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Research paper

Sarcopenic obesity

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ABSTRACT

"Sarcopenic obesity" describes the confluence of low muscle mass and/or strength with obesity in older adults, thought to contribute to increased risk for poor health outcomes compared to either condition alone. Despite almost 20 years of research into sarcopenic obesity, it currently lacks a consensus definition and this is a barrier to research and clinical investigations. This narrative review summarises current evidence of the role of sarcopenic obesity in age-related declines in musculoskeletal and cardiometabolic health, and potential treatment strategies. Research to date suggests that sarcopenic obesity, when defined by low muscle strength, contributes to significantly increased risk for poor physical function and possibly falls. It is likely that sarcopenic obese individuals have reduced bone quality relative to obese alone, and combined with an increased falls risk, may be predisposed to increased risk for fractures. Low muscle mass in obesity may also be associated with increased risk for cardiovascular disease, type II diabetes and mortality, however prospective studies are required to confirm the effects of sarcopenic obesity on musculoskeletal and cardiometabolic health in older adult populations. Similarly, large-scale randomised controlled trials are needed to clarify the most effective methods for reducing prevalence and incidence of sarcopenic obesity, but it is likely that lifestyle modification interventions which combine aerobic and resistance training, caloric restriction, and protein and/or vitamin D supplementation, may be most effective.

1. Introduction

The term "sarcopenic obesity" was first used to describe the condition of low skeletal muscle mass relative to fat mass by Heber et al. in 1996 [1]. Sarcopenia (age-related muscle wasting) was at the time considered an independent contributor to functional decline in older adults [2]. Given that obese older adults have increased risk for functional decline [3], it seemed logical that functional deficits would be even greater in those with sarcopenic obesity, described by Roubenoff as "the worst of both worlds" [4]. Furthermore, as the largest insulin sensitive tissue in the body, a loss of muscle mass could increase risk for poor cardiometabolic health in obese older adults [5].

Evidence soon began to challenge the concept that low muscle mass is an independent predictor of strength and functional declines. Longitudinal studies demonstrated that age-related loss of strength significantly exceeds the rate of loss of muscle mass [6,7]. Furthermore, associations of low muscle strength with functional decline, falls and mortality are generally observed to be independent of muscle mass [8]. These findings led to the proposal of several multi-dimensional consensus definitions of sarcopenia since 2010, which incorporate assessment of muscle function and/or physical performance in addition to muscle mass [9–13]. Others contend that research should focus on strength declines only, and have proposed the term dynapenia to describe this condition [14]. The lack of an internationally accepted definition for sarcopenia is a source of confusion for clinicians and researchers interested in the epidemiology, diagnosis and treatment of

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this condition. Confusion is exacerbated in the case of sarcopenic obesity because there is similarly poor agreement on measurement techniques or thresholds for obesity, and no consensus definition of sarcopenic obesity has yet been proposed. Stenholm et al. even proposed that sarcopenic obesity should be defined by muscle function rather than muscle mass [15]. However, generally sarcopenic obesity is considered a condition of low muscle mass alone in obese older adults [16], while some investigators have adopted the term "dynapenic obesity" to describe low muscle strength in obesity [17,18].

2. Prevalence

Any review of sarcopenic obesity is thus limited by a lack of comparability between studies and a recent review of population-based studies reported that prevalence estimates range from 0–41% depending on population characteristics (eg. ethnicity and age) and definitions applied [16]. A systematic review by Batsis et al. identified eight distinct definitions of sarcopenic obesity using varying cut-points for sarcopenia (usually defined as low appendicular lean mass relative to height in metres squared [ALM/m^2] measured by dual-energy X-ray absorptiometry [DXA]) and obesity (usually defined as high body fat percentage measured by DXA) [19]. In the NHANES study, sarcopenic obesity prevalence estimates for these eight different definitions ranged from 4–94% [19]. A limitation of ALM/m^2 in defining sarcopenic obesity was highlighted by the US Health ABC Study where no obese men or women were found to have sarcopenia, demonstrating that obese older adults have higher muscle mass and so normalising muscle mass to body weight or fat, rather than height alone, may be more appropriate for sarcopenic obesity case-finding [20]. Similarly, despite higher absolute muscle mass compared to non-obese, obese individuals demonstrate poorer relative force production suggesting that assessments of muscle quality, particularly high levels of inter-muscular adipose tissue (IMAT) which independently predict functional limitation [21], should be included in a future consensus definition of sarcopenic obesity [22].

