Research paper

# Long-Term Weight Patterns and Risk of Sarcopenia in Community-Dwelling Older Adults: A Three-Year Cohort Study

**Keywords**
Body mass index, Older adults, Sarcopenia, Muscle mass, Obesity dynamics

**Abstract**

Introduction

Static body mass index (BMI) measurements fail to capture the clinical implications of body weight fluctuations. Therefore, this study investigated the association between multi-dimensional obesity dynamics and incident sarcopenia in community-dwelling older adults.

Material and methods

The analytic cohort included 1,091 participants. Of these, 478 completed all assessments at the 3-year follow-up and were analyzed. Despite this attrition, baseline characteristics remained comparable between completers and those lost to follow-up. Obesity dynamics were assessed using BMI trajectories, variability, 3-year change, and cumulative overweight-years. Sarcopenia was defined according to the 2019 Asian Working Group for Sarcopenia (AWGS) criteria. We applied logistic regression models, adjusting for demographics, comorbidities, lifestyle factors, and physical activity.

Results

Persistent overweight (odds ratio (OR) = 0.31, 95% confidence interval (CI): 0.14-0.67, $p = 0.003$) and persistent obesity (OR = 0.21, 95% CI: 0.06-0.74, $p = 0.016$) were associated with significantly lower odds of developing sarcopenia compared with a stable normal BMI, whereas decreased BMI was linked to more than a twofold higher risk (OR = 2.70, 95% CI: 1.17-6.23, $p = 0.020$). Our analysis of sarcopenia components demonstrated that maintaining an overweight or obese status, along with higher cumulative overweight-years, correlated with a reduced risk of developing low muscle mass (all $p < 0.001$).

Conclusions

These findings emphasize the importance of monitoring weight trajectories to prevent unintentional weight loss rather than promoting obesity in later life. Clinicians should prioritize maintaining weight stability to mitigate sarcopenia risk.

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**KEYWORDS:** Sarcopenia, Body mass index, Obesity dynamics, Older adults, Muscle mass.

**ABBREVIATIONS:** BMI, body mass index; BIA, bioelectrical impedance analysis; AWGS, Asian Working Group for Sarcopenia; OR, odds ratio; CI, confidence interval; SMI, skeletal muscle mass index; CAD, coronary artery disease; IPAQ, International Physical Activity Questionnaire; SD, standard deviation; GLP-1, glucagon-like peptide-1.

## Introduction

Sarcopenia has emerged as a major public health challenge in aging societies [1, 2], substantially increasing the risk of falls, dementia, and mortality [3, 4]. Concurrently, obesity prevalence is rising among older adults [5]. While obesity is traditionally linked to cardiometabolic morbidity [6], its relationship with adverse events in later life remains controversial [7]. When obesity coexists with sarcopenia, it is defined as sarcopenic obesity [8, 9]. This condition may further increase the risk of adverse health outcomes [4, 10].

Although the concept and consequences of sarcopenic obesity have been widely studied [9-11], this static approach overlooks the longitudinal nature of obesity dynamics that may contribute to sarcopenia. Existing studies have provided limited evidence for this relationship. A U.S. study reported that a lower body mass index (BMI) was associated with a higher risk of low muscle mass [12]. Consistently, a study from China suggested that higher BMI may protect against sarcopenia [13]. In addition, one Brazilian study [14] found a protective effect of transitioning to overweight, though the trajectories partly relied on self-reported data. Moreover, evidence regarding the relationship between BMI trajectories and the components of sarcopenia remains scarce [15]. Most existing research relies on cross-sectional, single-point BMI measurements, which fail to distinguish between heterogeneous health states. For instance, a static BMI value cannot differentiate between stable health and a trajectory of rapid, unintentional weight loss that may signal underlying muscle wasting [16, 17].



To address these gaps, this study investigates obesity dynamics from four distinct longitudinal perspectives, including BMI trajectory, BMI variability, BMI change, and cumulative overweight-years. We hypothesize that persistent obesity is inversely associated with incident sarcopenia. We aim to identify which obesity dynamics are associated with the risk of incident sarcopenia and to provide more precise intervention recommendations for older adults.

