



Original article

Sarcopenic obesity prevalence and clinical implications in patients with advanced knee osteoarthritis and class II-III obesity

K. Godziuk ^{a,b,*}, F.T. Vieira ^{a,c}, M. Forhan ^d, J.A. Batsis ^{e,f}, L.M. Donini ^g, M.C. Gonzalez ^{h,i}, M. Siervo ^j, C.M. Prado ^{a, **}

^a Department of Agricultural, Food and Nutritional Science, Faculty of Agricultural, Life and Environmental Sciences, University of Alberta, Edmonton, AB, Canada

^b Department of Physical Therapy and Rehabilitation Science, University of California San Francisco, San Francisco, CA, USA

^c Department of Pediatrics, Faculty of Medicine, University of Alberta, Edmonton, AB, Canada

^d Department of Occupational Science and Occupational Therapy, University of Toronto, Toronto, ON, Canada

^e Division of Geriatric Medicine, School of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

^f Department of Nutrition, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

^g Experimental Medicine Department, Sapienza University, Rome, Italy

^h Postgraduate Program in Nutrition and Food, Pelotas, Federal University of Pelotas, Pelotas, Rio Grande do Sul, Brazil

ⁱ Pennington Biomedical Research Center, Louisiana State University, Baton Rouge, LA, USA

^j School of Population Health, Curtin University, Perth, Australia

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SUMMARY

Objective: Individuals with advanced knee osteoarthritis (OA) and a larger body size are at risk for sarcopenic obesity (SO), an unfavourable condition of high fat and low muscle mass and function that markedly impacts mobility and morbidity. We examined the prevalence and implications of SO in adults with knee OA and a $\text{BMI} \geq 35 \text{ kg/m}^2$, comparing various established diagnostic criteria.

Methods: We conducted a cross-sectional analysis of participants at baseline from the POMELO [Prevention of Muscle Loss in Osteoarthritis] pilot randomized clinical trial. The diagnosis of SO was based on published criteria, identifying the co-presence of low muscle function, low muscle mass, and high fat mass. Assessments included maximal handgrip strength (absolute and relative to body size), chair sit-to-stands, muscle [appendicular lean soft tissue] and fat mass measured by DXA, health-related quality of life by Euroqol EQ-5D, and physical function by 6-min walk (6MWT) and Western Ontario and McMaster Osteoarthritis Index (WOMAC).

Results: Out of 50 adults (74 % female, 63.7 ± 6.9 years, $\text{BMI} 42.1 \pm 4.6 \text{ kg/m}^2$), 28 % had criteria for SO (95%CI 15.5–40.4). Individuals with SO had shorter 6MWT distance, -78.6 m ($p = 0.012$), worse WOMAC function score, 7.2 ($p = 0.046$), and lower EQ-5D visual analog score, -14.7 ($p = 0.016$), compared to those without SO.

Conclusion: SO was present in 28 % (95%CI 15.5–40.4) of our sample with knee OA, with clinically unfavourable implications on measured and self-reported physical function and quality of life. Identification of SO may better stratify patients and enable personalized support to preserve muscle mass and function prior to weight loss or arthroplasty considerations.

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* Corresponding author. Department of Physical Therapy and Rehabilitation Science, University of California San Francisco, USA; Department of Agricultural, Food and Nutritional Science, University of Alberta, Canada.

** Corresponding author. Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Alberta, Canada.

E-mail addresses: kristine.godziuk@ucsf.edu (K. Godziuk), carla.prado@ualberta.ca (C.M. Prado).

Key points

- Older adults with advanced knee osteoarthritis (OA) are at high-risk for development of sarcopenic obesity (SO).
- Using a diagnostic framework from the Sarcopenic Obesity Global Research Initiative (SOGLI), individuals with SO and knee OA had clinically-relevant impairments in physical function and quality of life compared to counterparts without SO.
- SO should be considered before advising unsupervised weight loss in older adults with advanced knee OA.

Individuals with knee osteoarthritis (OA) and obesity are a high-risk population for muscle and strength loss [1,2]. This is due to complex interconnecting factors, including inflammatory repercussions of excess adiposity on joint and muscle tissues, aging-associated attenuation in protein synthesis, and OA-related pain and joint function changes that contribute to physical inactivity and increased risk for disability [3–5]. As a result, individuals with obesity and knee OA are susceptible to develop sarcopenic obesity (SO), an unfavourable condition of high fat and low muscle mass and function that markedly impacts mobility and morbidity. SO is an important clinical condition and prognostic factor for comorbidity, development of disability, and lower survival [6,7]. Past cross-sectional studies have shown that SO can negatively impact mobility, quality of life, and increase surgical and mortality risks [8].

Emerging studies highlight the relevance of SO in knee OA treatment and prevention [9,10]. The global prevalence of SO in older adults is estimated at 11% (95% CI 10–13) [11], yet rates up to 35% have been reported in individuals with knee OA [12]. However, inconsistencies in methodologies for identifying SO pose challenges in advancing our understanding of its role in OA. The now available consensus framework to standardize SO diagnosis [13] provides an opportunity to clarify regarding the prevalence of SO and its significance in knee OA management.

Consideration of SO in OA clinical management is also needed in light of growing interest in precision weight loss for knee OA using pharmacotherapy (i.e. obesity medications) [14]. Weight loss can reduce mechanical joint stress, inflammation, and improve symptom management with obesity and knee OA [15], however some individuals may unknowingly have low muscle mass and low function, or a reduced muscle-to-fat mass ratio indicative of SO [15]. Advising weight loss without accounting for SO may have negative consequences related to muscle health, particularly in older adults. This is an area of debate and uncertainty [16,17].

In view of these concerns, this study examined the prevalence and implications of SO in adults with advanced knee OA and a BMI $\geq 35 \text{ kg/m}^2$. We compared various established diagnostic criteria within the context of the SO consensus framework [13], to provide clinical insights regarding SO identification which could inform weight loss considerations in advanced knee OA.

1. Methods*1.1. Design and participants*

This cross-sectional study analyzed data from individuals enrolled at baseline in the POMELO (Prevention Of Muscle Loss in Osteoarthritis) parallel-arm randomized controlled pilot feasibility trial. The sample size of $n = 50$ was calculated based on the main trial outcomes. The protocol for POMELO was registered on clinicaltrials.gov (NCT05026385) and published [18]. Adults aged 40–75 years with a BMI $\geq 35 \text{ kg/m}^2$ and physician confirmed advanced knee OA [Kellgren–Lawrence grade 3–4 with clinical symptoms] were included. Individuals with a BMI $\geq 35 \text{ kg/m}^2$ were exclusively enrolled in POMELO to address evidence gaps regarding class II–III obesity and knee OA management [18]. Ethics approval was received in March 2021 and participant enrollment was conducted at a single center between September 21, 2021 and October 4, 2022. All study participants provided written informed consent. Reporting was guided by the STROBE checklist for observational studies [19].

