Bidirectional transitions of sarcopenia, obesity, and sarcopenic obesity: Transitional patterns in older adults from the CHARLS study

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To the Editor: Sarcopenia, defined by the European Working Group on Sarcopenia in Older People (EWGSOP) as the presence of both low muscle mass and low muscle strength or performance, has emerged as a critical public health concern in aging populations.[1] Moreover, sarcopenic obesity (SO), first characterized by Baumgartner as the coexistence of sarcopenia and obesity, was another high-risk geriatric syndrome.[2] Obesity and sarcopenia often coexist in the aging population and exhibit strong bidirectional associations. A recent meta-analysis encompassing 106 clinical studies ($N = 167,151$ older individuals) reported a pooled SO prevalence of 9% in both sexes.[3] Importantly, it revealed a 51% increased risk of all-cause mortality (pooled hazard ratio [HR] = 1.51, 95% confidence intetal [CI] 1.14–2.02, $P < 0.001$) among individuals with SO compared to their healthy counterparts.[3] Given its adverse health implications, emerging research underscores the need to approach SO as a distinct, multifactorial phenotype. Consequently, identifying robust predictors of SO is imperative for targeted interventions.

This study investigated the progression of SO and the factors influencing its dynamic nature. Such insights could be critical for identifying optimal intervention targets and determining the most effective time windows for tailored treatments. Since sarcopenia and obesity share several pathophysiological mechanisms and may exacerbate each other, this study aimed to investigate five-year transitions among sarcopenia, obesity, SO, and normal stages. In addition, we evaluated potential factors associated with these transitions in adults aged ≥50 years using data from the China Health and Retirement Longitudinal Study (CHARLS). The Biomedical Ethics Review Committee of Peking University approved the CHARLS study (approval number IRB00001052–11015), and all interviewees were required to provide informed consent.

Initially, 17,705 participants were included in 2011. From this cohort, we identified 5631 individuals aged ≥50 years who had complete objectively assessed data on walking speed, grip strength, height, weight, and waist circumference. Subsequently, 2420 individuals were excluded due to loss to follow-up or missing anthropometric data in 2013 or 2015, resulting in a final sample of 3211 participants.

Sarcopenia was assessed at baseline and each follow-up in accordance with the criteria of the Asian Working Group for Sarcopenia (AWGS2019). Muscle mass was quantified using the skeletal mass index (SMI), calculated as the appendicular skeletal muscle mass (ASM) divided by the square of the height in meters. ASM was estimated as follows: ASM = 0.193 × body weight + 0.107 × height – 4.157 × gender – 0.037 × age – 2.631 (where body weight is in kg, height in cm, age in years, and gender is coded as 1 for men and 2 for women).[4] Low muscle mass was defined as SMI <7.0 kg/m^2 for men and <5.7 kg for women. In addition, gait speed was considered low if it fell below 1.0 m/s. Following the AWGS2019 algorithm, sarcopenia was classified under the presence of "low muscle mass and low muscle strength" or "low muscle mass and low physical performance". "Severe sarcopenia" was defined as the combination of low muscle strength, low muscle mass, and low physical performance. "Non-sarcopenia" was defined as the presence of normal muscle mass and strength.

Obesity was assessed using waist circumference (WC), which was measured at the midpoint between the lower

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ribs and the ilium with participant standing upright. Obesity was defined as WC ≥80 cm for females and WC ≥90 cm for males.[5] Cognitive function was assessed by the Telephone Interview of Cognitive Status (TICS-10), word recall, and figure drawing, with data from the CHARLS.

To identify factors associated with transitions toward and away from sarcopenic obesity, we examined previously established risk factors. The following baseline (Year 1) variables were included: age, sex, sleep duration, living location, educational level, body mass index (BMI), health status, smoking habit, alcohol consumption, and chronic diseases.

