Obesity and Mobility in Advancing Age: Mechanisms and Interventions to Preserve Independent Mobility

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Abstract Obesity and aging adversely affect musculoskeletal health and functional independence through common physiological pathways. Muscle quality and strength decline as muscle issues become infiltrated with fat. Inflammation, insulin resistance and anabolic hormonal deficits develop. Musculoskeletal disease develops over time, causing painful movement. Muscle cell regenerative capacity also declines. The collective result is onset of mobility disability. Pain catastrophizing and kinesiophobia may exacerbate disability and accelerate muscle strength loss and overall fat accumulation. Interventional strategies to prevent mobility disability in the aging, obese population should be multifaceted. Long term success with independent mobility is probable when interventions include weight and pain management techniques, support and coaching, strengthening exercise for lower extremity and thoracic musculature, and special nutritional considerations to promote preservation of muscle mass.

Keywords Obesity · Mobility · Aging · Resistance exercise · Inflammation

Introduction

Aging adults face numerous health challenges, including the onset of musculoskeletal diseases and subsequent mobility impairment. Obesity exacerbates health complications and functional decline. Mobility is the ability to transfer body weight, climb stairs, walk independently and walk at moderate speeds. Better mobility reflects higher quality of life and longer survival [1, 2]. The population of persons over 60 years of age is projected to increase from 600 million to over two billion by the year 2050 [3] thereby increasing the demand for health care [4]. This is especially true in rural areas with unprecedented growth of the older demographic where obesity and related chronic conditions are prevalent [5]. Disability is related to hormonal and intracellular inflammatory changes, muscle strength deficits, musculoskeletal pain and psychosocial factors. Lifestyle modification, pain and weight management and nutritional adjustments may be helpful in combatting mobility decline in the obese, older adult.

Obesity and Aging: The Perfect Chemical Storm for Musculoskeletal Disability

Both obesity and aging confer independent, adverse effects on musculoskeletal health and functional independence. Body composition shifts toward an increased percentage of body fat. Adiposity, especially central adiposity, is related to the high circulating levels of inflammatory cytokines. These cytokines include interleukin-6 (IL-6), tumor necrosis factor (TNF- α) and C-reactive protein (CRP) [6]. TNF- α contributes to skeletal muscle catabolism [7] and perpetuates catabolism in obesity [6]. Once TNF- α binds to muscle receptors, cellular apoptosis begins. Cellular apoptosis corresponds to walking speed decline in healthy older adults [8•]. Elevated TNF- α impairs myofibrillar contractile activity and reduces

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muscle specific force values [9]. High IL-6 levels are also related to declines in strength, physical function and walking ability [10, 11].

In both obesity and aging, elevated levels of circulating free fatty acids are common. Fatty acids suppress growth hormone production and are accompanied by low testosterone [12]. Anabolic hormone levels are directly related to muscle strength and mobility. Both obesity and aging foster insulin resistance [13], especially when coupled with sedentary behavior. Under normal conditions, insulin is a powerful anabolic agent. With intramuscular lipid accumulation and physical inactivity, lipotoxicity occurs within muscle cells that blunts anabolic signaling [14]. Lipid accumulation decreases the efficiency of insulin and glucagon sensitivity, leading to insulin resistance. Fatty acids also accumulate within the pancreas [15], blunting insulin secretion. Leptin is an adipokine that normally regulates appetite. Intriguing associative data shows that circulating leptin levels are highest in older adults with sarcopenic obesity compared to those who are sarcopenic or viscerally obese alone [16]. Leptin may exacerbate inflammation and reduce muscle protein synthesis [16]. These physiological pathways contribute to the deterioration of skeletal muscle quality and contractile efficiency in the older adult [17].

