Long-term weight patterns and risk of sarcopenia in community-dwelling older adults: a three-year cohort study

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In later life, body weight changes over time and may provide clinically relevant information. A single body mass index (BMI) value describes size, yet it cannot reveal whether an older person is preserving physiological reserve, accumulating metabolically harmful adiposity, or losing skeletal muscle as a consequence of malnutrition, inflammation, heart failure, malignancy or other chronic disease. The cohort study published in this issue therefore addresses a clinically pertinent question: does the trajectory of weight convey information about incident sarcopenia that a cross-sectional BMI measurement fails to capture? [1].

A clinically important longitudinal signal

The investigators followed community-dwelling adults aged 65 years and over and characterized four dimensions of obesity dynamics: BMI trajectory, BMI variability, three-year BMI change and cumulative overweight-years. Of 1,091 eligible participants, 478 completed the three-year assessment. Compared with stable normal weight, persistent overweight and persistent obesity were associated with lower odds of incident sarcopenia, whereas a declining BMI was associated with higher odds (adjusted odds ratio 2.70, 95% confidence interval [CI] 1.17–6.23). The apparent advantage of higher BMI was confined to muscle mass: weight-pattern measures were not significantly associated with grip strength or gait speed [1].

Contemporary consensus definitions characterize sarcopenia as a disorder of muscle strength, muscle quantity or quality, and physical performance, rather than of low lean mass alone [2, 3]. A larger body imposes chronic mechanical loading and may therefore carry more absolute lean tissue, while muscle quality, strength relative to body weight, mobility and cardiorespiratory fitness remain impaired. The findings should not, therefore, be reduced to the reassuring but misleading conclusion that obesity protects older adults.

The cardiovascular interpretation

For cardiologists, unintentional weight loss often reflects clinical deterioration rather than treatment success. In heart failure, falling weight may reflect effective decongestion, but it may equally signal anorexia,

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systemic inflammation, intestinal edema with malabsorption, catabolic activation, cachexia or progressive frailty. Pooled data indicate that weight loss in patients with heart failure is independently associated with a 64% higher risk of cardiovascular mortality and a 75% higher risk of all-cause mortality [4]. The present results reinforce the need to interpret weight change in its clinical context and to establish which tissue compartment is being lost. Stable weight can conceal the replacement of muscle by fat, whereas weight reduction may be beneficial when it predominantly reflects loss of excess adiposity and is accompanied by preserved strength and function. In daily heart failure practice, we find it essential to distinguish three very different scenarios: rapid weight reduction due to successful diuresis, deliberate fat loss during obesity treatment, and unintentional weight loss accompanied by declining appetite or functional capacity. These trajectories may look similar on a scale but call for different clinical decisions. In patients receiving diuretics, serial weight measurements should therefore be interpreted together with symptoms, peripheral edema, signs of pulmonary congestion and, when available, objective congestion assessment rather than being treated as a stand-alone marker of nutritional status.

At the same time, the risk of sarcopenic obesity should not be overlooked. It combines excess adiposity with reduced muscle mass or function and is associated with adverse metabolic, functional and survival outcomes [5, 6]. Recent pooled evidence links this phenotype with a higher incidence of cardiovascular disease and, less consistently, with cardiovascular mortality [7]. Function appears to carry particular prognostic weight: among 2,997 Korean adults aged 65 years and over, sarcopenia defined by impaired strength and physical performance independently predicted incident cardiovascular disease (hazard ratio = 1.92, 95% CI: 1.37–2.70), and both new-onset and persistent sarcopenia over 4 years conferred excess cardiovascular risk [8]. Persistent obesity in the current cohort should therefore be regarded as an epidemiological marker associated with lower observed sarcopenia incidence, not as a desirable treatment target.

What the study cannot establish

Several limitations temper causal inference. First, only 478 of 1,091 eligible participants completed the final assessment, and those lost to follow-up had lower baseline grip strength; even when measured baseline characteristics appear broadly comparable, selective attrition of weaker participants may bias the observed associations. Second, BMI cannot distinguish fat mass from muscle mass, nor detect myosteatosis. Bioelectrical impedance analysis is pragmatic, but its estimates depend on hydration status and it is less precise than dual-energy X-ray absorptiometry or imaging-based methods [2]. Third, reverse causation remains plausible: occult disease may drive both weight loss and subsequent sarcopenia. Excluding participants with cancer or underweight mitigates, but does not eliminate, this concern. Finally, the cohort applied Asian BMI thresholds and the AWGS 2019 criteria; transportability to populations assessed using European thresholds and the EWGSOP2 algorithm requires validation [2, 3]. An additional issue for cardiovascular populations is fluid-related measurement error: changes in extracellular water during decongestion may alter both BMI and impedance-derived lean mass without representing true skeletal muscle loss. This is especially important when extrapolating the findings to patients with heart failure.