Baseline characteristics were summarized using frequencies (percentages) for categorical variables and means (standard deviation) for continuous variables. These characteristics were compared across six groups: normal, mild to moderate sarcopenia, severe sarcopenia, obesity, obesity with mild to moderate sarcopenia, and obesity with severe sarcopenia. Continuous variables, including age and sleep duration, were analyzed using one-way analysis of variance (ANOVA), the Kruskal–Wallis *H* test, and Student's *t*-test. For categorical variables like sex and living location, comparisons were conducted using χ^2 and Fisher's exact test. A value of $P < 0.05$ (two-side) was considered to be statistically significant.

The multistate Markov model (MSM) of sarcopenia was illustrated in Supplementary Figure 1, http://links.lww.com/CM9/C710. Individuals could transition between states under the assumption that both muscular conditions and obesity could improve (backward transitions), deteriorate (forward transitions), or remain stable (state maintenance) between successive study visits. Given the low mortality rate during the 4-year follow-up, we incorporated two absorbing states: State 0 (loss to follow-up) and State 7 (death). Once death occurs, no further transitions are possible. Thus, the final model comprised eight states (six transient and two absorbing). Observed transitions over the 4-year period were quantified as absolute counts and proportions.

To estimate transition probabilities, a continuous-time multistate Markov model was constructed, which was appropriate for modeling longitudinal state progression. After excluding 187 transitions leading to death (an absorbing state) from the 2013–2015 follow-up, the final dataset included 9446 transitions across 3211 participants for model training. The fitted model was applied to estimate transition probabilities at 1-year (short-term), 2-year (short-term), and 4-year (mid-term) intervals for each state. To investigate how covariates influence the instantaneous risk of specific transitions, MSM with covariates were trained to obtain HRs along with 95% CIs, following Jackson's methodology.

As for factor selection, variables with a scientific theoretical basis for influencing transitions between sarcopenia and obesity were prioritized. These included sociodemographic characteristics (gender, age, living location, educational level), nutritional status (BMI), comorbidity index (number of chronic diseases), and risk behaviors (smoking and drinking habits). To balance model complexity and convergence, variables were standardized or categorized into low, medium, and high levels. For time-varying variables (age, BMI, chronic disease status), the dataset was updated when changes were reported in the survey.

Factors were tested individually by incorporating them into the model one at a time, and these were then used to train the final model. To improve model convergence, the Broyden–Fletcher–Goldfarb–Shanno quasi-Newton optimization algorithm was used. Considering that sarcopenia and obesity conditions evolve gradually rather than abruptly, our model assumed transitions only between adjacent states, implying that individuals skipping states still transitioned through unrecorded intermediate stages. Thus, the predictions were limited to transitions between adjacent and absorbing states.

For sensitivity analyses, the Kruskal–Wallis single-factor analysis indicated that patients with chronic diseases exhibited a higher likelihood of transitioning to sarcopenia (either mild-to-moderate or severe). To further investigate, we performed sensitivity analyses by excluding individuals with chronic comorbidities, thereby exploring transition-associated factors in a comorbidity-free subset.

The baseline characteristics of the 4856 included participants are presented in Supplementary Table 1, http://links.lww.com/CM9/C710. At baseline (2011), the cohort was stratified into six distinct clinical states: normal (1187, 24.4%), mild-to-moderate sarcopenia (1065, 21.9%), severe sarcopenia (325, 6.7%), obesity (1734, 35.7%), obesity with mild-to-moderate sarcopenia (431, 8.9%), and obesity with severe sarcopenia (114, 2.3%).

Participants in the normal and obesity group were significantly younger, reported longer sleep durations, and attained higher educational levels compared to the other four states. In contrast, individuals with sarcopenia exhibited an age-dependent severity gradient, with increasing age correlating with sarcopenia progression (from mild-to-moderate to severe). Furthermore, sarcopenic individuals tended to have a lower educational level, lower BMI index, poor health status, lower muscle strength, and lower walking speed than those in the normal state. The sarcopenic obesity individuals tended to be older, have a lower educational level, and poorer health status than those in the normal state [Supplementary Table 1, http://links.lww.com/CM9/C710].