Sarcopenia and Obesity on Mobility Limitations

With normal aging, there is a deterioration in motor neurons innervating type II muscle fibers that results in a loss of type II muscle fibers mass and disproportionately greater loss in strength. Appendicular muscle mass declines at an annual rate of approximately 1 %, but strength declines at an annual rate of 2.6–3.4 % [18]. With obesity, there is a relative deficit in muscle mass or strength to support the large body mass (sarcopenia) [19]. Sarcopenia may be defined by very low appendicular skeletal muscle mass that is less than two standard deviations below that of a young adult reference population, or a body fat percentage >60th percentile for the same gender and age. Sarcopenic obesity currently affects 16–17.5 % of elderly women and men [20]. Controversy exists regarding the independent and combined relationships between obesity, sarcopenia and mobility impairment. Intuitively, obesity and sarcopenia may act synergistically in older individuals to impair mobility. Some studies support this concept [21–23], whereas others do not [24–27]. Possible reasons are presented next.

First, several methodological variations exist in the literature. Variations include different sarcopenia definitions and calculations, fat determination methods and the use of self-report versus objective functional tests to determine mobility. Body mass index (BMI) has been extensively used to characterize obesity status because of the ease of administration, and

the strong associations with lifestyle disease risks, but body composition analyses may provide more insight [28] of obesity effects on mobility. Dufour et al. [29••] compared mobility limitations in elderly men and women relative to different sarcopenia calculations and presence of obesity. Interestingly, obesity and sarcopenia did not synergistically affect the risk of developing a mobility limitation. Different perceptions of mobility limitations depending on their life experience, and the limiting nature of dichotomization of lean mass to fit sarcopenia definitions may explain these results [29••]. Studies are warranted to determine if there is a lean mass ‘threshold’ at which mobility dramatically deteriorates.

Second, muscle quality deteriorates as fat infiltrates aging muscle. Normally, mature muscle cells experience microtrauma and subtle injury. Satellite cells either fuse together to form new muscle fibers or meld with mature muscle cells to assist existing myonuclei [30•]. Muscle function can be maintained. With aging, satellite cell activity and regenerative capacity are reduced [30•]. In obesity, lipids accumulate in skeletal muscle [14], and this activates proinflammatory mediators such as TNF- α , and IL-1 and IL-6. TNF- α interferes with satellite cell function and regeneration. The presence of adipogenic and fibrous progenitor cells that exist in muscle tissue may give rise to fat accumulation in skeletal muscle [31], and may perpetuate the skeletal muscle repair processes that reduce strength with aging.

Third, there may be data interpretation complications and survivorship bias. The Health, Aging and Body Composition study examined the relationships between knee extensor strength, muscle quality (muscle strength/lean leg mass), cytokines and c-reactive protein over eight years in elderly adults [32]. Leg muscle mass and quality consistently decreased in men and women over time. Because some participants were unable to participate and some had died, the rate of change in body composition values was likely much different than relatively healthier completers. The relationships between leg lean mass, strength and obesity might have been underestimated [32].

Consider Non-weight Bearing Muscles in Mobility Impairment: Trunk and Hip

Leg strength is certainly important for maintenance of mobility. Yet, new data from our laboratory and others [33••, 34•] suggest that trunk muscles are important in maintaining mobility, particularly if the individual is experiencing axial or lower extremity pain. With aging, there is an increasing prevalence of these painful joint conditions [35, 36]. Back extensor muscle strength decreases annually by an average of 2.5 % [37]. Hip strength can be compromised in older adults, especially women [33••]. This is clinically relevant, because hip flexors may assume an active role during complex

activities as obese people rock forward to rise from a chair or bed [33••] and to maintain balance after a perturbation [34•].

Community-dwelling, older adults with mobility limitations were tested using a trunk flexion-extension device. Functional tests were performed including the Short Physical Performance Battery (SPPB), Berg Balance Scale muscle strength and muscle endurance [38]. Trunk extension endurance contributed similarly to the SPPB scores as leg press strength. The authors identified trunk endurance and strength as rehabilitation targets. We recently completed a randomized, controlled study examining the comparative efficacy of two different resistance exercise programs on mobility and low back pain in obese adults aged 60–85 years. Regression analyses revealed that normalized lumbar extensor strength contributed 20 % of the variance of walking velocity. Normalized leg press, leg extension and leg curl strength did not significantly contribute to walking velocity. Thus, for older obese adults experiencing chronic low back pain, strengthening the supporting musculature surrounding the painful joint is very important for mobility.

