



Sarcopenic Obesity in Older Adults with Type 2 Diabetes: Measuring What the BMI Hides

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Abstract

Sarcopenic obesity is the coexistence of excess adiposity with low muscle mass or strength, and in older adults with type 2 diabetes it is common and largely invisible to routine measurement. The problem is structural: body mass index cannot distinguish muscle from fat, and the standard sarcopenia definitions were built for people who are not obese. In a cohort of older men, 60.8 per cent of those with a body mass index of 32 or above had low muscle strength, and the conventional sarcopenia criteria classified none of them as sarcopenic, while a consensus definition designed for obesity identified 9.6 per cent. Those identified had significantly weaker grip, slower gait and longer chair-stand times, with five times the odds of disability in basic daily activities and a raised rate of falls. Definition still determines prevalence: in 90 hospitalised older adults with severe obesity, four permitted combinations of tests produced figures of 23.3, 25.5, 31.1 and 40.0 per cent in the same people. This review sets out what is measurable, what the numbers depend on, and why weight alone conceals the finding.

Keywords: Sarcopenic obesity; Type 2 diabetes; Body composition; Muscle strength; Diagnostic criteria

Introduction

Loss of skeletal muscle mass and function accompanies ageing, and it occurs alongside excess adiposity often enough to have acquired its own name [1,2]. Sarcopenic obesity is defined by international consensus as the coexistence of excess adiposity with low muscle mass or function [1,3].

Type 2 diabetes accelerates the process. Insulin resistance, chronic low-grade inflammation and reduced physical activity all contribute to muscle loss, and muscle is the principal site of insulin-mediated glucose disposal, so the relationship runs in both directions [4,5,6,19]. Muscle strength declines faster with age in type 2 diabetes than without it, and both muscle mass and muscle quality track gait speed [20,21,22].

The clinical difficulty is that the condition is invisible to the measurement most often taken. Body mass index records total mass and cannot distinguish muscle from fat, so a patient losing muscle and gaining fat may show no change at all [7,8,24]. The combination was described as the confluence of two epidemics

well before criteria existed [24,25,26]. This review examines what the available definitions detect, what they miss, and how much of the reported variation is biological rather than definitional.

Eligibility: Studies were included if they applied a defined diagnostic framework to a described population, compared frameworks against one another, or reported functional or metabolic outcomes in those identified. Studies in mixed populations are cited where results for older adults or for diabetes are separable and are identified as such.

Table 1: Principal studies reviewed, with the finding as reported by the original investigators

Material and Methods

Search strategy: PubMed and the archives of the major geriatric medicine, clinical nutrition, endocrinology and obesity journals were searched for studies of sarcopenic obesity. Search terms combined sarcopenic obesity and sarcopenia with type 2 diabetes, older adults, diagnostic criteria, body composition, handgrip strength and prevalence. Reference lists of the consensus statement [1] and clinical overviews [2,9] were screened by hand for primary sources.

Data extraction and handling: Design, population, the diagnostic framework applied and the reported estimate were extracted as published. The framework and the specific tests used are stated alongside every prevalence figure, because the choice of muscle function test and muscle mass index changes the answer substantially in the same patients. This review reports measured findings and makes no dietary, caloric or exercise recommendations. No pooling or re-analysis was performed. Figures 1 to 6 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

What the standard definition misses: A secondary analysis of a prospective cohort of older men compared two frameworks. Among men with a body mass index of 32 kg/m² or above, 60.8 per cent had low muscle strength, yet not one was classified as having confirmed sarcopenia under the conventional European operational definition (Figure 1) [10]. Applying the consensus definition designed for obesity, 9.6 per cent of the cohort met criteria for sarcopenic obesity [10]. The conventional criteria use absolute or height-adjusted muscle mass, which a person with obesity will usually exceed, so the diagnosis becomes structurally unreachable in the group most likely to have functional impairment.

