear=0.400) showed linear relationships. **Conclusion** TG, TC, and LDL-C are risk factors for SO in Chinese middle-aged and elderly populations, whereas HDL-C is protective, with TG showing an inverted L-shaped association. Lipid management may thus be important for SO prevention and treatment in this population.

Keywords Middle aged; Aged; Sarcopenic obesity; Hyperlipidemias; Cholesterol, LDL; Cholesterol, HDL; Lipid levels; China Health and Retirement Longitudinal Study

1. Subjects and Methods

1.1 Study Population

Data were derived from the China Health and Retirement Longitudinal Study (CHARLS), a nationally representative longitudinal aging cohort of community-dwelling adults aged 45+ years. The study is publicly available at <https://charls.pku.edu.cn> with surveys conducted every 2-3 years (2011-2020). The CHARLS protocol was approved by the Peking University Biomedical Ethics Review Committee (approval IRB00001052-11015), and all participants provided written informed consent. To examine longitudinal associations

between lipids and SO, we used data from the 2011, 2013, and 2015 waves, with 2011 as baseline and 2015 as the outcome assessment point. The 2018 and 2020 waves lacked physical examination data required for SO assessment and were excluded.

Inclusion criteria comprised respondents with complete SO-related indicators in 2011. Exclusion criteria were: (1) age <45 years or missing data on age, lipids, sex, or medical history; (2) baseline SO diagnosis; and (3) missing SO information during follow-up (2013 or 2015). The final sample included 5,268 participants [Figure 1: see original paper].

1.2 Measurements

Exposure variables were four baseline lipid parameters: TC, TG, HDL-C, and LDL-C. Fasting venous blood samples were collected by trained medical staff after overnight fasting, centrifuged, and shipped to the Capital Medical University laboratory in Beijing for enzymatic colorimetric assay. Baseline lipid levels were categorized into quartiles: TC (Q1: <167.39 mg/dL, Q2: 167.39-190.59 mg/dL, Q3: 190.59-214.56 mg/dL, Q4: \$214.56 mg/dL); TG (Q1: <73.46 mg/dL, Q2: 73.46-102.66 mg/dL, Q3: 102.66-148.67 mg/dL, Q4: \$148.67 mg/dL); HDL-C (Q1: <40.97 mg/dL, Q2: 40.97-50.25 mg/dL, Q3: 50.25-60.69 mg/dL, Q4: \$60.69 mg/dL); and LDL-C (Q1: <93.55 mg/dL, Q2: 93.55-114.04 mg/dL, Q3: 114.04-136.37 mg/dL, Q4: \$136.37 mg/dL).

Outcome variable was incident SO, defined using combined criteria for sarcopenia and obesity. Obesity was defined as BMI ≥ 25 kg/m² based on Asia-Pacific standards [8]. For sarcopenia, we used weight-adjusted appendicular skeletal muscle mass (ASM/W) as the metric [9], calculated as: sarcopenia index = (ASM/W) \times 100%. ASM was estimated using a validated anthropometric equation for Chinese populations that shows good agreement with dual-energy X-ray absorptiometry (DXA) [10-11]: ASM = 0.193 \times weight (kg) + 0.107 \times height (cm) - 4.157 \times sex - 0.037 \times age (years) - 2.631, where sex was coded as 1 for male and 2 for female. Height and weight were measured using Seca TM213 stadiometers and Omron TM HN-286 scales. Following Janssen et al. [9], sarcopenia was defined as sarcopenia index below the sex-specific mean -1 SD of a young reference group (aged 18-39). Since CHARLS lacks young participants, we applied reference values from Kim et al. [12] derived from 4,918 Asian adults aged 20-39, with cutoffs of 30.18% for men and 23.75% for women.

Covariates included sociodemographic characteristics (sex, age, education, marital status, residence type), lifestyle factors (smoking and alcohol use), medical history (hypertension, diabetes, lipid-lowering medication), and C-reactive protein (CRP) levels.