In elderly women, timed-up-and-go scores and 6-meter walking speed were not related to muscle mass or sarcopenia measures, but isometric hip strength was a significant predictor of both functional scores [39]. Hip abductors are critical for pelvis stabilization and for performing activities of daily living. As hip strength declines, pelvic tilt occurs and walking speed slows [39]. These collective data suggest that mobility can be improved with the incorporation of stabilizing strengthening exercise for the muscles of the lower back and hip. In obese older adults with low back pain, lumbar muscle strength was inversely related to chair rise time and stair climb time, but not with daily steps taken, or intensity of steps taken [40••]. Normalized leg press strength was also 25 % lower in severely obese persons compared to overweight counterparts [40••]. Furthermore, lumbar strength was a significant and independent predictor of walking endurance and steps taken per day. For every unit increase in lumbar strength, there was a daily step activity increase of 284 steps. Low back strengthening may foster increased habitual physical activity.

Musculoskeletal Pain and Mobility

Obesity-related muscle and joint pain precludes adherence to exercise and physical activity recommendations [41]. Arthritis affects 35.6 % of obese adults and this disease is a likely barrier to participation in exercise [42]. In the elderly, the prevalence of chronic pain is 52 %, with the greatest risk occurring in persons with abdominal obesity [43]. As weight decreases, musculoskeletal pain also decreases [44]. Even in older adults up to 99 years, the odds ratio of reporting pain was 3.1 to 4.4 in persons who were stage I to stage III obese

compared to normal weight referents [45•]. A 10-year prospective study found that obese women had a 20 % increased risk of developing chronic low back, neck and shoulder pain; this risk was partly offset by participation in ≥ 1 hour of physical activity per week [46]. Severe obesity dramatically impacts the weight-bearing joints such as the hip, knee and ankle through skeletal misalignment, mechanical compression and progression of degenerative joint disease [47•]. Pain sensitivity may be dependent in part on metabolic mediators such as oxytocin, leptin or ghrelin [48]. Emerging evidence suggests that abnormal levels of these hormones may adversely modulate nociception and decrease pain thresholds [49–51].

Pain complicates the relationship between aging and mobility decline. Deconditioning and obesity contribute to increased pain sensitivity [52]. Even with significant weight loss, pain sensitivity may or may not be restored to normal. Pain severity is typically greater in lower extremity sites of obese compared to non-obese persons [52]. Pain is a common symptom of degenerative musculoskeletal diseases, and is a main factor contributing to mobility disability [53]. Indeed, severe foot [54], knee [55], hip and back pain all disrupt normal gait, walking indoors/ outdoors, shopping, standing for a long period, and participation in other mobility activities. We, and others, surmise [33••] that during various activities individuals may develop different movement strategies because of sensorial deficits, or to offload the painful joint [40••]. Specifically, obese individuals have reduced trunk flexion and have a posterior movement of the feet compared to non-obese individuals during a sit to stand task [56]. This biomechanical shift minimizes torque on the lumbar spine, and helps to suppress pain symptoms while shifting loading and pain to the knees. While gait and movement adaptations temporarily solve one pain issue, downstream effects include aberrant loading.

Psychosocial Issues that Impact Mobility

Obesity adversely affects attitudes about the benefits of regular exercise [57•]. For example, overweight individuals with ankylosing spondylitis reported fewer benefits of physical activity and more perceived barriers to exercise than healthy counterparts [57•]. Obese persons are more likely to have low self-compassion (the ability to be touched by one's own suffering and to make behavioral changes to alleviate the suffering), a state which contributes to negative affect, pain catastrophizing and pain-related disability. The fear of exacerbating joint pain with loading and rumination on pain may also be problematic for this population.