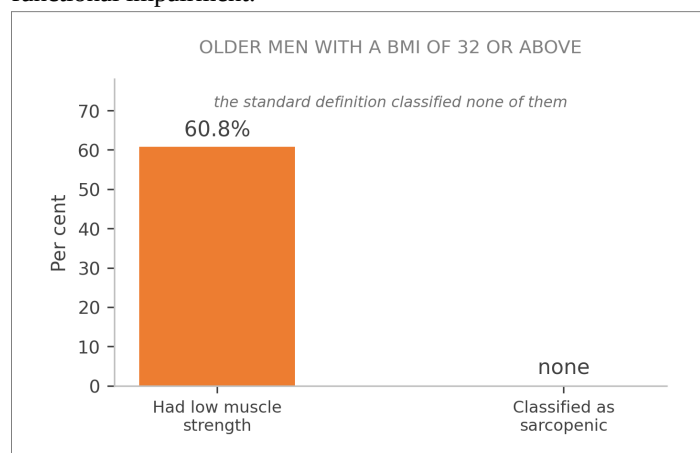


Figure 1: Low muscle strength against formal classification in older men with a body mass index of 32 or above [10]

What identification predicts: Men meeting the consensus definition had significantly lower handgrip strength, longer chair-stand times and slower gait speed, all at $p < 0.05$.

Compared with men who had neither sarcopenia nor obesity, they had an odds ratio of 5.02 (95% CI 1.85 to 13.58) for disability in basic activities of daily living, 2.18 (1.38 to 3.45) for instrumental activities, and an incident rate ratio of 1.59 (1.03 to 2.44) for falls over 12 months (Figure 3) [10].

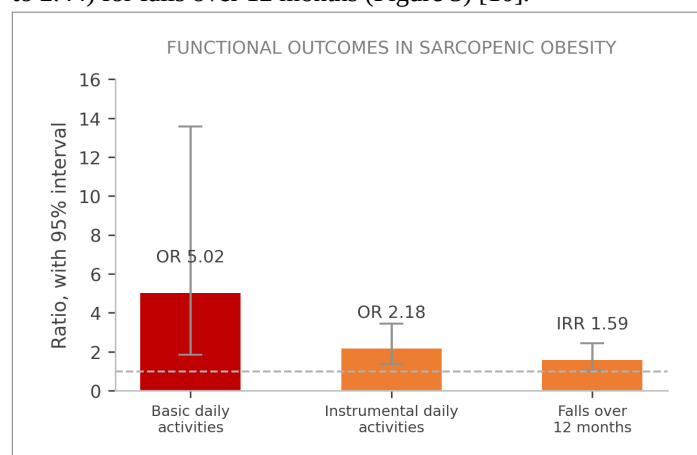


Figure 3: Functional outcomes associated with sarcopenic obesity [10]

The consensus algorithm: The joint expert panel proposed a three-step approach: screening on raised adiposity together with clinical suspicion, diagnosis by low muscle strength and low muscle mass, and severity grading by the presence of complications (Figure 4) [1,3]. Body mass index appears only at the screening step; the diagnosis itself rests on function and composition [1,11]. Cut-off points for the component measures have themselves required separate derivation [31], and the health consequences of sarcopenia in general are well characterised [23,32].

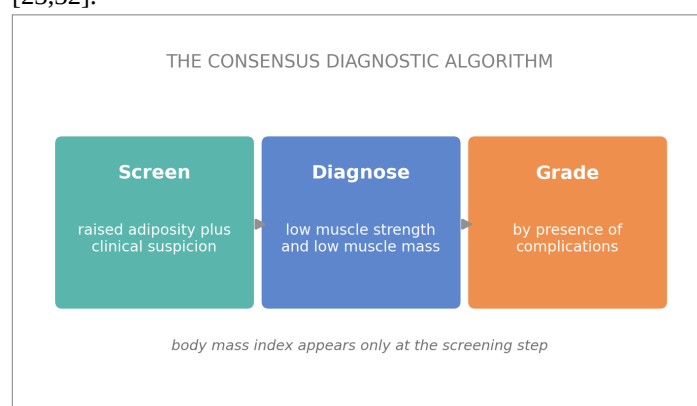


Figure 4: The three-step consensus diagnostic algorithm [1,3]

Applying the algorithm: In 76 free-living older adults with obesity drawn from an Italian primary care study of metabolic syndrome, 61 screened positive and seven of those met the diagnosis, 9 per cent of the whole cohort. No individual who screened negative met the diagnosis (Figure 5) [12]. Those diagnosed had higher insulin resistance and an altered plasma ghrelin profile, and both predicted five-year risk independently of age, sex and body mass index [12].