## Material and Methods

### Study population

This prospective cohort study included community-dwelling adults aged 65 years and older who underwent annual health examinations. Participants who were unable to communicate effectively or undergo body composition analysis were excluded. Participants with implanted cardiac pacemakers were also excluded to prevent potential electrical interference during bioelectrical impedance analysis (BIA) and to ensure measurement accuracy. After obtaining written informed consent, baseline data were collected, and participants were subsequently followed annually for three years. A three-year window is considered sufficient in geriatric research to observe measurable declines in muscle health and the transition from robust status to incident sarcopenia in community-dwelling populations [18]. Demographic information, including age, sex, and a history of chronic medical conditions, was obtained through a structured questionnaire. Clinical assessments and relevant laboratory tests were then performed. The Institutional Review Board of (anonymity) approved this study, and all procedures followed the principles of the Declaration of Helsinki.



### Assessment of obesity dynamics

We used BMI as the diagnostic parameter for obesity because it serves as a widely accepted and practical screening tool in routine clinical practice [8, 19]. BMI ($kg/m^2$) was measured at baseline and during follow-up visits at 12, 24, and 36 months. We derived four indicators to capture different aspects of longitudinal BMI change. First, we modeled BMI trajectories using latent class trajectory analysis to identify distinct patterns of BMI change over time [20]. This statistical technique identifies subgroups of participants who share similar longitudinal patterns (trajectories). Second, BMI variability [21] was calculated across repeated BMI assessments to reflect intraindividual fluctuations. Third, BMI change was defined as the difference between baseline and 36-month BMI. Finally, we estimated cumulative overweight-years using the trapezoidal method [22] by summing the extent and duration of BMI values above the Taiwanese cutoff of 24 kg/$m^2$ [23]. This localized BMI cutoff is used because Asian populations exhibit a higher risk of cardiometabolic risk at lower BMI levels compared to Western cohorts. Detailed definitions are provided in the Supplementary Methods.

**Sarcopenia assessment**

We assessed sarcopenia according to the Asian Working Group for Sarcopenia (AWGS) 2019 criteria [24]. Sarcopenia-related parameters included low muscle mass (<7.0 kg/$m^2$ in males and <5.7 kg/$m^2$ in females, measured by InBody 720 BIA), low muscle strength (handgrip strength <28 kg in males and <18 kg in females, measured by Jamar Hand Dynamometer), and poor physical performance (defined as a 6-meter walking speed <1.0 m/s). Robust was defined as normal muscle mass, strength, and performance. Possible sarcopenia was defined by low muscle strength or low physical performance alone. Sarcopenia was diagnosed when low muscle mass is present with either low strength or performance, and severe sarcopenia was defined by the coexistence of all three. These parameters were evaluated at baseline and again at 36 months. To focus on incident cases, we excluded participants with possible or confirmed sarcopenia at baseline.



**Covariates**

Covariates were selected a priori based on evidence from the literature as potential confounders of both obesity and sarcopenia. Anthropometric measurements included height (meters), body weight (kilograms), and waist circumference (centimeters).

Medical history was collected through self-reported questionnaires, including a history of stroke, physician-diagnosed coronary artery disease (CAD), osteoporosis diagnosed based on a bone mineral density examination, chronic arthritis (e.g., osteoarthritis or rheumatoid arthritis), and depression. Smoking status was categorized into two groups based on participants' responses: (1) non-smoker (never smokers and former smokers) and (2) current smoker. Alcohol consumption was categorized into two groups according to drinking frequency: (1) non-regular drinkers (never, <1 time per month, or 1–2 times per month) and (2) regular drinkers (once per week, 2–3 times per week, or almost every day). We evaluated physical activity using the International Physical Activity Questionnaire (IPAQ) [25].

**Statistical analyses**

Descriptive statistical analyses were performed. We assessed the normality of continuous variables using the Shapiro–Wilk test. The Kruskal–Wallis test and ANOVA were applied for continuous variables, whereas the chi-square test was used for categorical variables to compare the defined sarcopenia groups. Logistic regression analysis was performed to evaluate the association between obesity dynamics and sarcopenia groups in separate models. This approach was preferred over time-to-event analysis because sarcopenia assessments were conducted at discrete annual intervals. Due to the binary nature of logistic regression, we categorized participants into two groups. Robust and possible sarcopenia were combined as the reference, while sarcopenia and severe sarcopenia formed the comparison group to focus on the clinical progression toward advanced stages. Four BMI indicators were used as independent variables. The trajectory of BMI was identified using the latent class trajectory models, resulting in three distinct classes. BMI variability was categorized into tertiles based on its distribution. BMI change was classified into three groups, and cumulative overweight-years were also divided into tertiles for further analysis. Two multivariable models were constructed. Model 1 was adjusted for age and sex. Model 2 additionally included stroke, coronary artery disease, arthritis, depression, smoking, alcohol consumption, and physical activity.