Pain Catastrophizing Pain catastrophizing is a robust psychosocial dimension that is defined as ‘the tendency to focus

on pain and negatively evaluate one's ability to deal with pain' [58]. Overweight and obesity amplify pain osteoarthritis symptoms and can induce pain catastrophizing and higher psychological disability. As pain catastrophizing worsens, pain severity increases, and behaviors that perpetuate weight gain increase, such as physical inactivity [59, 60]. Catastrophizing has serious ramifications especially in single person households, as it relates to work disability, limited physical function and increased healthcare use [60]. In obese persons with knee osteoarthritis, the use of pain coping skills training (group sessions that taught patients how to control and decrease pain by distraction, changing activities and relaxation, and decrease maladaptive pain catastrophizing) coupled with behavioral weight management (nutritional and physical activity modifications, attitude change, relationships and lifestyle) effectively reduced joint pain, reduced perceived physical disability and improved self-efficacy [61•].

Kinesiophobia Fear of movement due to pain may impact mobility. In our recent work in obese adults with back or knee pain, kinesiophobia (fear of movement) levels were measured at the onset of therapy for their pain. In morbidly obese patients with knee pain compared to non-obese counterparts, average pain scores were higher (7.5 vs 4.8 points out of 10; $p < .05$) and average kinesiophobia scores were 18.8 % higher [62]. The international knee documentation scores (IKDC; representative of mobility tasks and activities of daily living) were relatively low in the morbidly obese patients. Both pain and kinesiophobia were significant predictors of IKDC scores.

In patients with chronic low back pain, we examined the relationship between obesity status, kinesiophobia and back related disability due to pain [63]. Obese persons with back pain reported higher Tampa Scale of Kinesiophobia scores (TSK) than non-obese persons and these scores were significant contributors to the variance of regression models for self-reported walking ability. In a group of obese, older men and women, we found that ambulatory pain was related to BMI, but not TSK scores. TSK scores were also not related to walking endurance time [64•]. Variations in pain coping ability may explain these results. Some participants with moderate walking pain ratings (5 to 7 out of a 0–10 point scale) kept walking on the treadmill and [65] reported that they were “used to it” and knew that “they had to walk even if it hurts [64•].” Individuals who were positive and optimistic throughout the testing treated the testing as a challenge and not as a fearful event. This behavior might reflect pain acceptance (a state in which an individual accepts what cannot be changed). Persons with high levels of acceptance in chronic pain conditions demonstrate lower levels of pain-related disability [66].

Minimizing Mobility Disability in the Aging, Obese Population

National health and cost implications for a growing obese, aging demographic are ominous. The societal impact of reducing mobility disability could be highly substantial by reducing functional dependence, healthcare resource use, chronic comorbidities and related medical costs. Interventions that may offset this health care burden include positively changing perceptions of health, pain management, lifestyle coaching, managing body weight and filling dietary deficits.

Positively Affect Perceptions Based on our focus group work, participation in regular physical activity appears to be multifactorial. For women, the negative self-perceptions of body weight, adiposity and onset of joint pain coupled with the use of food as comfort are common factors that perpetuate obesity and physical limitations. For men, the frustration of joint pain combined with the inability to perform specific physical tasks or regular exercise are common driving forces to seek help for obesity management. The presence of an engaged, enthusiastic spouse can have strong effects on the perceptions of lifestyle modification on the other. Specifically, when couples have participated in our research, the perceptions of one pessimistic or skeptical spouse positively change over time similar to that of the spouse with the positive attitude such that regular physical activity and diet modification become important life goals.

Lifestyle change counselors or coaches may be valuable assets to future mobility programs. These professionals should ideally have experience in program planning and troubleshooting, nutrition awareness, exercise, and psychology expertise to non-judgmentally provide guidance. The Treatment of Obesity in Underserved Rural Settings study demonstrated the effectiveness of telephone and face-to-face counseling for weight loss in obese older adults in rural areas; counseling included methods of problem solving issues related to achieving nutritional and exercise goals [67]. Fostering self-compassion reduces negative affect, pain catastrophizing and pain-related disability [65]. Setting realistic expectations for lifestyle intervention programs is essential for long term sustenance of healthy behaviors. Preparing the individual with the likely weight change and the realistic effects on functional mobility is critical to prevent disappointment and dropout. Informing individuals of the potential sensations of dietary modification and physical activity also decreases fear and uncertainty of lifestyle programs. Discussion of hunger acceptance, muscle soreness, perceived fatigue during exercise and discernment of acute injury from muscle soreness are helpful in setting expectations.