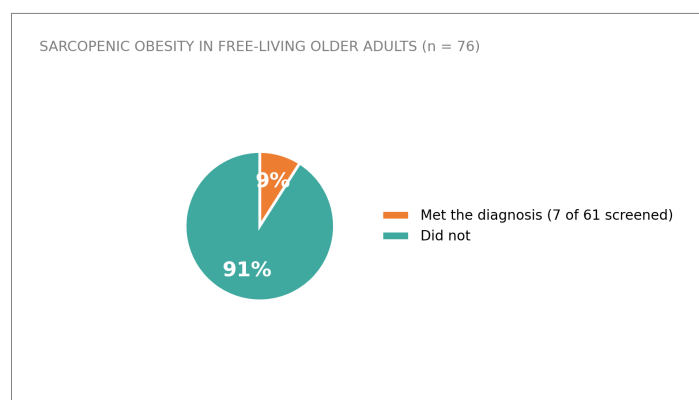


Figure 5: Yield of the consensus algorithm in free-living older adults [12]

Definition still determines prevalence: In 90 hospitalised older adults with severe obesity, prevalence was calculated using four permitted combinations of muscle function test and muscle mass index. The results were 23.3, 25.5, 31.1 and 40.0 per cent in the same patients (Figure 2). Agreement between the two muscle function tests was weak across the whole sample and all subgroups, while agreement between the two muscle mass indices was moderate to strong [13].

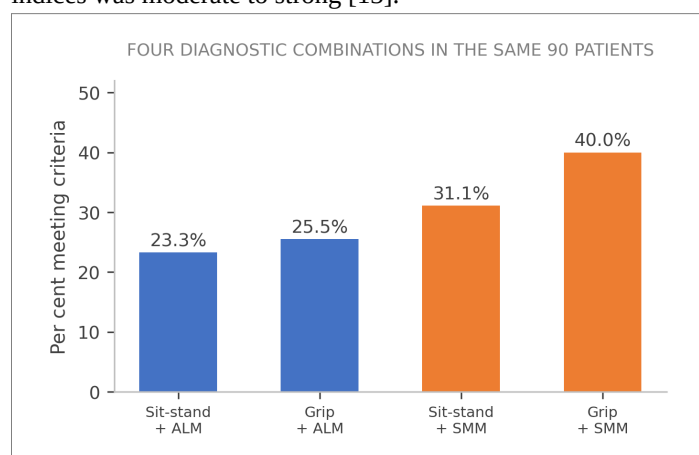


Figure 2: Four permitted diagnostic combinations applied to the same 90 patients [13]

Study	Population and framework	Principal reported finding
Older men cohort [10]	Two frameworks compared	60.8% had low strength; none met conventional criteria
Same cohort [10]	Consensus definition applied	9.6% identified; ADL disability OR 5.02
Italian MoMa cohort [12]	76 free-living adults over 65	9% met the diagnosis; none of the screen-negative did
Hospitalised cohort [13]	90 adults with severe obesity	Prevalence 23.3% to 40.0% by test combination
Diabetes comparison [14,15]	Regional and Asian cohorts	16.4% against approximately 26%
ESPEN and EASO panel [1]	Consensus statement	Three-step screening, diagnosis and grading

In type 2 diabetes specifically: Reported prevalence differs markedly between settings. One regional cohort reported 16.4 per cent, against approximately 26 per cent in broader Asian cohorts (Figure 6) [14,15]. Commentary on that difference attributes it principally to heterogeneity in the diagnostic criteria

used for both sarcopenia and obesity rather than to underlying biology, and notes that using adiposity-based rather than body mass index criteria is the direction the field is moving [14,18]. Population survey analyses report functional impairment in those identified [27,28], and diabetes-specific cohorts have described determinants and associated complications [29,30]. Advancing age and reduced calf circumference were identified as independent risk factors, with handgrip strength protective [14].