1) Screen for obesity (High body mass index and waist circumference)

If present, then proceed with diagnosis process:

2) Assess for low muscle function

Low handgrip strength (HGS)



Low chair sit-to-stands in 30-seconds (CSTS)

If present →

3) Assess for low muscle mass and high fat mass

Low appendicular lean soft tissue (ALST) and high fat mass (FM) assessed by dual-energy x-ray absorptiometry (DXA)

4) Sarcopenic obesity = low muscle function + low muscle mass + high fat mass

Fig. 1. Algorithm used for the diagnosis of sarcopenic obesity in individuals with advanced knee osteoarthritis, following Sarcopenic Obesity Global Leadership Initiative (SOGLI) consensus recommendations [24,25].

Images adapted from Prado et al. Clin Nutrition, 2022 [49].

1.2. Anthropometrics and body composition

All assessment procedures are outlined in the protocol paper [18]. Briefly, anthropometrics included height and weight, and BMI was calculated. The average of three measures of waist circumference (top of the iliac crest over light clothing) and calf circumference (widest point of the gastrocnemius in standing position over skin) were assessed. Whole body composition was determined using dual-energy x-ray absorptiometry (DXA) using a GE Healthcare Lunar iDXA, ENCORE software version 18. Total body and regional lean soft tissue (LST), fat mass (FM), and bone mineral content were assessed; percent FM was calculated. Appendicular lean soft tissue (ALST), a surrogate of muscle mass, was calculated as LST of arms plus legs.

Obesity was screened for study inclusion using BMI cut-points from World Health Organization (i.e. class II obesity, $BMI \geq 35 \text{ kg/m}^2$). High waist circumference was confirmed using cut-points above thresholds associated with optimal health outcomes [$>101.2 \text{ cm}$ in females (F), $>103 \text{ cm}$ in males (M)] [20]. Study participants self-reported the presence of other physician-diagnosed chronic health conditions.

1.3. Strength, physical function, quality of life and pain

Strength and physical function assessments included maximal handgrip strength (HGS), 30-s chair sit-to-stand test (CSTS), and the six-minute walk test (6MWT) [18]. HGS was recorded as highest grip strength scored after three attempts in each hand measured with a hydraulic Jamar® dynamometer. HGS was considered by absolute value and adjusted by BMI to reflect relative strength. CSTS was number of complete repetitions moving from seated to full stand in 30-s. 6MWT was measured as meters walked in a 6-min timed interval on an indoor course. Self-reported health-related quality of life, pain, and function were also assessed. Patients rated their perceived quality of life on the Euroqol Foundation EQ-5D-5L (EQ-5D) [21] from 1 'no problems' to 5 'extreme problems' across five dimensions of health: mobility, self-care, usual activities, pain/discomfort, and anxiety/depression. Results were dichotomized into no/mild problems (score of 1–2), and moderate/severe problems (scores of 3–5). Perceived overall health was rated on a visual analog scale (VAS) from 0 mm (worst health) to 100 mm (best health). OA-specific pain and function were assessed with the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) [22], scored on a 5-point Likert scale for pain (0–20; 5 items each scored 0–4), stiffness (0–8; 2 items each scored 0–4) and function (0–68; 17 items each scored 0–4), for a total of 0–96, normalized to 0–100 by multiplying total score by 100/96. Higher scores on WOMAC indicate worse status.

1.4. Sarcopenic obesity diagnosis

Sarcopenic obesity was assessed using Sarcopenic Obesity Global Research Initiative (SOGLI) consensus criteria [13,23]. This involved first screening for high BMI and clinical suspicion, then identifying low muscle function, and if present, completing body composition assessment to identify low muscle and high fat mass (summarized in Fig. 1). Low muscle function was assessed using thresholds for low absolute HGS [$<20 \text{ kg F, } <30 \text{ kg M}^{24}$] and low CSTS [$<15 \text{ in F, } <16 \text{ in M}^{23,25}$]. If low muscle function was present, low muscle mass was then identified using sex-and age-specific thresholds for low ALST/weight based on mixed-ethnicity populations [$<19.4\% \text{ F, } <25.7\% \text{ M age } \geq 65 \text{ years [26]; } <23.47\% \text{ F, } <28.27\% \text{ M age } <65 \text{ years [27]}$]. High percent fat mass (FM) was identified as $>40\% \text{ F, } >30\% \text{ M [28]}$ for white, or $>41\% \text{ F, } >29\% \text{ M in}$

black or asian racial groups [29]. Low muscle function and muscle mass were also considered using relative HGS [(HGS/BMI), below OA-specific cut-points $<0.65 \text{ kg/kg/m}^2$ in F, $<1.1 \text{ kg/kg/m}^2$ in M³⁰, or cut-points from the Sarcopenia Definitions and Outcomes Consortium (SDOC) $<0.79 \text{ kg/kg/m}^2$ in F, $<1.05 \text{ kg/kg/m}^2$ in M [31,32]], and low ALST/BMI [$<0.512 \text{ kg/kg/m}^2$ in F, $<0.789 \text{ kg/kg/m}^2$ in M³¹]. Notably these were not recommended diagnostic approaches from the SOGLI group due to a lack of robust evidence, however we wanted to explore in our dataset, in line with recommendations for further research on the topic [13]. Low calf circumference was also evaluated as a practical marker of low muscle mass [34]. Adjustments to calf circumference were applied to account for excess subcutaneous fat mass in the calf region with a higher BMI [BMI 30–39 kg/m^2 : -7.0 cm ; BMI $\geq 40 \text{ kg/m}^2$: -12.0 cm], and then applying cut-offs for low calf circumference [$<31 \text{ cm F, } <32 \text{ cm M}^{34}$].

1.5. Statistical methods

Analyses were completed using IBM SPSS Statistics v29 (IBM Corp., Armonk, NY). Data distribution normality was assessed using Shapiro Wilk test. Descriptive analyses are reported as mean (standard deviation) and frequency (proportion). Student's independent t-test was used for between-group comparisons of normally distributed continuous outcomes, and Mann Whitney U test was used when distribution of the outcome variable was not parametric. Chi Square or Fisher's exact test were used for between-group comparisons of categorical variables, depending on sample numbers being compared. All testing was two-tailed and p-values <0.05 were considered significant. Sensitivity analyses were conducted to examine if OA-related strength, physical function, quality of life and pain outcomes in individuals with and without SO were similar using alternative diagnostic criteria.

2. Results

Complete baseline data from fifty adults ($n = 50$) were included in this cross-sectional analysis, 74 % female, mean age 63.7 ± 6.9 years (range 48–75), $BMI 42.1 \pm 4.6 \text{ kg/m}^2$ (range 35–52.4). Racial background was self-reported as 90 % White, 6 % Indigenous, 2 % Black, and 2 % South Asian. Bilateral knee OA was present in 78 % of the sample.