Finally, several sensitivity analyses were performed to evaluate the robustness of the results. First, since cancer may lead to weight loss, we excluded participants with a history of cancer and performed the logistic regression analysis again. Second, because some underweight participants (BMI < 18.5 kg/m²) might influence the results, we excluded them and repeated the logistic regression analysis. Third, because categorizing BMI variability and cumulative overweight-years into tertiles may lead to loss of information and reduce statistical power, we additionally conducted sensitivity analyses treating these measures as continuous variables. Logistic regression models were repeated using the continuous forms of BMI variability and overweight-years to examine whether the observed associations were consistent with those obtained from the categorical analyses.

Obesity trajectory patterns were identified using Mplus (version 8.1), and other statistical analyses were conducted with SPSS version 25 (SPSS, Inc., Chicago, IL).

## Results

### Characteristics of the Study Population

A total of 1,819 older adults were enrolled. We excluded those with communication difficulties, inability to complete body composition assessment, or cardiac pacemakers (n=18), and those with possible sarcopenia or sarcopenia at baseline (n=710). The analytic cohort included 1,091 participants. Of these, 478 completed all assessments at the 3-year follow-up and were analyzed. Among them, 359 (75.1%) were robust, 83 (17.4%) had possible sarcopenia, 29 (6.1%) had sarcopenia, and 7 (1.5%) had severe sarcopenia (**Table 1**). Participants with sarcopenia and severe sarcopenia were significantly older than robust individuals, and they had lower BMI, waist circumference, grip strength, gait speed, and skeletal muscle mass index (SMI) (all $p < 0.001$). In contrast, the possible sarcopenia group had higher BMI and waist circumference compared with the robust group. No significant differences were observed for CAD, osteoarthritis, depression, smoking, alcohol drinking, or physical activity. To assess attrition bias, we compared baseline characteristics between participants who completed the study and those who were lost to follow-up (**Supplementary Table 1**). At baseline, age, sex, BMI, and most comorbidities did not significantly differ between participants who completed follow-up and those who did not. However, lost participants had lower grip strength. Overall, the attrition bias was minimal. In summary, participants who developed sarcopenia were generally older and had poorer baseline physical metrics.

BMI changes differed by subgroup over the 3-year follow-up. BMI levels remained relatively stable within each trajectory (**Figure 1**). Participants with decreased BMI showed a mean reduction of 1.7 kg/m² (from 26.0 ± 3.4 to 24.3 ± 3.4), whereas those with increased BMI had a mean gain of +1.7 kg/m² (from 23.6 ± 3.0 to 25.3 ± 3.2).

For cumulative overweight-years, the difference between the highest and lowest tertiles persisted at both baseline (27.5 vs. 21.5 kg/m²) and follow-up (27.5 vs. 21.4 kg/m²), reflecting stable but distinct BMI levels across the tertile groups (**Supplementary Table 2**).

**Association between obesity dynamics and sarcopenia**

In our trajectory analysis, we observed that participants who maintained a stable overweight or obese status throughout the 3-year period were significantly less likely to develop sarcopenia compared to those with a stable normal BMI. Conversely, individuals who experienced a decline in BMI over the study period showed a higher risk of incident sarcopenia. Participants with persistent overweight (OR = 0.31, 95% CI: 0.14-0.67) and persistent obesity (OR = 0.21, 95% CI: 0.06-0.74) had significantly lower odds of developing sarcopenia than those with stable normal BMI across all models (**Table 2**). The ORs remained consistent after adjusting for demographics, comorbidities, and lifestyle factors.



In contrast, participants with decreased BMI showed more than a twofold increased risk of sarcopenia (OR = 2.70, 95% CI: 1.17-6.23), whereas increased BMI was not associated with sarcopenia risk. BMI variability across tertiles did not show significant associations with sarcopenia risk. Regarding cumulative overweight-years, higher exposure was associated with reduced risk: participants in the highest tertile had lower odds of sarcopenia (OR = 0.20, 95% CI: 0.06-0.60) than those in the lowest tertile, consistent across all adjusted models. Taken together, these results indicate that while stable overweight or obese status and higher cumulative exposure were associated with reduced sarcopenia risk, a longitudinal decline in BMI emerged as a significant risk factor.