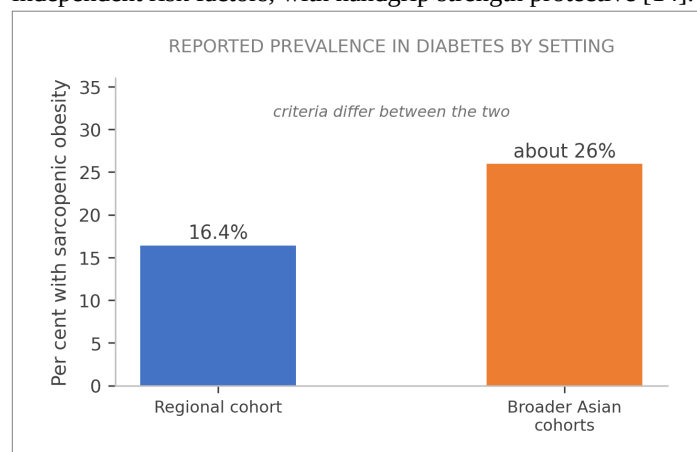


Figure 6: Reported prevalence in type 2 diabetes by setting [14,15]

Component	What is measured	Common tools
Adiposity	Excess fat mass	Body mass index at screening; fat mass on analysis
Muscle function	Strength or performance	Handgrip strength; five-repetition sit-to-stand
Muscle mass	Quantity adjusted to body size	Appendicular lean mass or skeletal muscle mass, by weight
Severity	Presence of complications	Functional limitation and comorbidity

Table 2: Components of the consensus definition and the measurements used for each [1,13]

Framework	How muscle mass is handled	Effect in obesity
Conventional sarcopenia criteria	Absolute or height-adjusted mass	Threshold rarely crossed; cases missed
Consensus sarcopenic obesity criteria	Mass adjusted to body weight	Identifies cases the other framework does not
Body mass index alone	Total mass only	Cannot separate muscle from fat

Table 3: Why the framework chosen determines whether the diagnosis is reachable [1,7,10]

Why muscle matters metabolically: Skeletal muscle is the principal site of insulin-mediated glucose disposal, so losing it has consequences beyond mobility [6,19]. Cohort data show that muscle strength declines faster with age in people with type 2 diabetes than in those without, and that the association between diabetes and slower gait speed is mediated by muscle quality rather than muscle quantity alone [20,21,22]. Meta-analysis reports an increased risk of sarcopenia in type 2 diabetes, and sarcopenic obesity in diabetes has been associated with adverse outcomes in systematic review [15,16,19].

What the diagnosis is associated with: Beyond function, health outcomes associated with sarcopenia have been summarised in meta-analysis and include falls, fractures, hospitalisation and mortality [23,32]. In diabetes cohorts specifically, sarcopenia has been associated with incident albuminuria and with the determinants identified in Korean and other populations [29,30]. Whether these associations reflect a distinct syndrome or the accumulation of two separate risks is not settled by the observational data [2,18].

Discussion

The most instructive result in this literature is a negative one. In older men with a body mass index of 32 or above, three in five had low muscle strength and the conventional sarcopenia criteria identified none of them [10]. That is not a small sample artefact or a threshold that could be nudged; it is a structural consequence of defining low muscle mass in absolute or height-adjusted terms in people who carry more of everything. The diagnosis was unreachable by construction in exactly the group with the functional problem.

The consequence is that a clinician relying on the older framework will conclude that sarcopenia does not occur in their patients with obesity, and will be wrong. The consensus definition addresses this by adjusting muscle mass to body weight and by putting function first, and the group it identifies is not a statistical curiosity: five times the odds of needing help with basic daily activities and a raised falls rate over twelve months [1,10].