1.3 Statistical Analysis

Data were analyzed using SPSS 27.0 and R 4.2.2. Normally distributed continuous variables are presented as mean \pm SD, non-normally distributed variables

as median (P25, P75), and categorical variables as percentages. Group comparisons used independent t-tests, Mann-Whitney U tests, or χ^2 tests as appropriate. Cox proportional hazards regression examined associations between baseline lipid levels and SO risk across three models: Model 1 (unadjusted), Model 2 (adjusted for age, sex, residence, education, marital status), and Model 3 (additionally adjusted for smoking, alcohol use, disease history, medication use, and CRP). Lipid levels were analyzed as categorical variables (quartiles) with Q1 as reference. Trend tests were performed across quartiles. Results are expressed as hazard ratios (HR) with 95% confidence intervals (CI). Restricted cubic spline analysis was used to explore potential non-linear associations for significant lipid parameters. Two-sided $P < 0.05$ was considered statistically significant.

2. Results

2.1 Baseline Characteristics

The 5,268 participants had a median age of 58 (52, 64) years and accumulated 20,592 person-years of follow-up. Incident SO occurred in 382 participants, yielding a cumulative incidence of 7.25% (5.22% in men [128/2,451] and 9.02% in women [254/2,817]). Incidence by age group was 4.85% (87/1,795) for ages 45- $<$ 55, 7.55% (163/2,158) for 55- $<$ 65, 10.22% (106/1,037) for 65- $<$ 75, and 9.35% (26/278) for \geq 75 years. Compared with those who remained SO-free, participants who developed SO were older and had higher baseline weight, BMI, CRP, TC, TG, and LDL-C, but lower height, sarcopenia index, and HDL-C. They were also more likely to be female, urban residents, less educated, non-smokers, non-drinkers, hypertensive, and taking lipid-lowering medication (all $P < 0.05$). Marital status and diabetes history did not differ significantly between groups .

2.2 Lipid Levels and SO Risk: Cox Regression Analysis

Using incident SO during follow-up as the outcome (yes=1, no=0) and baseline lipid quartiles as predictors (with Q1 as reference), Cox proportional hazards regression revealed significant associations after adjusting for sociodemographic characteristics, health behaviors, and chronic disease history ($P < 0.05$ for all lipids). Specifically, compared with TC Q1, the Q4 group showed elevated SO risk (HR=1.35, 95%CI=1.00-1.82). For TG, Q2 (HR=1.55, 95%CI=1.08-2.21), Q3 (HR=2.07, 95%CI=1.48-2.90), and Q4 (HR=2.53, 95%CI=1.82-3.52) all demonstrated significantly increased risk. For LDL-C, Q2 (HR=1.38, 95%CI=1.02-1.88) and Q4 (HR=1.44, 95%CI=1.07-1.95) showed elevated risk. Conversely, higher HDL-C quartiles were protective: Q2 (HR=0.75, 95%CI=0.58-0.96), Q3 (HR=0.54, 95%CI=0.41-0.71), and Q4 (HR=0.43, 95%CI=0.31-0.58). Significant dose-response trends were observed for all lipids (P -trend $<$ 0.05) .

2.3 Dose-Response Relationship Between Lipids and SO

Restricted cubic spline models adjusting for multiple confounders revealed a non-linear, inverted L-shaped association between baseline TG levels and SO risk (P-nonlinear<0.001), with risk increasing sharply as TG rose but plateauing when TG exceeded 116.67 mg/dL. In contrast, TC (P-nonlinear=0.731), HDL-C (P-nonlinear=0.600), and LDL-C (P-nonlinear=0.400) showed linear associations with SO risk [Figure 2: see original paper].

3. Discussion

This national longitudinal study of middle-aged and elderly Chinese examined the relationship between four lipid parameters and incident SO. We found positive associations of TC, TG, and LDL-C with SO risk, and an inverse association for HDL-C. Notably, TG exhibited an inverted L-shaped dose-response relationship.