The component analyses carry a further methodological caveat. Persistent overweight and obesity showed very strong inverse associations with low muscle mass, but not with strength or gait speed. This contrast may partly reflect the mathematical and biological coupling between body size and estimated lean mass. It also illustrates why function should remain the clinical anchor: preserved kilograms of lean tissue do not necessarily equate to preserved force generation, mobility or cardiovascular resilience.

Implications in the incretin era

These observations are particularly timely, as glucagon-like peptide-1 receptor agonists and dual incretin agonists now achieve weight losses previously attainable mainly through bariatric surgery. In a recent systematic review of 35 randomized trials, conducted largely in middle-aged adults, a median of 28% of the weight lost with incretin-based therapy was attributable to muscle-related indices, exceeding prespecified benchmarks in about two thirds of interventions; strikingly, no trial reported objective physical function outcomes [9]. Lean-mass estimates are imperfect surrogates for contractile muscle, yet observational data in older adults with type 2 diabetes have described declines in grip strength and gait speed during semaglutide treatment [10]. These observations should not discourage the use of effective anti-obesity therapy in appropriately selected older adults. Instead, they emphasize the need for more comprehensive definitions of treatment success: reduction of excess fat, improvement in symptoms and cardiometabolic risk, and preservation of muscle strength and physical function. In clinical practice, this argues against automatically reducing or withholding indicated incretin therapy

solely because some lean mass is lost. Instead, the decision should balance cardiometabolic benefits against frailty, nutritional vulnerability and functional trajectory. Particular vigilance is warranted in older patients with HFpEF and obesity, in whom exertional limitation may arise from both cardiovascular dysfunction and impaired skeletal-muscle reserve. The available evidence does not establish that a measured reduction in lean mass necessarily translates into clinically meaningful sarcopenia in every treated patient.

From BMI surveillance to phenotype-guided care

A pragmatic response is to incorporate trajectory and function into routine cardiovascular assessment. Clinicians should document whether weight loss is intentional, quantify its pace, review appetite and dietary intake and, where appropriate, seek evidence of congestion, depression, malignancy, gastrointestinal disease and medication-related adverse effects. Waist circumference or a direct estimate of body composition can complement BMI, while simple measures such as grip strength, chair-rise performance and gait speed identify loss of functional reserve [2, 3]. Preventive management should combine adequate energy and protein intake with progressive resistance exercise, adapted to renal function, cardiovascular stability and the individual's goals [2, 3]. During pharmacological weight loss in older adults, serial functional assessment should accompany serial weighing [10]. Our practical approach is to establish a functional baseline before substantial weight reduction: recent unintentional weight change, congestion status, dietary protein intake, ability to rise from a chair, habitual walking capacity and, where feasible, handgrip strength. At follow-up, we should assess not only kilograms lost but whether the patient can walk farther, climb stairs more easily and maintain strength. A new decline in strength, recurrent falls, reduced food intake or unexpectedly rapid weight loss should trigger reassessment of nutrition, exercise, medication tolerability and possible intercurrent disease. In patients with heart failure, resistance training and nutritional advice should be individualized to clinical stability, renal function and rehabilitation capacity. The bioimpedance results should be interpreted cautiously during fluid shifts.

The most useful message from this study is therefore not that "higher BMI is better", but that "a falling BMI in an older adult deserves an explanation". Weight stability may be reassuring, but only when interpreted alongside adiposity, muscle strength, mobility, symptoms and disease burden. Body weight alone provides only limited information. Its interpretation should also consider weight trajectory, body composition and functional status. A clinically meaningful target is therefore not the lowest attainable BMI, but a healthier body composition with preserved independence. For an older patient, losing fat while maintaining the ability to stand, walk and perform daily activities is a more persuasive marker of success than weight loss alone.

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Conflict of interest

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