Against that, the prevalence figures should be treated with caution rather than quoted. Four permitted combinations of tests applied to the same 90 patients produced 23.3, 25.5, 31.1 and 40.0 per cent, and agreement between the two muscle function tests was weak [13]. A range of that width in one population means published prevalence is as much a statement about method as about people, and the difference between 16.4 and 26 per cent reported in diabetes cohorts is plausibly the same phenomenon rather than a real geographical difference [14,15].

For diabetes practice the specific point is that the routine measurement is the unhelpful one. Weight and body mass index are recorded at every review and neither can detect this condition; a patient can lose muscle and gain fat with no change in either [7,8]. Handgrip dynamometry and a sit-to-stand test take a few minutes, require little equipment and address the functional half of the definition directly [1,13]. The screening step of the consensus algorithm was designed to be usable in general practice and no study reviewed here suggests it is not [1,12].

The metabolic association is worth noting without overstating it. Insulin resistance and an altered ghrelin profile predicted five-year risk independently of age, sex and body mass index in one cohort of 76 people [12]. That is a small study and a hypothesis-generating finding, and the direction of causation

between muscle loss and insulin resistance is not resolved by it. What it does suggest is that in type 2 diabetes the two processes are not simply coincident.

There is a practical asymmetry between the two halves of the definition. Muscle function can be measured in a clinic room in a few minutes with a dynamometer or a chair, and both tests have published cut-offs [1,31]. Muscle mass requires bioelectrical impedance or dual-energy absorptiometry, which most diabetes clinics do not have to hand. Since the consensus algorithm requires both for a formal diagnosis, the accessible half is the one that identifies risk and the inaccessible half is the one that confirms it [1,13].

That suggests a pragmatic reading of the evidence. Measuring grip strength in older patients with type 2 diabetes identifies a group with worse function and higher fall risk regardless of whether a formal diagnosis can be completed [10,23]. Treating low grip strength as a finding worth acting on, rather than as a step towards a label, avoids making the whole assessment contingent on equipment that is not there. What action should follow remains the open question, since the exercise trials in older adults with obesity were not designed around this diagnosis [33,34].

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Study limitations

The limitations belong to the underlying literature. Prevalence estimates are not comparable across studies because the diagnostic framework, the muscle function test and the muscle mass index all vary, and the same patients yield figures differing by a factor of nearly two [1,13]. The principal comparative cohort is restricted to older men, so the findings may not transfer to women, in whom body composition and grip norms differ [10]. The free-living cohort applying the consensus algorithm includes 76 participants, of whom seven met the diagnosis, so its estimates rest on very small numbers [12]. The hospitalised cohort was recruited to a weight reduction programme and is not representative of community populations [13]. Most studies are cross-sectional, so the temporal relationship between muscle loss, adiposity and glycaemic control cannot be established [4,14]. Bioelectrical impedance and dual-energy absorptiometry are not interchangeable for muscle mass. And no trial has yet shown that identifying sarcopenic obesity and acting on it improves function or metabolic outcomes, which is the question that would justify systematic screening [2,9]. Trials of exercise modality in older adults with obesity exist but were not designed around this diagnosis [33,34]. Sarcopenia risk in type 2 diabetes has been quantified in meta-analysis [16,17].

Conclusion

Sarcopenic obesity is common in older adults with type 2 diabetes and is not visible in the measurements taken at a routine review. The conventional sarcopenia criteria are worse than uninformative in this group: applied to older men with a body mass index of 32 or above, they classified none as sarcopenic while three in five had low muscle strength. A consensus framework that adjusts muscle mass to body weight and leads with function identifies a group with five times the odds of disability in basic daily activities. What it does not yet provide is a stable prevalence figure, since four permitted test combinations disagreed by nearly twofold in the same patients. The practical step is smaller than the definitional debate: measuring grip strength costs very little and detects what weight cannot.

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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