## Associations between obesity dynamics and sarcopenia components

When examining the components of sarcopenia, we found that long-term exposure to excess weight was inversely associated with the development of low muscle mass. Specifically, those with higher cumulative overweight-years had significantly lower odds of developing low muscle mass. For low muscle mass, persistent overweight (OR = 0.10, 95% CI: 0.06-0.17) and persistent obesity (OR = 0.02, 95% CI: 0.01-0.07) were associated with lower odds of low muscle mass compared with stable normal weight. Higher cumulative overweight-years were also associated with reduced risk (T3: OR = 0.04, 95% CI: 0.02-0.09; **Table 3**). In contrast, BMI change and variability were not significantly associated with low muscle mass. For low grip strength, none of the obesity dynamics measures showed significant associations, although persistent obesity suggested a non-significant trend toward increased risk (**Supplementary Table 3**). Similarly, for low gait speed, persistent overweight and obesity were not significantly associated with risk (**Supplementary Table 4**). Overall, obesity dynamics were associated with muscle mass preservation but showed no statistically significant associations with muscle strength or physical performance.



## Sensitivity analyses

Excluding participants with cancer (n=39) or with BMI < 18.5 (n=9) did not substantially change the results (**Supplementary Table 5-6**). Persistent overweight (OR = 0.32-0.33, 95% CI: 0.14-0.74) and obesity (OR = 0.21-0.23, 95% CI: 0.06-0.85) remained associated with lower odds of sarcopenia, whereas decreased BMI consistently showed higher odds (OR = 3.00-3.02, 95% CI: 1.24-7.27). The highest tertile of cumulative overweight-years was also associated with lower odds of sarcopenia (OR = 0.20-0.24, 95% CI: 0.06-0.73), whereas BMI variability was not statistically significant. In addition, when modeled as continuous variables, higher BMI variability was associated with increased risk of sarcopenia (β = 0.18, 95% CI: 0.04-0.33), whereas greater cumulative overweight-years were inversely associated (β = -2.8, 95% CI: -4.85 to -0.76), consistent across adjusted models (**Supplementary Table 7**).



## Discussion

To our knowledge, this is the first prospective study to evaluate multi-dimensional obesity dynamics and incident sarcopenia in community-dwelling older adults. Persistent overweight and persistent obesity were associated with a reduced risk of sarcopenia. The effects were also more pronounced among individuals with higher cumulative overweight-years. In contrast, BMI decline was linked to a significantly elevated risk of sarcopenia, while BMI variability showed no consistent associations. Furthermore, obesity dynamics were associated with more favorable outcomes for muscle mass than for muscle strength or physical performance. These findings provide longitudinal evidence consistent with one aspect of the "obesity paradox," suggesting that a higher but stable BMI in later life may mitigate specific adverse outcomes such as sarcopenia. However, this does not imply that obesity is universally protective. Recent evidence indicates that such paradoxical associations in older adults cannot be reliably defined without precise body composition assessment and a multidimensional frailty framework [26, 27].

This study showed that persistent overweight or obesity was associated with a significantly lower risk of sarcopenia compared with stable normal weight. Similar findings have been reported elsewhere, but the evidence remains limited by study design and population differences. A Korean study indicated that obesity defined by high BMI in older women may help preserve muscle mass and reduce sarcopenia risk [28], while a Swedish cohort study found that each one-unit increase in baseline BMI was linked to a 25% lower risk of sarcopenia [29]. Importantly, our findings extend current knowledge by addressing limitations of static BMI snapshots. For example, an individual with a BMI of 25 $kg/m^2$ could be on a trajectory of weight loss due to illness or on a trajectory of weight gain associated with improved health. This study's strength lies in using longitudinal BMI changes to provide a more precise understanding of sarcopenia risk than single measurements.

Conversely, participants who experienced a significant decline in BMI during the study period had more than a twofold increased risk of sarcopenia. This finding suggests that weight loss rather than low BMI may serve as an important warning sign for the development of sarcopenia. Such weight loss may indicate disease, malnutrition, or muscle wasting, all of which are closely linked to sarcopenia [16, 30]. Notably, the mean BMI in the decreased BMI group declined to 24.3. Although this level is close to the normal-weight range, this reduction was associated with an increased risk of sarcopenia. Because a decline in BMI more than doubles the risk of developing sarcopenia, weight loss associated with glucagon-like peptide-1 (GLP-1) receptor agonists may warrant close clinical monitoring [31]. While these agents offer metabolic benefits, our results underscore the potential need for clinical attention regarding muscle health.