Pain Management Reduction of bodily pain through psychological therapy or medically managed pharmaceuticals can be helpful in restoring some or most physical function and mobility in the obese older adult [68]. Cooperation of healthcare providers and counselors to maximize psychological outlook and control joint pain is necessary to achieve long term success with weight management and functional independence. When joint symptoms worsen, the use of intra-articular injectable agents (corticosteroids or hyaluronic acid) may temporarily relieve pain [68] and permit the individual to keep exercising and performing activities of daily living.

Implement Weight Management Strategies Sustained weight loss improves mobility in the obese older adult [69]. Design and administration of weight loss interventions for the older adult is complicated however. Supervised nutrition and exercise programs are among the most effective for long-term weight loss, musculoskeletal pain reduction and mobility improvement [70•]. Individual or combined diet (typically caloric restriction by 500 kcal/day) and exercise programs can improve functional performance in walking (gait patterns, velocity, distance) and stair climb time [71]. Riecke et al. [72] evaluated the effect of weight loss on knee pain and disability in obese adults with osteoarthritis using two low energy diets (415 or 810 kcal/day) for 8 weeks followed by a hypocaloric diet 1200 kcal/day) for an additional three months. Knee pain symptoms and physical disability were reduced by 11 % and 13 %, respectively [72]. Evidence from the Arthritis, Diet and Activity Promotion Trial [55, 73, 74] showed that a combined diet/exercise intervention reduced body weight and fat, but only the groups with exercise exhibited functional improvements. Body composition changed only in the groups that had dietary intervention. Walking distance and stair climb time improved up to 23 % and knee pain was reduced by 30 % in the combined treatment, respectively. Muscle quality improved and muscle power and endurance improved necessary for stair climb and walking distance. From the biomechanical perspective, the mean weight loss of 9.2 kg reduced the walking-related compressive forces at the knee by 2.6 % [75]. Even very short, very aggressive inpatient exercise and diet programs (~three week intervention) improved stair climb time [76]. Greater body weight and fat reductions occur with supervised training rather than self-monitored training [77], or when the program is performed with a partner who is successful with weight loss [78]. To minimize kinesiophobia, a trained exercise physiologist or therapist can guide the participant through the exercise activities that instill fear. Teaching the participant about “normal” pain versus “acutely worsening” pain during the daily activities may improve self-efficacy, foster full engagement in exercise and reduce kinesiophobia [79].

Designing weight loss programs in this population with joint pain is particularly challenging. Typically, 1500 kcal of week caloric expenditure with aerobic exercise is recommended for weight loss [80]. Yet, focusing on exercise-related caloric expenditure is often overwhelming for this demographic. We suggest that the strengthening of the musculature and stabilization of painful joints may be effective prior to starting aerobic exercise. Strength exercise increases confidence and self-efficacy, both of which can help prepare a participant for sessions of load-bearing aerobic exercise. The accumulation of shorter bouts of load-bearing exercise over a day may be palatable and more tolerable with joint pain. A slow gradual approach of adding resistance and aerobic exercises over several weeks is recommended. For example, the completion of five daily 10 minute bouts of brisk walking would increase caloric expenditure with less sustained discomfort. To increase long-term compliance, high intensity exercises (e.g., brisk incline treadmill walking, sport activity) might be best tolerated after significant weight loss (5–10 % of body weight) [81]. Convert a ‘painful load bearing exercise’ into a non-painful equivalent; if treadmill activity is unappealing, prescribe elliptical, aquatic or stationary cycling sessions.