Figure 2 and Table 1 present the prevalence of low muscle function, low muscle mass, high fat mass, and sarcopenic obesity (low muscle function + low muscle mass + high fat mass) in this cohort using published assessment criteria. Only 2 % had low absolute HGS (1 female), whereas 98 % had low CSTS (only 1 male was above threshold). Low HGS/BMI was present in 17 F (46 %) and 5 M (38 %). All individuals had high measured %FM. Low ALST/weight alone was present in 60 % of the sample, including 21 females (57 %) and 9 males (69 %). Following SOGLI guideline criteria to identify SO, prevalence ranged from 0 % (no individuals were identified with original criteria of low HGS + low ALST/weight + high FM) to 60 % (all individuals had low CSTS and high FM, and 60 % had low ALST/weight). When SOGLI criteria were adjusted to consider relative rather than absolute low HGS (low HGS/BMI + low ALST/weight + high FM), SO was diagnosed in 10 F and 4 M, with an overall SO prevalence of 28 % (95%CI 15.5–40.4).

Clinical outcomes between individuals with and without SO were compared across various diagnostic criteria, reported in Table 2. Comparisons using adjusted SOGLI criteria (i.e. low HGS/BMI + low ALST/weight + high FM) identified differences between individuals with and without SO (Table 2, and Fig. 3). Compared to those without SO (non-SO), individuals with SO had a higher BMI (5.0 kg/m^2 , $p < 0.001$), walked less distance in the 6MWT

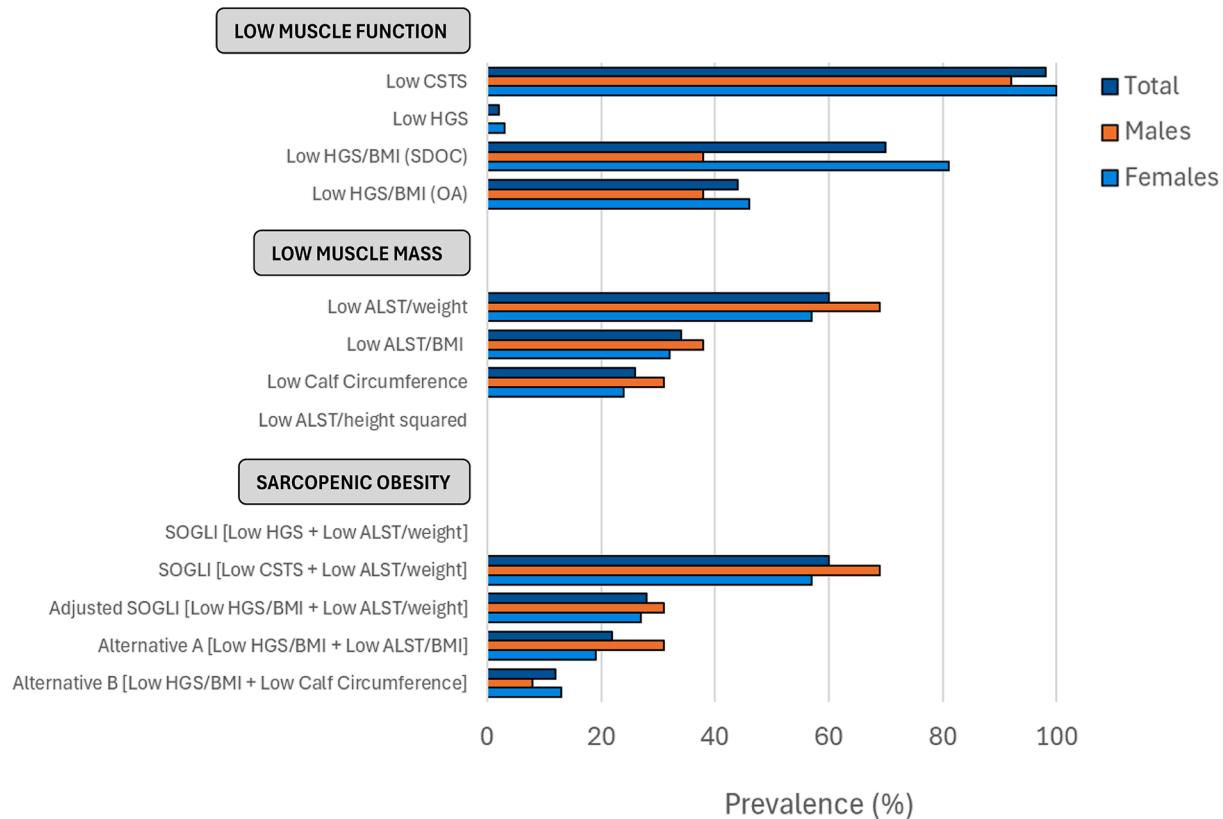


Fig. 2. Graphic summary of prevalence (reported as % of sample meeting criteria) of low muscle function, low muscle mass, and sarcopenic obesity (low muscle function + low muscle mass + high fat mass) in n = 50 adults with advanced knee osteoarthritis and a BMI ≥ 35 kg/m 2 , using different published criterion (cutpoints detailed in Table 1) ALST = appendicular lean soft tissue, BMI = body mass index, CSTS = chair sit-to-stands in 30-s, HGS = maximal handgrip strength, OA = criteria from osteoarthritis cohort, SDOC = criteria from the Sarcopenia Definitions and Outcomes Consortium, SOGLI = criteria from the Sarcopenic Obesity Global Leadership Initiative.

(-78.6 m, p = 0.012), and had worse self-reported physical function [higher WOMAC function score, 7.2, p = 0.046] (Table 2). Overall perception of health status was poorer in the SO versus non-SO groups based on lower EQ-5D VAS scores (-14.7, p = 0.016). There were also higher rates of moderate-severe problems in the EQ-5D anxiety/depression dimension in the SO group compared to non-SO (31 % higher prevalence, p = 0.042). Similar differences in physical function were found between SO and non-SO groups identified using the alternative SO definition of low HGS/BMI + low ALST/BMI + high FM, but not the unadjusted SOGLI criteria of low CSTS + low ALST/weight + high FM (Table 2).

3. Discussion

Appling adjusted SOGLI criteria, sarcopenic obesity (SO) was identified in 28 % of individuals with advanced knee OA and a BMI ≥ 35 kg/m 2 . This substantive SO prevalence highlights that individuals with a BMI ≥ 35 kg/m 2 are not a uniform group and have considerable heterogeneity. Body composition assessment is needed to distinguish phenotypes within this BMI category. Recognizing hidden conditions like SO earlier in OA management and prior to arthroplasty is pivotal, as they can influence surgical outcomes, mobility, and overall health. This is the first study to consider SOGLI consensus criteria to identify SO in OA patients, and compare different approaches to identify low muscle function and mass in this clinical population. Notably, our data show the importance of adjusting diagnostic criteria for low muscle function to consider relative strength (e.g. HGS/BMI), which may be relevant for other SO clinical conditions.