The inflows and outflows between the six clinical states at follow-up intervals (2011, 2013, and 2015) are illustrated in Figure 1. The total observable transitions between consecutive assessments are detailed in Supplementary Table 2, http://links.lww.com/CM9/C710 (censored states excluded) and Supplementary Table 3, http://links.lww.com/CM9/C710 (censored states included). Based on these observations and the transition intensity matrix, the 1-, 2-, and 4-year transition probabilities are estimated in Supplementary Table 4, http://links.lww.com/CM9/C710.

As shown in Supplementary Table 5, http://links.lww.com/CM9/C710, several factors associated with sarcopenia transitions in our sample were presented. For comprehensive multivariate analyses, including transitions to

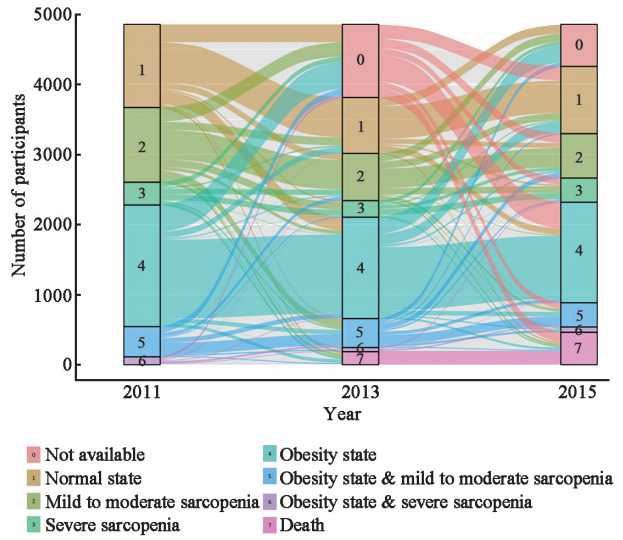


Figure 1: Alluvial plot illustrating the transitions between sarcopenia stages in the CHARLS study participants over 4-year follow-up ($N = 4856$).

death, refer to Supplementary Table 6, http://links.lww.com/CM9/C710. The sensitivity analysis, which excluded individuals with at least two comorbidities from the total sample, examined the associations of the concerned indices with sarcopenia-state transition probabilities through MSM in different models [Supplementary Table 7, http://links.lww.com/CM9/C710].

The MSM was used to delineate the transition dynamics of sarcopenic obesity and assess the associations between modifiable and non-modifiable factors with progression or reversion from baseline states (non-sarcopenic/non-obese). The prevalence of sarcopenic obesity in our sample was 11.2%, which was slightly higher than rates reported in comparable populations.[6] Discrepancies likely arise from methodological variations, particularly in sarcopenia assessment. Unlike studies relying on direct measurements (e.g., Dual-energy X-ray absorptiometry), we estimated SMI via anthropometric equations, which may inflate prevalence estimates compared to other studies.

However, several limitations should also be noted. First, the limited number of deaths during follow-up (187 in 2013 and 464 in 2015) and the substantial loss to follow-up may have influenced the results. Second, the follow-up period was relatively short, encompassing only the short- to medium-term phase and not extending into the long term.

In conclusion, sarcopenia, obesity, and sarcopenic obesity were dynamic conditions that typically progress slowly during early stages, with potential reversions to a normal state. Elevated levels of physical activity and cognitive performance would be associated with an increased likelihood of transitioning from mild or moderate sarcopenia or obesity back to normal status. Conversely, advancing age consistently predicted transitions toward severe sarcopenia. Furthermore, longer sleep duration appeared to facilitate transitions from severe to milder sarcopenia. These findings highlighted the importance of early interventions aimed at preserving physical activity, maintaining cognitive function, and managing sleep time. Future investigations would develop and assess effective interventions for individuals already with sarcopenia or sarcopenic obesity.

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Conflicts of interest

None.

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