Compared with the lowest quartile, the highest quartiles of TC (HR=1.35, 95%CI=1.00-1.82), TG (HR=2.53, 95%CI=1.82-3.52), and LDL-C (HR=1.44, 95%CI=1.07-1.95) were risk factors for SO, while HDL-C (HR=0.43, 95%CI=0.31-0.58) was protective, with TG showing a non-linear relationship. Previous research on dyslipidemia and SO is limited and mostly cross-sectional [6-7,13-14]. The Korea National Health and Nutrition Examination Survey (KNHANES) of 3,483 adults aged ≥ 65 found that SO was associated with dyslipidemia (OR=2.82, 95%CI=1.76-4.51), and in men, high TC and TG and low HDL-C were independently associated with SO [6]. Lu et al. [13] analyzed body composition in 600 Chinese adults and found high TG and low HDL-C correlated with increased SO risk. A cross-sectional study of 4,500 Chinese adults aged ≥ 50 reported TC (OR=1.35, 95%CI=1.12-1.63) and LDL-C (OR=1.45, 95%CI=1.15-1.83) as SO risk factors, but found no association for TG or HDL-C [7]. Another observational study of 14,926 adults aged 35-74 found only univariate associations of TC and HDL-C with SO that disappeared after adjustment [14], contrasting with our findings. These discrepancies may reflect differences in SO definitions. Baek et al. [6] and Lu et al. [13] used weight-adjusted muscle mass (ASM/W) and BMI $\geq 25 \text{ kg/m}^2$ for obesity, consistent with our approach, whereas Liu et al. [7] and Yin et al. [14] used height-squared adjustment (ASM/Ht²) and body fat percentage for obesity classification. Additionally, our longitudinal design examining baseline lipids and incident SO revealed the novel non-linear TG relationship, providing new evidence linking lipid metabolism to SO pathogenesis. Variations in adjusted covariates across studies may also contribute to divergent results.

The mechanisms linking lipids to SO remain incompletely understood. SO development is mediated by aging-related structural and functional changes in adipose and muscle tissues that interact reciprocally. Adipose tissue inflammation triggers ectopic fat distribution, particularly infiltration into skeletal muscle, im-

pairing muscle strength and function and promoting SO [15]. Intramyocellular lipid accumulation disrupts mitochondrial function, increases reactive oxygen species production, and impairs fatty acid β -oxidation, leading to lipotoxicity, insulin resistance, and local inflammation [16]. Inflammation further exacerbates muscle fat deposition and insulin resistance [17]. As muscle is a primary target of insulin, muscle mass loss worsens insulin resistance [18]. These processes create a vicious cycle of chronic inflammation, insulin resistance, and hyperlipidemia that drives SO progression. Thus, dyslipidemia may critically promote SO through multiple pathways including enhanced fat deposition, inflammation, and insulin resistance.

This study has limitations. First, skeletal muscle mass was estimated using a validated Chinese-specific anthropometric equation rather than DXA or bioelectrical impedance analysis, though this equation shows good agreement with DXA. Second, we relied on a single baseline lipid measurement, precluding assessment of lipid trajectories' impact on SO. Finally, although we adjusted for multiple confounders, unmeasured factors such as nutritional status and physical activity level could influence results. Physical activity data were too incomplete for inclusion, and CHARLS does not collect nutrition data.

In conclusion, this cohort study demonstrates that TG, TC, and LDL-C are risk factors for SO, while HDL-C is protective, with TG showing an inverted L-shaped association. These findings underscore the importance of lipid management for SO prevention in Chinese middle-aged and elderly populations and provide potential biomarkers and targets for intervention.

Author Contributions: XU Chunyan conceptualized the study, curated data, performed statistical analysis, and wrote the original draft. HE Ling curated data, provided statistical guidance, and assisted with manuscript revision. GUO Canhui and LAI Hurong curated data and contributed to feasibility analysis. LIAO Caifeng curated data. TU Huaijun supervised the project, ensured quality control, and was responsible for the overall article. All authors approved the final manuscript.

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