Individuals identified with SO had clinically relevant deficiencies in physical function and health-related quality of life compared to individuals with non-SO. These observations in individuals with both SO and OA are of particular concern. Under-diagnosed SO could influence various facets of OA management, spanning both surgical and non-surgical approaches. Functional impairments and diminished quality of life are key patient symptomatic indicators guiding decision pathways on OA treatment approaches, including whether to proceed to joint replacement surgery. It is plausible that functional limitations related to SO may affect efficacy of exercise interventions aimed at managing knee OA [35]. Moreover, studies also suggest that SO may affect TKA outcomes, including complications, recovery time, and reoperation rates [36,37]. It is crucial to expand SO assessment among patients with high BMI and OA in both research and practice to better understand the impact on OA treatment outcomes.

There is potential that observed differences in function in the SO group may be influenced by the higher BMI noted in comparison to counterparts without SO. Mobility tests such as the 6MWT may be influenced by body size or degree of obesity [38]. Notably, DXA measures of ALST/weight or ALST/BMI may over-identify low muscle mass in those with the highest weight or BMI due to the reciprocal relationship between FM and LST in a three-compartment body composition model [39] (i.e. individuals with the highest %FM would reciprocally have lower %LST). Nonetheless, low relative muscle mass may be as important as low absolute muscle mass in the context of SO [13]. Identification of relative low muscle in those with greater obesity may be appropriate if it enables identification of clinically meaningful health impairments (i.e. function, quality of life, disability, mortality, or

Table 1

Cutpoints and prevalence of low muscle function, low muscle mass, high fat mass, and sarcopenic obesity (low muscle function + low muscle mass + high fat mass) identified in n = 50 individuals with knee osteoarthritis and a BMI $\geq 35 \text{ kg/m}^2$.

	Total n = 50	Females n = 37	Males n = 13
Low muscle function			
Low absolute HGS	1 (2)	1 (3)	0 (0)
<20 kg F, <30 kg M			
Low CSTS	49 (98)	37 (100)	12 (92)
<15 F, <16 M			
Low HGS/BMI (SDOC)	35 (70)	30 (81)	5 (38)
<0.79 kg/kg/m ² F, <1.05 kg/kg/m ² M			
Low HGS/BMI ^a	22 (44)	17 (46)	5 (38)
<0.65 kg/kg/m ² F, <1.1 kg/kg/m ² M			
Low muscle mass			
Low ALST/weight	30 (60)	21 (57)	9 (69)
<19.4 % F, <25.7 % M in age ≥ 65 years			
<23.47 % F, <28.27 % M in age <65 years			
Low ALST/BMI	17 (34)	12 (32)	5 (38)
<0.512 kg/kg/m ² F, <0.789 kg/kg/m ² M			
Low ALST/height ²	0	0	0
<5.45 kg/m ² F, <7.26 kg/m ² M			
Low BMI-adjusted Calf Circumference ^b	13 (26)	9 (24)	4 (31)
<31 cm F, <32 cm M			
High fat mass			
High %FM	50 (100)	37 (100)	13 (100)
>40 % F, >30 % M if white			
>41 % F, >29 % M if black or asian			
Low muscle function + low muscle mass + high fat mass (sarcopenic obesity)			
Low HGS + low ALST/weight + high %FM (SOGLI)	0 (0)	0 (0)	0 (0)
Low CSTS + low ALST/weight + high %FM (SOGLI)	30 (60)	21 (57)	9 (69)
Low HGS/BMI ^a + low ALST/weight + high %FM (Adjusted SOGLI)	14 (28)	10 (27)	4 (31)
Low HGS/BMI ^a + low ALST/BMI + high %FM (Alternative A)	11 (22)	7 (19)	4 (31)
Low HGS/BMI ^a + low calf circumference + high %FM (Alternative B)	6 (12)	5 (13)	1 (8)

Data presented as n (%).

ALST = appendicular lean soft tissue, BMI = body mass index, CI = confidence interval, CSTS = chair sit to stands in 30-s, F = female, FM = fat mass, HGS = maximal handgrip strength, M = male, OA-specific = criteria from osteoarthritis cohort [32], SD = standard deviation, SDOC = criteria from the Sarcopenia Definitions and Outcomes Consortium [33,34], SOGLI = criteria from the Sarcopenic Obesity Global Leadership Initiative [24,25].

^a OA-specific criteria [32].

^b Calf circumference adjusted for BMI; BMI 30–39 kg/m²: -7.0 cm; BMI >40 kg/m²: -12.0 cm.

institutionalization) [40] that can be influenced by appropriate treatment recommendations (i.e. interventions that support retention of muscle mass while reducing FM and body weight to improve this relative distribution). Additional research in this area is needed.

The compromised function in those with SO raises concerns about persistent mobility deficiencies after arthroplasty. This may influence patient outcomes and their post-operative satisfaction, but to our knowledge there has been no examination of the influence of SO in this regard. Studies suggest that those with SO experience poorer range of motion [41] and a 2x higher hazard ratio for persistent walking disability after TKA compared to those with obesity or sarcopenia alone [42]. Research on patients with sarcopenia underscore its negative impact on long-term TKA outcomes [43], suggesting a potential significance for SO. Further investigation in this area should be a priority.

The higher rates of moderate-to-severe issues in the anxiety/depression dimension of the EQ-5D observed in our SO group might reflect the combined risk of depression associated with both obesity and low muscle mass and function [44]. Poor mobility has been independently associated with risk for anxiety and depression [44], while depression has been associated with decreased strength and functional capacity in individuals with obesity [45]. This suggests a complex causality that is difficult to decipher. Nevertheless, since depression and anxiety can influence self-efficacy and chronic disease self-management, they could be prioritized for treatment alongside nutrition and exercise strategies in SO management.