Logging daily activity into a diary is helpful for tracking exercise sessions. In the log, self-reporting of activity type, emotions present, and feelings of muscle effort during the session are important. For every ten-minute block of physical activity, the individual earns a point. This information provides the individual with a sense of personal accomplishment at the conclusion of the day as they earn themselves points. If the individual cannot exercise for long, encourage less sitting, and encourage patience and a positive mental outlook. For this population, the goal may not be massive weight loss which is often not sustained, but a focus on the accumulation of small changes than can make a big functional difference over time. The increase of daily caloric expenditure by 100 kcal from standing more or from walking short bouts can lead to a 10 pound weight loss over a year. Improvement in mobility will occur when exercise and some weight loss are consistent. Finally, growing evidence supports the concept that while strengthening exercise can stimulate protein synthesis and muscle growth, the changes in mobility in function may also be explained in part by improvement in proprioception and improved awareness of postural control [82]. Inclusion of postural training and multisensorial training may enhance mobility [82].

Consider Special Nutritional Issues A paradoxical phenomenon occurs in obese, older adults, where despite increased adiposity, food intake declines with aging. Malnutrition is not uncommon in older adults and is a risk factor for sarcopenia. Decreased food intake, coupled with food insecurity (defined

as the uncertain availability of nutritionally adequate and safe food) [83], contribute to suboptimal dietary quality, specifically energy, protein and micronutrient intake.

Protein Compared to adequate or high protein intakes, lower protein intake decreased lean tissue mass, leg muscle strength and leg power in older women [84]. Counteracting decreased protein intake may help increase or maintain muscle mass. Studies in this area, however, have yielded mixed results. In sarcopenic elderly adults, supplementation with oral essential amino acids for four months increased lean mass to levels of those in non-sarcopenic counterparts [85]. Insulin resistance and TNF- α levels decreased, and IGF-1 increased [85]. In contrast, Chale et al. [86••] randomized mobility-limited, older adults who were participating in a 6-month resistance exercise training program to receive 40 g of whey protein or not. Both groups improved muscle strength, power and physical performance measures similarly; there were small but non-significant increases in total body lean mass with the whey supplement. The addition of 50 g/day of whey protein to a reduced calorie diet (1400 kcal/day) contributed to a greater weight loss, thigh muscle mass gain and a greater reduction in intramuscular and subcutaneous fat than a maltodextrin supplement in older obese women [87]. There were no differences in physical function tests, leg strength and balance between supplement groups. However, the increase in lean soft tissue volume predicted the amount of improvement in up-and-go test time, muscle strength, balance and strength [87]. Protein/amino acid supplementation coupled with a balanced protein distribution throughout the day may help counteract sarcopenia [83].

Micronutrients Cumulative evidence suggests that specific micronutrient deficiencies are related to poor physical performance and muscle strength in older, overweight adults. Vitamin D has received a lot of attention as it appears to modulate functional impairment, and vitamin D deficiency (serum 25-OHD $\leq$ 25 nmol/L) doubles the risk of sarcopenia [88, 89]. Persons with levels $\leq$ 53 nmol 25-OHD/L have slower performance times for chair stands and gait, shorter 6-minute walk test distance and lower handgrip strength [90•]. Some data suggests maintaining blood levels of 25-OHD at least between 70–100 nmol/L [89, 90•], as this was related with best mobility performance. Supplementation with vitamin D under medical supervision should be considered [90•].

Lower levels of fruit and vegetable-derived vitamins and minerals may contribute to mobility impairment and muscle decline [91]. Low carotenoid and selenium levels predict walking speed in cross sectional studies [91], and in prospective studies up to six years [92, 93]. A possible mechanism may be a lower anti-inflammatory and antioxidant defense against aging and obesity-related perturbations within skeletal

muscle tissue. Older, obese adults can benefit from education on food choices, adding colorful fruits and vegetables into daily meals or snacks, and cooking strategies to maintain micronutrient content.

Conclusions

There is a global challenge of escalating mobility disability in the obese, aging adult. The probability of long-term success with independent mobility is increased when interventions include sustained weight management strategies, support and coaching, pain management, strengthening exercise for lower extremity and thoracic musculature, and special nutritional considerations to promote preservation of muscle mass.

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Compliance with Ethics Guidelines

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- Of importance
- Of major importance

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