In contrast, when BMI variability was categorized into tertiles, no significant association with sarcopenia risk was observed. However, sensitivity analyses using BMI variability as a continuous variable revealed that greater variability was associated with an increased risk of sarcopenia. This indicates that large fluctuations in BMI, even over relatively short periods, are associated with adverse outcomes. Such effects may result from asynchronous changes in adipose and muscle tissues during repeated weight cycling [32]. Repeated weight cycling is associated with faster and more complete regain of fat mass compared to muscle mass. This process results in a net loss of muscle and a higher risk of low muscle mass and strength.

When sarcopenia is examined by its three components of muscle mass, strength, and physical performance, the influence of obesity trajectories appears to be mainly restricted to muscle mass. Persistent overweight or obesity, as well as longer cumulative exposure to excess weight, are significantly associated with greater muscle mass. This effect is likely due to chronic mechanical loading on bones and muscles [33] that promotes hypertrophy and structural adaptations [34]. However, this benefit does not extend to muscle strength or physical performance and may be offset by fat infiltration and impaired neuromuscular activation [35]. Muscle mass represents the pathological substrate of sarcopenia, whereas declines in grip strength and gait speed reflect functional manifestations [36]. The preservation of muscle mass may be more readily detected by current assessment methods, while functional decline often emerges later [37, 38]. Notably, overweight and obesity have been associated with a higher likelihood of probable sarcopenia when defined by lower limb strength, but a protective effect when defined by handgrip strength [39]. Nevertheless, while a higher BMI may reduce the risk of low muscle mass, it does not necessarily confer protection against all adverse outcomes. For example, sarcopenic obesity (the coexistence of excess adiposity and low muscle mass) is associated with increased morbidity and mortality [40].

Our findings suggest that persistent overweight or obesity may mitigate the risk of developing low muscle mass. However, excess adiposity remains a well-established risk factor for cardiometabolic disease, functional decline, and increased mortality in older adults [41]. Clinical efforts should remain focused on preventing rapid weight loss trajectories rather than promoting weight gain. Muscle mass preservation does not necessarily translate into preserved muscle strength, physical performance, or overall health outcomes. Meanwhile, metabolic phenotypes related to obesity may also influence muscle health. Recent studies have shown that the low-density lipoprotein cholesterol (LDL-C) to apolipoprotein B (ApoB) ratio, which reflects the cholesterol content within each atherogenic lipoprotein particle, is associated with the risk of sarcopenia [42]. In addition, the triglyceride-glucose index, a surrogate marker of insulin resistance, has also been associated with low muscle mass [43], suggesting more complex interactions between muscle health and metabolic risk. In fact, sarcopenic obesity characterized by the coexistence of high adiposity and low muscle mass has been consistently associated with frailty, disability, cardiovascular disease, and premature death [9]. Furthermore, sarcopenic obesity is closely linked to metabolic dysfunction-associated steatotic liver disease, a condition that further elevates the risk of chronic kidney disease as well as cardiovascular and all-cause mortality [44, 45]. Therefore, our results should not be interpreted as advocating for obesity in late life. Rather, they highlight the need to prevent unintentional weight loss and to promote strategies that preserve both muscle mass and function while minimizing the adverse consequences of excess adiposity.

Strengths and Limitations

Nevertheless, this study has several potential limitations. First, although participants with sarcopenia at baseline were excluded, the possibility of reverse causation cannot be fully ruled out. Underlying comorbidities may precipitate weight loss and subsequently lead to sarcopenia. We conducted sensitivity analyses excluding participants with cancer or underweight. The results remained consistent and partially alleviated this concern. Second, our reliance on BMI as the primary indicator of obesity is a notable limitation. Although BMI is a practical clinical tool, it serves only as a proxy and cannot distinguish between fat mass and muscle mass. Other obesity indicators such as waist to height ratio and fat mass measured by BIA were not extensively analyzed. Consequently, our findings should be interpreted with caution regarding the specific role of fat mass. In addition, while our assessments utilized BIA due to its clinical practicality in community settings, we acknowledge its limitations in precision compared to dual-energy X-ray absorptiometry. Third, participants who were lost to follow-up exhibited lower grip strength at baseline. This suggests that attrition may be related to the study outcome. This bias could attenuate the negative association between BMI and grip strength, as weaker individuals were more likely to drop out. Consequently, faster participants were overrepresented at follow-up. Such bias tends to drive the association toward the null. The persistence of a significant negative association suggests that the true effect might have been even stronger. Fourth, our study did not collect detailed information on dietary intake or specific nutritional status. The absence of dietary data may leave residual confounding in the association between BMI trajectories and incident sarcopenia. Another limitation is the lack of data on educational attainment and cognitive function, which were not included in our baseline assessment. Education and cognitive health are known to be associated with lifestyle choices, nutritional intake, and physical activity levels in older adults, all of which could influence the development of sarcopenia. Fifth, our study population consisted of community-dwelling older adults, and the findings may not be fully generalizable to other populations.