The current study findings align with our previous work [10], indicating a consistently high prevalence of SO in patients with knee OA. In that research, we identified SO using similar criteria of low ALST (adjusted by weight or BMI) in conjunction with low function. With efforts to establish consensus methods for SO diagnosis, such as the SOGLI criteria [13,23], we anticipate more consistency in prevalence reporting as research in this area advances. Importantly, specific OA-related guidelines for SO identification may be needed as some accepted tests are not suitable in this patient group. Using low CSTS was not an effective approach to assess for low function among our patients with knee OA, as 98 % fell below the set thresholds. This discrepancy is likely due to the influence of knee OA on this functional movement pattern. Further, absolute HGS cut-points were poor discriminators of low muscle function, with only 1 individual scoring below set thresholds [30]. Specific cut points to discern low CSTS and HGS in OA populations, or alternative functional tests, may still be necessary to improve the specificity of identifying SO-related low muscle function. Low HGS adjusted by BMI was more sensitive than absolute low HGS in this clinical population with OA and obesity, and may better reflect relative low muscle strength. While we were able to compare varied assessment approaches for low muscle (ALST relative to weight and BMI, and calf circumference), the SOGLI consensus recommended ALST/weight criteria [13] identified the largest number of individuals. Further examination in larger studies enabling statistical comparisons by subgroups is required to define which adjustments, cut-points, or assessments correlate with clinically relevant impairments in specific age

Table 2

Comparison of osteoarthritis clinical outcomes of pain, physical function, and quality of life between patients with and without sarcopenic obesity by various diagnostic criteria.

	SOGLI Criteria ^a			Adjusted SOGLI Criteria ^b			Alternative A Criteria ^c		
	Sarcopenic Obesity (SO) n = 30	Obesity (Non-SO) n = 20	Mean difference (95 % CI) [difference in proportion, %]	Sarcopenic Obesity (SO) n = 14	Obesity (Non-SO) n = 36	Mean difference (95 % CI) [difference in proportion, %]	Sarcopenic Obesity (SO) n = 11	Obesity (Non-SO) n = 39	Mean difference (95 % CI) [difference in proportion, %]
Age, years	60.3 (6.7)	68.8 (3.0)	−8.5 (-11.7, -5.3)^d	61.8 (6.8)	64.5 (6.9)	−2.7 (−7.0, 1.7)	66.4 (6.3)	63.0 (6.9)	3.4 (−1.2, 8.1)
BMI, kg/m ²	43.1 (4.9)	40.6 (3.7)	2.5 (−0.1, 5.1)	45.7 (4.8)	40.7 (3.7)	5.0 (2.4, 7.5)^d	45.2 (4.6)	41.2 (4.3)	4.0 (1.0, 7.0)^d
Objectively assessed physical function:									
Six-minute walk test (6MWT), meters	367.7 (104.7)	366.8 (97.3)	0.9 (−58.2, 60.0)	310.7 (78.1)	389.3 (100.9)	−78.6 (-138.9, -18.2)^d	280.7 (66.4)	391.8 (95.7)	−111.1 (-173.1, -49.0)^d
Patient-reported pain, physical function, and quality of life:									
WOMAC Pain, 0-20	10.6 (3.2)	10.4 (3.8)	0.2 (−1.8, 2.2)	11.1 (3.2)	10.3 (3.5)	0.8 (−1.4, 2.9)	12.1 (2.8)	10.1 (3.5)	2.0 (-0.3, 4.3)^d
WOMAC Stiffness, 0-8	4.5 (1.3)	4.6 (1.6)	−0.1 (−1.0, 0.7)	4.6 (1.4)	4.5 (1.4)	0.1 (−0.8, 1.0)	5.1 (1.2)	4.4 (1.4)	0.7 (−0.3, 1.6)
WOMAC Function, 0-68	34.6 (9.4)	32.8 (14.2)	1.7 (−5.0, 8.5)	39.1 (6.8)	31.9 (12.3)	7.2 (0.1, 14.2)^d	38.2 (10.2)	32.7 (11.5)	5.5 (−2.3, 13.3)
WOMAC Total, 0-100	51.8 (13.5)	49.9 (19.0)	1.9 (−7.3, 11.1)	57.1 (10.4)	48.7 (16.9)	8.4 (−1.4, 18.2) ^e	57.7 (13.3)	49.1 (16.0)	8.5 (-2.1, 19.2)^d
EQ-5D visual analog scale (VAS), 0-100 mm	50.1 (18.2)	60.4 (20.5)	−10.3 (−21.4, 0.8)	43.6 (16.1)	58.3 (19.5)	−14.7 (-26.5, -2.8)^d	50.0 (17.6)	55.4 (20.2)	−5.4 (−18.9, 8.1)
EQ-5D, moderate to severe problems, n (%)									
Mobility	30 (100)	18 (90)	[10]	14 (100)	34 (94)	[6]	11 (100)	37 (95)	[5]
Self-care	5 (17)	2 (10)	[7]	2 (14)	5 (14)	[0]	1 (9)	6 (15)	[−6]
Usual activities	23 (77)	14 (70)	[7]	11 (79)	26 (72)	[6]	9 (82)	28 (72)	[10]
Pain/discomfort	29 (97)	18 (90)	[7]	14 (100)	33 (92)	[8]	10 (91)	37 (95)	[−4]
Anxiety/depression	11 (37)	3 (15)	[22]	7 (50)	7 (19)	[31]^d	4 (36)	10 (26)	[10]

Data presented as mean (standard deviation) unless otherwise indicated.

Between-group comparisons were conducted with Student's t-test for continuous outcomes, and Fishers Exact for categorical outcomes, unless otherwise indicated.

Alternative B Criteria (low HGS/BMI + low calf circumference + high %FM) had small n preventing comprehensive comparisons.

BMI = body mass index, COPD = chronic obstructive pulmonary disease, CSTS = chair sit-to-stands, EQ-5D = EuroQol 5 Dimension quality of life, FM = fat mass, HGS = maximal handgrip strength, Non-SO = no sarcopenic obesity, SO = sarcopenic obesity, SOGLI = Sarcopenic Obesity Global Leadership Initiative, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index.

^a SO defined by **unadjusted SOGLI criteria** = low CSTS + low ALST/weight + high %FM [No individuals were identified with SO based on unadjusted SOGLI criteria considering low absolute HGS].^b SO defined by **adjusted SOGLI criteria** = low relative HGS (HGS/BMI) + low ALST/weight + high %FM.^c SO defined by **alternative A criteria** = low relative HGS (HGS/BMI) + low ALST/BMI + high %FM.^d Significant difference between groups (p=<0.05).^e Trending towards significance based on p > 0.05 < 1.0.

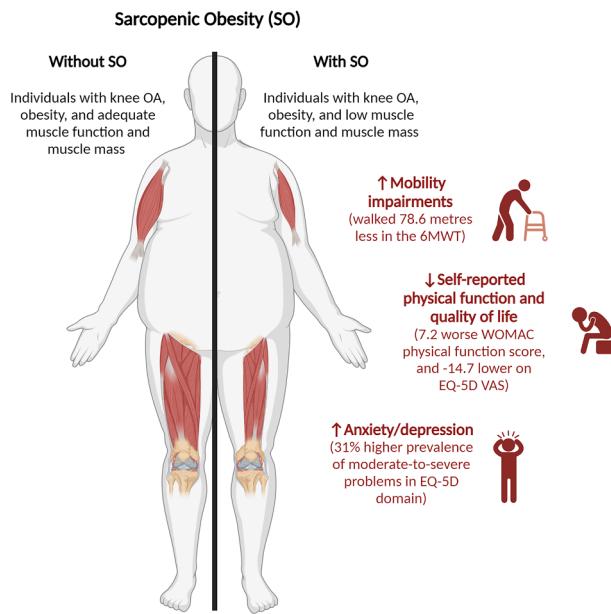


Fig. 3. Representation of impairments in osteoarthritis-related mobility and health outcomes in individuals with sarcopenic obesity[†] compared to counterparts without this condition.