3. Pathogenesis

Body fat mass generally peaks in middle to older age and plateaus before declining in very old age, whereas lean mass decreases progressively from adulthood [23]. Age-related increases in fat mass often occur in the absence of weight gain due to concomitant loss of lean mass [24]. Furthermore, declines in lean mass may contribute to gains in fat mass, and vice-versa [4]. For example, decreases in muscle mass may reduce basal metabolic rate and physical activity, with subsequent decreases in energy expenditure contributing to fat increases, while gains in visceral adipose tissue and IMAT in particular may exacerbate sarcopenia through secretion of pro-inflammatory cytokines [4,15,22]. Higher levels of pro-inflammatory cytokines are thought to contribute to declines in muscle mass and strength, and indeed elevated interleukin-6 and C-reactive protein levels have been observed in sarcopenic obesity [25]. Inflammation may also mediate insulin resistance in obesity and this is associated with increased loss of muscle mass in community-dwelling older adults [26]. Leptin resistance may follow gains in fat and this may lead to poorer fatty acid oxidation and an increase in IMAT deposition in muscle, with subsequent functional declines [22]. Low levels of testosterone and growth hormone are also common in obesity and older age, and have also been associated with lower muscle mass [27,28].

Modifiable lifestyle factors, particularly poor physical activity, diet and sun exposure, may additionally contribute to the development and progression of sarcopenic obesity, and will be discussed later in this article.

4. Musculoskeletal health

4.1. Mobility, disability and falls

The majority of research on musculoskeletal outcomes of sarcopenic obesity has focused on mobility and disability with the rationale that older adults are at increased risk of disability if their muscle mass is inadequate for their body size [20]. However, some cross-sectional studies have reported increased mobility disability and overall disability in older adults with sarcopenic obesity [29–31], while others report that physical function is similar to that of obese [33] or sarcopenic [34] alone, and even to non-sarcopenic non-obese older adults [32]. Few data are available from longitudinal studies but are also controversial; Baumgartner et al. reported older adults with sarcopenic obesity had two and a half-fold increased risk for incident self-reported disability over eight years [35], whereas there was no change in physical components associated with falls risk for sarcopenic obese compared to non-sarcopenic non-obese older adults over five years in the Tasmanian Older Adult Cohort study [36].

When defined by low muscle strength, however sarcopenic obesity appears to be consistently associated with poorer physical function [37–39]. In both US and Chinese older adults, those in the lowest tertile for muscle strength with high fat mass or BMI have significantly poorer objectively-assessed mobility and self-reported physical function than those with low muscle strength or high fat mass alone [17,40,41]. A Korean study reported a significant interaction suggesting that low muscle strength and high fat mass may confer a synergistic rather than additive effect on disability [42].

Mobility disability predicts increased falls in older adults [43], but research to date has not extensively examined whether falls are more common in sarcopenic obese individuals. In the New Mexico Elder Health Survey, sarcopenic obesity was associated with a three-fold increased likelihood of self-reported past-year falls in men, but not women [29]. Sarcopenic obese older adults in a New Zealand study also reported the highest number of past-year falls but the difference was not significant compared to other groups [44]. As described above, sarcopenic obesity in the TASOAC study was not associated with decreased performance in a validated assessment of falls risk over five years, although when defined by low muscle strength, sarcopenic obese individuals had a significantly greater increase in falls risk scores [36]. Large prospective studies are required to determine the risk of incident falls in sarcopenic obesity defined according to muscle mass and strength.

4.2. Bone health and fractures

Higher skeletal muscle mass and strength are generally associated with better bone health [45–47], and sarcopenia is prevalent amongst older men and women with hip fractures (95 and 64%, respectively) [48]. Obesity has generally been considered to reduce fracture risk, due primarily to the higher bone mineral density (BMD) of obese individuals [49,50]. However, in the US, approximately half of all fractures in those aged 65–74 years occur in overweight and obese individuals [51], and obese women are more likely to experience ankle and upper leg fractures than non-obese women [49].

Higher fat mass may improve BMD both through mechanical loading and estrogen metabolism [52]. However, recent research suggests that higher visceral adipose tissue in particular may compromise bone health and this may be related to increased insulin resistance, adipokine and inflammatory markers, all of which may have negative effects on bone and are more commonly