**Conclusion**

This study highlights the importance of considering more than single-point BMI measurement when evaluating the impact of obesity on sarcopenia in older adults. The primary clinical takeaway is that preventing unintentional weight loss and maintaining stable weight trajectories are critical for preserving muscle health in later life. Importantly, clinical decisions should not rely on BMI alone. Instead, practitioners should prioritize comprehensive strategies that emphasize muscle preservation, nutritional support, and the maintenance of physical function. The observed associations relate mainly to muscle mass and do not justify interpreting overweight or obesity as protective for overall health or physical function. These findings may help inform more precise clinical guidelines and individualized strategies for sarcopenia prevention and management in aging populations.



## ACKNOWLEDGEMENT

The study was funded and supported by the National Science and Technology Council in Taiwan(112-2314-B-016-019-). The funders had no role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript.

## CONFLICT OF INTEREST

There are no potential conflicts of interest to disclose.



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**Figure legend**

Figure 1. BMI trajectories derived from 478 participants. Each plot depicts the mean BMI at each time point. Groups represent distinct trajectory categories, with the proportion of participants in each group shown in parentheses. A thicker dashed line represents a BMI of 24, while a thinner dashed line represents a BMI of 27.

## Obesity Dynamics and Sarcopenia in Older Adults

**Obesity dynamics**

1. BMI trajectory
2. BMI variability
3. BMI change
4. Cumulative overweight-years

**Study population & Follow up**

Community-dwelling older adults (≥65 years)

3-years follow up

**Incident sarcopenia risk**

Persistent overweight and obesity
↓odds of sarcopenia

Decreased BMI
>2-fold increase

**Clinical takeaway: preventing unintentional weight loss and maintaining stable weight trajectories.**

Table 1. Baseline characteristics categorized by sarcopenia status at the 3-year follow-up.

| Characteristic | Overall (n=478) | Robust (n=359) | Possible sarcopenia (n=83) | Sarcopenia (n=29) | Severe sarcopenia (n=7) | p-value |
|---|---|---|---|---|---|---|
| Age, years | 70.50 ± 5.54 | 69.73 ± 4.92 | 72.75 ± 6.44 | 72.48 ± 7.38 | 74.86 ± 6.09 | **< 0.001[a]** |
| BMI, kg/m² | 24.06 ± 3.16 | 23.88 ± 3.13 | 25.64 ± 2.73 | 22.13 ± 2.72 | 22.19 ± 3.60 | **< 0.001[b]** |
| Waist circumference, cm | 79.71 ± 8.99 | 79.41 ± 8.98 | 83.33 ± 7.91 | 73.39 ± 7.52 | 78.29 ± 10.48 | **< 0.001[b]** |
| Grip strength, kg | 30.43 ± 8.69 | 31.16 ± 8.88 | 29.28 ± 7.91 | 26.34 ± 6.38 | 23.86 ± 6.41 | **< 0.001[b]** |
| Gait speed, m/s | 1.35 ± 0.24 | 1.38 ± 0.24 | 1.23 ± 0.18 | 1.24 ± 0.19 | 1.19 ± 0.17 | **< 0.001[b]** |
| SMI | 6.74 ± 1.05 | 6.79 ± 1.07 | 6.94 ± 0.86 | 5.89 ± 0.86 | 5.87 ± 0.84 | **< 0.001[b]** |
| Body fat, % | 29.77 ± 7.05 | 29.07 ± 6.81 | 32.70 ± 7.2 | 29.97 ± 7.84 | 30.47 ± 6.2 | **< 0.001[b]** |
| Physical activity, MET-minutes/week | 9948.06 ± 3404.30 | 10008.11 ± 3433.74 | 9850.18 ± 3507.13 | 9581.48 ± 2872.23 | 9538.07 ± 3169.96 | 0.893[a] |
| Sex | | | | | | 0.389[c] |
| Male | 204 (42.7%) | 161 (44.8%) | 31 (37.3%) | 10 (34.5%) | 2 (28.6%) | |
| Female | 274 (57.3%) | 198 (55.2%) | 52 (62.7%) | 19 (65.5%) | 5 (71.4%) | |
| Stroke | 5 (1%) | 2 (0.6%) | 2 (2.4%) | 0 (0.0%) | 1 (14.3%) | **0.002[c]** |
| CAD | 26 (5.4%) | 19 (5.3%) | 5 (6.0%) | 1 (3.4%) | 1 (14.3%) | 0.946[c] |
| Arthritis | 67 (14%) | 48 (13.4%) | 13 (15.7%) | 2 (6.9%) | 4 (57.1%) | 0.053[c] |
| Osteoarthritis | 64 (13.4%) | 46 (12.8%) | 13 (15.7%) | 4 (13.8%) | 1 (14.3%) | 0.941[c] |
| Depression | 15 (3.1%) | 12 (3.3%) | 1 (1.2%) | 1 (3.4%) | 1 (14.3%) | 0.594[c] |
| Current smoking | | | | | | 0.764[c] |
| Non-smoker | 463 (96.9%) | 349 (97.2%) | 79 (95.2%) | 28 (96.6%) | 7 (100%) | |
| Smoker | 15 (3.1%) | 10 (2.8%) | 4 (4.8%) | 1 (3.4%) | 0 (0.0%) | |
| Alcohol drinking | | | | | | 0.426[c] |
| Non-regular drinkers | 407 (85.1%) | 301 (83.8%) | 74 (89.2%) | 25 (86.2%) | 7 (100%) | |
| Regular drinkers | 71 (14.9%) | 58 (16.2%) | 9 (10.8%) | 4 (13.8%) | 0 (0.0%) | |