[†] defined by Adjusted SOGLI criteria (low HGS/BMI + low ALST/weight + high % fat mass)

6MWT = 6-min walk test, EQ-5D = Euroqol 5-Dimension health-related quality of life, SOGLI = Sarcopenic Obesity Global Leadership Initiative, VAS = visual analog scale, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index
Image created with Biorender

groups, sex, racial backgrounds, and/or chronic health conditions [13,23]. Clarity on assessment methods will improve the precision of low function and low muscle identification for SO in OA patients.

Our study design was cross-sectional, which does not allow us to infer causality. SO can be a risk factor for worsening physical function, pain, and quality of life in knee OA. Longitudinal data suggest that SO may influence knee OA incidence [46], and trajectories of worse functioning [47]. Conversely, the presence of poor physical function, pain, and inactivity with knee OA may amplify the risk for muscle and strength loss leading to SO [48]. Both causal pathways are likely relevant, with overlapping and potentially cyclic biomechanical, metabolic, and inflammatory influences [12]. Metabolic and inflammatory-related muscle impairments associated with concurrent chronic diseases, including OA and type II diabetes, may also play a role. Research on SO in OA is still emerging, and our understanding will improve with further mechanistic studies and a consistent approach in SO diagnosis [13].

A barrier to increased implementation of SO diagnosis in research and clinical practice is related to limitations of access and cost of body composition assessment. Increased attention towards the relevance of SO in OA and implications on health care system outcomes and cost-effectiveness may be necessary to improve accessibility and use of body composition diagnostic tools. Clinicians and researchers should consider following the SOGLI framework [13] to screen and identify SO in individuals with OA prior to recommending weight loss. This information would indicate a need for more tailored support, including effective treatment approaches to increase muscle mass and strength while reducing fat mass [49,50]. SO treatment and resolution may need to be prioritized over absolute weight loss targets in OA management.

A strength of this study is the targeted sample of patients with a $\text{BMI} \geq 35 \text{ kg/m}^2$ and advanced knee OA, a group that has not been well-represented in OA clinical research [51]. Limitations include the cross-sectional study design and recruitment from a single clinical site, which may impact the generalizability of findings. Only a small number of males were enrolled, which did not allow for sex-based comparisons of clinical outcomes by SO status. Sex differences in body fat, muscle mass, muscle fiber types, and susceptibility to disuse or inflammation-related muscle atrophy may influence risk for SO onset and progression, and clinical repercussions [13]. We did not assess muscle composition (i.e. myosteatosis) [52]. Excess intra-and inter-muscular fat, and changes in muscle fiber number or type may be crucial in understanding SO. Further considerations are needed to determine whether these factors can be effectively assessed and monitored in the clinical setting for patients with knee OA. We may have been statistically underpowered to compare differences between groups using varied SO diagnostic approaches, therefore our findings provide preliminary evidence to guide future comparative investigations. Additional limitations include the use of indirect methods to assess muscle mass (i.e. DXA and calf circumference). Consequently, other measurement approaches or thresholds for low muscle may provide different results [53]. Nonetheless, DXA is an accepted method for SO identification [53].

4. Conclusion

Prevalence of SO was 28 % (95%CI 15.5–40.4) in individuals with advanced knee OA and a $\text{BMI} \geq 35 \text{ kg/m}^2$, adversely affecting mobility and quality of life. SO is an unrecognized but relevant condition in OA. Individuals with SO and advanced knee OA may benefit from tailored support to maintain or improve muscle mass and function, particularly ahead of surgery or presurgical weight loss decisions.

Authors' contributions

KG, MF, and CMP were involved in the study design. KG and FTV contributed to data collection. KG led analyses. All authors contributed to interpretation and writing of the manuscript and have read and approved the final version.

Data availability statement

The data can be made available on request pending written application and approval.

Ethics approval

Provided by the Health Research Ethics Board at the University of Alberta, Edmonton, Alberta, Pro00107201, on March 31, 2021.

Impact statement

We certify that this work is novel and contributes important new knowledge about diagnostic criteria for sarcopenic obesity in older adults with concurrent knee osteoarthritis and a higher body mass index.

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Conflict of interest

All authors declare no competing interests that would create a conflict of interest in connection with this manuscript. KG, FTV, and MF have no disclosures. CMP has received honoraria and/or paid consultancy from Abbott Nutrition, Nutricia, Nestle Health Science, Pfizer, and AMRA Medical, and funding from Almased for research not related to this study.

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References

- [1] Shorter E, Sannicandro AJ, Poulet B, Goljanek-whysall K. Skeletal muscle wasting and its relationship with osteoarthritis: a mini-review of mechanisms and current interventions. *Curr Rheumatol Rep* 2019;21(40).
- [2] Papalia R, Zampogna B, Torre G, Lanotte A, Vasta S, Albo E, et al. Sarcopenia and its relationship with osteoarthritis: risk factor or direct consequence? *Musculoskelet Surg* 2014;98(1):9–14. <https://doi.org/10.1007/s12306-014-0311-6>.
- [3] Gross DC, Cheever CR, Batsis JA. Understanding the development of sarcopenic obesity. *Expt Rev Endocrinol Metabol* 2023;18(6):469–88. <https://doi.org/10.1080/17446651.2023.2267672>.
- [4] Thijssen E, van Caam A, van der Kraan PM. Obesity and osteoarthritis, more than just wear and tear: pivotal roles for inflamed adipose tissue and dyslipidaemia in obesity-induced osteoarthritis. *Rheumatology* 2015;54(4):588–600. <https://doi.org/10.1093/rheumatology/keu464>.
- [5] Dondero K, Friedman B, Rekant J, Landers-Ramos R, Addison O. The effects of myosteatosis on skeletal muscle function in older adults. *Phys Rep* 2024;12(9). <https://doi.org/10.1481/phy2.16042>.
- [6] Axelrod CL, Dantas WS, Kirwan JP. Sarcopenic obesity: emerging mechanisms and therapeutic potential. *Metabolism* 2023;146:155639. <https://doi.org/10.1016/j.metabol.2023.155639>.
- [7] Prado CM, Batsis JA, Donini LM, Gonzalez MC, Siervo M. Sarcopenic obesity in older adults: a clinical overview. *Nat Rev Endocrinol* 2024;20:261–77. <https://doi.org/10.1038/s41574-023-00943-z>.
- [8] Zhang X, Xie X, Dou Q, Liu C, Zhang W, Yang Y, et al. Association of sarcopenic obesity with the risk of all-cause mortality among adults over a broad range of different settings: a updated meta-analysis. *BMC Geriatr* 2019;19(183):1–14.
- [9] Godziuk K, Prado CM, Woodhouse LJ, Forhan M. The impact of sarcopenic obesity on knee and hip osteoarthritis: a scoping review. *BMC Musculoskelet Disord* 2018;19(1). <https://doi.org/10.1186/s12891-018-2175-7>.
- [10] Godziuk K, Prado CM, Woodhouse LJ, Forhan M. Prevalence of sarcopenic obesity in adults with end-stage knee osteoarthritis. *Osteoarthr Cartil* 2019;27(12):1735–45. <https://doi.org/10.1016/j.joca.2019.05.026>.
- [11] Gao Q, Mei F, Shang Y, Hu K, Chen F, Zhao L, et al. Global prevalence of sarcopenic obesity in older adults: a systematic review and meta-analysis. *Clin Nutr* 2021;40(7):4633–41. <https://doi.org/10.1016/j.clnu.2021.06.009>.
- [12] Balogun S, Scott D, Aitken D. Association between sarcopenic obesity and knee osteoarthritis: a narrative review. *Osteoarthr Cartil Open* May 2024: 100489. <https://doi.org/10.1016/j.oocarto.2024.100489>.
- [13] Cappellari GG, Guillet C, Poggio-galle E, Ballesteros Pomar MD, Batsis JA, Boirie Y, et al. Sarcopenic obesity research perspectives outlined by the sarcopenic obesity global leadership initiative (SOGLI) – proceedings from the SOGLI consortium meeting in rome November 2022. *Clin Nutr* 2023;42(5):687–99. <https://doi.org/10.1016/j.clnu.2023.02.018>.
- [14] Yuan S, Hunter DJ. Precision weight management in people with knee osteoarthritis and overweight or obesity. *Nat Rev Rheumatol* 2024. <https://doi.org/10.1038/s41584-024-01209-2>.
- [15] Donini LM, Poggio-galle E. Body composition in sarcopenic obesity: systematic review of the literature. *Med J Nutrition Metab* 2013;6:191–8. <https://doi.org/10.1007/s12349-013-0135-1>.
- [16] Conte C, Hall KD, Klein S. Is weight loss-induced muscle mass loss clinically relevant? *JAMA* 2024. <https://doi.org/10.1001/jama.2024.6586>.
- [17] Prado CM, Phillips SM, Gonzalez MC, Heymsfield SB. Muscle matters: the effects of medically induced weight loss on skeletal muscle. *Lancet Diabetes Endocrinol* November 2024. [https://doi.org/10.1016/s2213-8587\(24\)00272-9](https://doi.org/10.1016/s2213-8587(24)00272-9).
- [18] Godziuk K, Prado CM, Forhan M. Protocol for the POMELO (Prevention of Muscle Loss in Osteoarthritis) randomized pilot feasibility trial. *Osteoarthr Cartil Open* 2022;4(4):100312. <https://doi.org/10.1016/j.oocarto.2022.100312>.
- [19] Vandebroucke JP, Von Elm E, Altman DG, Gøtzsche PC, Mulrow CD, Pocock SJ, et al. Strengthening the reporting of observational studies in epidemiology (STROBE): explanation and elaboration. *PLoS Medicine* 2007;4. <https://doi.org/10.1371/journal.pmed>.
- [20] Wang Z, Ma J, Si D. Optimal cut-off values and population means of waist circumference in different populations. *Nutr Res* 2010;23(2):191–9. <https://doi.org/10.1017/S0954422410000120>.
- [21] Janssen M, Pickard A, Golicki D, Gudex C, Niewada M, Scalzone L, et al. Measurement properties of the EQ-5D-5L compared to the EQ-5D-3L across eight patient groups: a multi-country study. *Qual Life Res* 2013;22:1717–27. <https://doi.org/10.1007/s11136-012-0322-4>.
- [22] Western Ontario and McMaster Universities osteoarthritis index (WOMAC). *Am Coll Rheumatol* 2015. <http://www.rheumatology.org/I-Am-A/Rheumatologist/Research/Clinician-Researchers/Western-Ontario-McMaster-Universities-Osteoarthritis-Index-WOMAC>. [Accessed 23 August 2016].
- [23] Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA, et al. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. *Clin Nutr* 2022;41(4):990–1000. <https://doi.org/10.1016/j.clnu.2021.11.014>.
- [24] Studenski SA, Peters KW, Alley DE, Cawthon PM, McLean RR, Harris TB, et al. The FNIH sarcopenia project: rationale, study description, conference recommendations, and final estimates. *J Gerontol Ser A Biol Sci Med Sci* 2014;69 A(5):547–58. <https://doi.org/10.1093/gerona/glu010>.
- [25] Rikli RE, Jones CJ. Development and validation of criterion-referenced clinically relevant fitness standards for maintaining physical independence in later years. *Gerontol* 2013;53(2):255–67. <https://doi.org/10.1093/geront/gns071>.
- [26] Levine ME, Crimmins EM. The impact of insulin resistance and inflammation on the association between sarcopenic obesity and physical functioning. *Obesity* 2012;20(10):2101–6. <https://doi.org/10.1038/oby.2012.20>.
- [27] Poggio-galle E, Lubrano C, Sergi G, Coin A, Gnessi L, Mariani S, et al. Sarcopenic obesity and metabolic syndrome in adult caucasian subjects. *J Nutr Health Aging* 2016;20(9).
- [28] Woolcott OO, Bergman RN. Defining cutoffs to diagnose obesity using the relative fat mass (RFM): association with mortality in NHANES 1999–2014. *Int J Obes* 2020;44(6):1301–10. <https://doi.org/10.1038/s41366-019-0516-8>.
- [29] Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. *Am J Clin Nutr* 2000;72:694–701. <https://doi.org/10.3945/ajcn.2009.282141>.
- [30] Godziuk K, Woodhouse L, Prado C, Forhan M. Clinical screening and identification of sarcopenic obesity in end-stage knee osteoarthritis. *Clin Nutr ESPEN* 2020.
- [31] Cawthon PM, Manini T, Patel SM, Newman A, Travison T, Kiel DP, et al. Putative cut-points in Sarcopenia components and incident adverse health outcomes: an SDOC analysis. *J Am Geriatr Soc* 2020;68(7):1429–37. <https://doi.org/10.1111/jgs.16517>.
- [32] Bhasin S, Travison TG, Manini TM, Patel S, Pencina KM, Fielding RA, et al. Sarcopenia definition: the position statements of the Sarcopenia definition and outcomes consortium. *J Am Geriatr Soc* 2020;68(7):1410–8. <https://doi.org/10.1111/jgs.16372>.
- [33] Batsis JA, Mackenzie TA, Lopez-Jimenez F, Bartels SJ. Sarcopenia, sarcopenic obesity, and functional impairments in older adults: National health and nutrition examination surveys 1999–2004. *Nutr Res* 2015;35(12):1031–9. <https://doi.org/10.1016/j.nutres.2015.09.003>.
- [34] Gonzalez MC, Mehrnezad A, Razaviarab N, Barbosa-Silva TG, Heymsfield SB. Calf circumference: cutoff values from the NHANES 1999–2006. *Am J Clin Nutr* 2021;104(1):1–9. <https://doi.org/10.1093/ajcn/nqab029>.
- [35] Hinman RS, Jones SE, Nelligan RK, Campbell PK, Hall M, Foster NE, et al. Absence of improvement with exercise in some patients with knee osteoarthritis: a qualitative study of responders and nonresponders. *Arthritis Care Res* 2023. <https://doi.org/10.1002/acr.25085>.
- [36] Ardeljan AD, Polisetty TS, Palmer J, Vakharia RM, Roche MW. Comparative analysis on the effects of Sarcopenia following primary total knee arthroplasty: a retrospective matched-control analysis. *J Knee Surg* 2022;35(2):128–34. <https://doi.org/10.1055/s-0040-1713355>.