Abbreviations: BMI, body mass index; SMI, skeletal muscle mass index; CAD, coronary artery disease.
Continuous variables are presented as mean ± standard deviation, and categorical variables are presented as number (percentage).
Classification of sarcopenia status was based on follow-up assessments.
Values with p < 0.05 are shown in bold.
Test statistic: a = Kruskal–Wallis test, b = ANOVA, and c = Chi-square test

Table 2. Multivariable-adjusted odds ratios (95% CI) of incident sarcopenia according to obesity dynamic indicators among older adults over 3 years.

| | Model 1<br>OR (95% CI) | Model 2<br>OR (95% CI) |
|---|---|---|
| BMI trajectory | | |
| Stable-normal weight (n=157) | 1.00 | 1.00 |
| Persistent overweight (n=231) | **0.31 (0.14-0.66)** | **0.31 (0.14-0.67)** |
| Persistent obesity (n=90) | **0.21 (0.06-0.73)** | **0.21 (0.06-0.74)** |
| BMI change | | |
| Stable (n=341) | 1.00 | 1.00 |
| Decreased (n=66) | **2.50 (1.10-5.66)** | **2.70 (1.17-6.23)** |
| Increased (n=71) | 1.05 (0.37-2.92) | 1.07 (0.37-3.04) |
| BMI variability | | |
| Tertile 1 (n=139) | 1.00 | 1.00 |
| Tertile 2 (n=141) | 0.66 (0.26-1.73) | 0.63 (0.24-1.68) |
| Tertile 3 (n=141) | 1.20 (0.52-2.77) | 1.15 (0.50-2.69) |
| Cumulative overweight-years | | |
| Tertile 1 (n=200) | 1.00 | 1.00 |
| Tertile 2 (n=80) | **0.32 (0.11-0.97)** | 0.33 (0.11-1.01) |
| Tertile 3 (n=141) | **0.20 (0.07-0.61)** | **0.20 (0.06-0.60)** |

Abbreviations: BMI, body mass index; OR, odds ratio; CI, confidence interval.

Model 1 Adjusted age and sex.

Model 2 Additional adjustment stroke, coronary artery disease, arthritis, depression, smoking, alcohol consumption, and physical activity.

Values with $p < 0.05$ are shown in bold.