- [37] Babu JM, Kalagara S, Durand W, Antoci V, Deren ME, Cohen E. Sarcopenia as a risk factor for prosthetic infection after total hip or knee arthroplasty. *J Arthroplast* 2019;34(1):116–22. <https://doi.org/10.1016/j.arth.2018.09.037>.
- [38] Donini LM, Poggiofalle E, Mosca V, Pinto A, Brunani A, Capodaglio P. Disability affects the 6-Minute walking distance in obese subjects (BMI>40 kg/m²). *PLoS One* 2013;8(10). <https://doi.org/10.1371/journal.pone.0075491>.
- [39] Toombs RJ, Ducher G, Shepherd JA, De Souza MJ. The impact of recent technological advances on the trueness and precision of DXA to assess body composition. *Obesity* 2012;20(1):30–9. <https://doi.org/10.1038/oby.2011.211>.
- [40] Batsis JA, Barre LK, Mackenzie TA, Pratt SI, Lopez-Jimenez F, Bartels SJ. Variation in the prevalence of sarcopenia and sarcopenic obesity in older adults associated with different research definitions: dual-energy X-ray absorptiometry data from the National health and nutrition examination survey 1999–2004. *J Am Geriatr Soc* 2013;61(6):974–80. <https://doi.org/10.1111/jgs.12260>.
- [41] Liao C De, Huang SW, Huang YY, Lin CL. Effects of sarcopenic obesity and its confounders on knee range of motion outcome after total knee replacement in older adults with knee osteoarthritis: a retrospective study. *Nutrients* 2021;13(11). <https://doi.org/10.3390/nu13113817>.
- [42] Liao C-D, Chen H-C, Liou T-H, Lin C-L, Huang S-W. Impact of Sarcopenia and obesity on gait speed after total knee replacement. *J Am Med Dir Assoc* 2022;4:631–7. <https://doi.org/10.1016/j.jamda.2022.01.056>.
- [43] Humphrey TJ, Sina Salimy M, Jancuska JM, Egan CR, Melnic CM, Alpaugh K, et al. Sarcopenia is an independent risk factor for failure to achieve the 1-year MCID of the KOOS, JR and PROMIS PF-SF10a after TKA. *Knee* 2023;42: 64–72. <https://doi.org/10.1016/j.knee.2023.02.018>.
- [44] Ishii S, Chang C, Tanaka T, Kuroda A, Tsuji T, Akishita M, et al. The association between sarcopenic obesity and depressive symptoms in older Japanese adults. *PLoS One* 2016;11(9). <https://doi.org/10.1371/journal.pone.0162898>.
- [45] Venant V, Pouget M, Lahaye C, Gentes E, Pereira B, Lambert C, et al. Depression severity as a risk factor of sarcopenic obesity in morbidly obese patients. *J Nutr Health Aging* 2019;23(8):761–7.
- [46] Misra D, Fielding RA, Felson DT, Niu J, Brown C, Nevitt M, et al. Risk of knee osteoarthritis with obesity, sarcopenic obesity, and Sarcopenia. *Arthritis Rheumatol* 2019;71(2):232–7. <https://doi.org/10.1002/art.40692>.
- [47] Godziuk K, Tilley S, LaValley M, Nevitt M, Lewis C, Torner J, et al. Obesity subtypes and trajectories of functional change after 7-years of follow-up: the multicenter osteoarthritis (MOST) study. In: American college of rheumatology congress 2024; 2024. Washington, DC, <https://acrabstracts.org/abstract/obesity-subtypes-and-trajectories-of-functional-change-after-7-years-of-follow-up-the-multicenter-osteoarthritis-most-study/>. [Accessed 4 January 2025].
- [48] Barazzoni R, Bischoff S, Boirie Y, Busetto L, Cederholm T, Dicker D, et al. Sarcopenic obesity: time to meet the challenge. *Obes Facts* 2018;11(4): 294–305. <https://doi.org/10.1159/000490361>.
- [49] Cortes TM, Chae K, Foy CM, Houston DK, Beavers KM. The impact of lifestyle-based weight loss in older adults with obesity on muscle and bone health: a balancing act. *Obesity* 2025. <https://doi.org/10.1002/oby.24229>.
- [50] Chen AS, Batsis JA. Treating sarcopenic obesity in the era of incretin therapies: perspectives and challenges. *Diabetes* July 2025. <https://doi.org/10.2337/dbi25-0004>.
- [51] Chu IJH, Lim AYT, Ng CLW. Effects of meaningful weight loss beyond symptomatic relief in adults with knee osteoarthritis and obesity: a systematic review and meta-analysis. *Obes Rev* 2018;19(11):1597–607. <https://doi.org/10.1111/obr.12726>.
- [52] Prado CM, Landi F, Chew STH, Atherton PJ, Molinger J, Ruck T, et al. Advances in muscle health and nutrition: a toolkit for healthcare professionals. *Clin Nutr* 2022;41(10):2244–63. <https://doi.org/10.1016/j.clnu.2022.07.041>.
- [53] McCarthy C, Tinsley GM, Bosy-Westphal A, Müller MJ, Shepherd J, Gallagher D, et al. Total and regional appendicular skeletal muscle mass prediction from dual-energy X-ray absorptiometry body composition models. *Sci Rep* 2023;13(1):2590. <https://doi.org/10.1038/s41598-023-29827-y>.