Table 3. Longitudinal associations between BMI dynamic indicators and the risk of developing low muscle mass in older adults over 3 years.

| | Model 1<br>OR (95% CI) | Model 2<br>OR (95% CI) |
|---|---|---|
| BMI trajectory | | |
| Stable-normal weight (n=157) | 1.00 | 1.00 |
| Persistent overweight (n=231) | **0.10 (0.06-0.17)** | **0.10 (0.06-0.17)** |
| Persistent obesity (n=90) | **0.02 (0.01-0.07)** | **0.02 (0.01-0.07)** |
| BMI change | | |
| Stable (n=341) | 1.00 | 1.00 |
| Decreased (n=66) | 0.94 (0.52-1.68) | 1.02 (0.57-1.85) |
| Increased (n=71) | 0.74 (0.40-1.37) | 0.79 (0.42-1.48) |
| BMI variability | | |
| Tertile 1 (n=139) | 1.00 | 1.00 |
| Tertile 2 (n=141) | 0.60 (0.36-1.02) | 0.60 (0.35-1.02) |
| Tertile 3 (n=141) | 0.66 (0.39-1.11) | 0.63 (0.37-1.07) |
| Cumulative overweight-years | | |
| Tertile 1 (n=200) | 1.00 | 1.00 |
| Tertile 2 (n=80) | **0.10 (0.04-0.21)** | **0.10 (0.04-0.21)** |
| Tertile 3 (n=141) | **0.04 (0.02-0.10)** | **0.04 (0.02-0.09)** |

Abbreviations: BMI, body mass index; OR, odds ratio; CI, confidence interval.

Model 1 Adjusted age and sex.

Model 2 Additional adjustment stroke, coronary artery disease, arthritis, depression, smoking, alcohol consumption, and physical activity.

Values with $p < 0.05$ are shown in bold.

Low muscle mass definition: <7.0 kg/m² in males and <5.7 kg/m² in females.

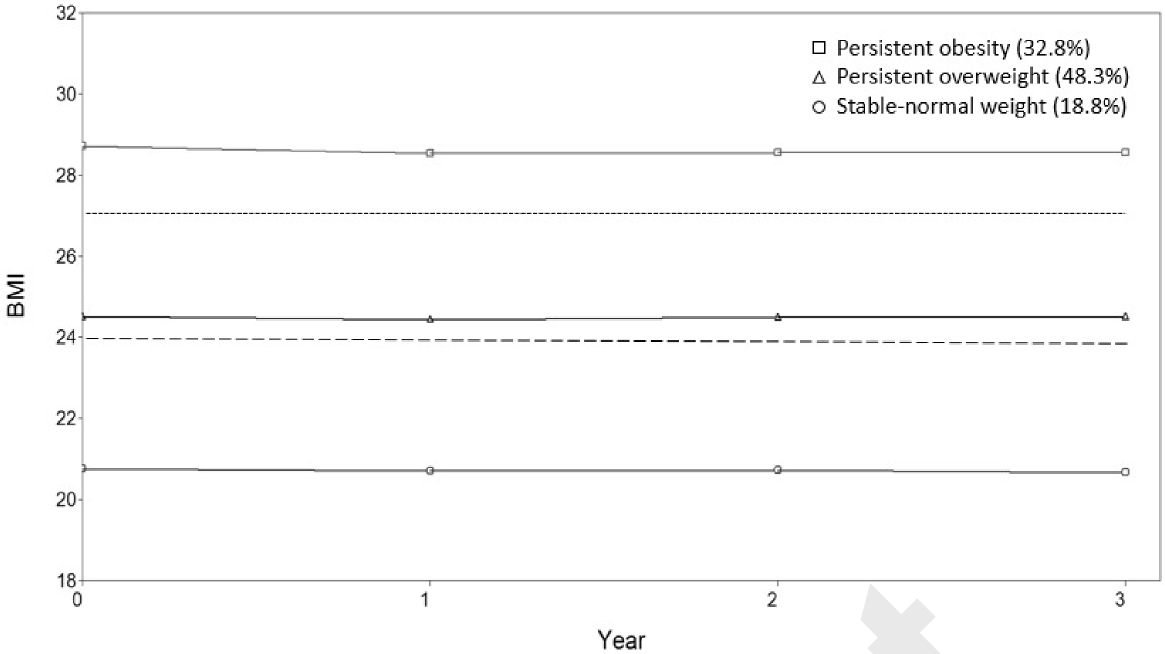


Figure 1. BMI trajectories derived from 478 participants. Each plot depicts the mean BMI at each time point. Groups represent distinct trajectory categories, with the proportion of participants in each group shown in parentheses. A thicker dashed line represents a BMI of 24, while a thinner dashed line represents a BMI of 27.