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Narrative Review of Obesity and Musculoskeletal Health

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ABSTRACT

Obesity has emerged as a global health epidemic and a major determinant of musculoskeletal (MSK) morbidity across the lifespan. This narrative review synthesizes epidemiological, biomechanical, metabolic, and clinical evidence to elucidate how excess adiposity affects the structure and function of the musculoskeletal system. Obesity is strongly associated with a higher prevalence and severity of osteoarthritis, intervertebral disc degeneration, foot and ankle disorders, sarcopenic obesity, and chronic back pain. Mechanical loading alone cannot fully explain these relationships, as adipose tissue acts as an active endocrine organ producing adipokines and proinflammatory mediators that impair cartilage integrity, alter bone remodeling, and contribute to chronic pain. Metabolic comorbidities, including insulin resistance, dyslipidaemia, and systemic inflammation, further compromise skeletal homeostasis and muscle quality. Site-specific effects are noted across the spine, hip, knee, ankle, and peripheral joints, with the knee demonstrating the most consistent obesity-related structural deterioration. The bidirectional nature of obesity and MSK disorders is increasingly recognized, as musculoskeletal pain limits physical activity and may reinforce weight gain. Evidence from rehabilitation studies demonstrates that tailored exercise interventions, weight management strategies, and targeted nutritional or pharmacological therapies can improve pain, function, and overall musculoskeletal health. However, substantial gaps remain, including limited longitudinal data, inconsistent measurement of adiposity, and underexplored mechanisms linking metabolic syndrome, sarcopenic obesity, and degenerative joint disease. This review underscores the need for integrated clinical, public health, and policy interventions to mitigate the burden of obesity-related musculoskeletal disorders and improve functional outcomes across populations.

Keywords: Obesity and Musculoskeletal Disorders, Osteoarthritis and Joint Degeneration, Sarcopenic Obesity, Adipokines and Inflammatory Pathways, and Mechanical Loading and Skeletal Health.

INTRODUCTION

Obesity has reached epidemic proportions worldwide; in 2016, more than 650 million adults were classified as obese, with a body mass index (BMI) greater than 30 (World Health Organization, 2019)[4]. At greater risk of numerous chronic diseases, individuals with obesity also bear the greatest burden from musculoskeletal disorders. Obesity is the most readily modifiable risk factor for osteoarthritis (OA), a degenerative joint condition that affects at least 10% of the population and increases steadily with age [1]. Elevated BMI is associated with greater prevalence, severity, and functional impairment among individuals with OA, particularly in the knee and hip joints [2]. Sarcopenic obesity, characterized by excess body fat despite a reduced proportion of muscular mass, impairs muscle strength and other factors impacting functional capacity [3]. The association between obesity and OA remains incomplete, necessitating further systematic exploration. While weight loss has beneficial effects on joint health, the specific mechanisms by which obesity imposes deleterious effects remain poorly understood. Unraveling how excess body weight influences the occurrence and progression of musculoskeletal disorders has important implications for early intervention, treatment, and the prevention of secondary diseases [2].

Epidemiology of Obesity and Musculoskeletal Disorders

According to the World Health Organization (WHO) definition, obesity is an excessive accumulation of body fat that presents a risk to health [5]. The body mass index (BMI) is a simple index of weight-for-height that is

commonly used to classify overweight and obesity in adults. Individuals with a BMI between 25 kg/m2 and 29.9 kg/m2 are classified as overweight, while BMI values of 30 kg/m2 or higher are termed obese [1]. Obesity, heavier load on the musculoskeletal system, and diseases arise as cultures embrace less physically demanding jobs, more sedentary recreation such as television and computer use, and more appealing food that is more accessible and easier to consume. Obesity is associated with an increased risk of developing chronic musculoskeletal conditions, such as osteoarthritis (OA), rheumatoid arthritis (RA), lupus, and fibromyalgia [3]. According to data from the WHO, more than 1.4 billion adults worldwide are considered overweight, and more than 300 million are obese [2]. Early intervention with lifestyle changes (weight reduction and increased physical activity) and/or pharmacotherapy markedly improves musculoskeletal symptoms and reduces the burden of these preventable locomotor diseases.

Mechanisms Linking Obesity to Musculoskeletal Health

Obesity is a global epidemic, associated with a greater risk of degenerative and inflammatory musculoskeletal conditions, especially osteoarthritis. Osteoarthritis is a common joint disease, affecting nearly 10% of the population, with prevalence increasing with age [2]. Obesity represents the greatest modifiable risk factor for osteoarthritis, as studies indicate that obese individuals are significantly more likely to develop knee osteoarthritis. The economic burden of osteoarthritis related to obesity is substantial, second only to diabetes, and is expected to rise with increasing obesity prevalence and longevity [1]. Musculoskeletal conditions are a leading cause of disability, contributing significantly to years lived with disability worldwide [1]. Musculoskeletal pain can lead to avoidance of physical activity and weight gain, resulting in bidirectional interaction with obesity [3]. Excessive joint loading remains an oversimplified explanation; studies have shown conflicting findings on the link between mechanical loading and pain. Body tissues exhibit heterogeneous properties, with muscle potentially providing protective effects. Body mass index is widely used to define obesity, but it does not account for tissue type or distribution, rendering it a poor measure of adiposity [5]. Fat mass index may be more relevant for predicting pain. Adipose tissue functions as an active endocrine organ, secreting cytokines and hormones that may contribute to musculoskeletal pain. Emerging evidence highlights the role of body composition in joint pain, with techniques such as dual-energy X-ray absorptiometry and bioelectrical impedance analysis enabling assessment. Beyond mechanical factors, metabolic, structural, and psychological mechanisms can link adiposity and pain, underscoring the importance of understanding how body fat influences musculoskeletal health [4].

Mechanical Loading and Joint Degeneration

Obesity is acknowledged as a leading risk factor for the development of musculoskeletal disorders [2]. Obesity affects mechanical loading in the skeletal system, leading to an increase in joint osteoarthritis, particularly in the lower back, lower limb, and upper extremity [3]. Internal joint degeneration, linked through the healthy aging loading mechanism, is also accelerated due to the mechanical loading instigated by obesity [1]. Obesity in the population increases the risk of developing knee osteoarthritis [4]. The contour of the frontal plane loading angle prevails the same in overweight and normal-weight individuals. A weight-dependent corridor of knee joint motion is introduced so that increased loading can be accommodated [5].

Inflammatory Pathways and Adipokines

A variety of inflammatory mediators influence the link between body mass and musculoskeletal health [1]. For example, obesity promotes synovial inflammation and increases systemic concentrations of leptin, a protein secreted by adipose tissue that acts on the hypothalamus and peripheral tissues to regulate energy metabolism. Leptin also enhances the expression of IL-6 by chondrocyte–synovial fibroblast interactions within articular cartilage, thereby contributing to metabolic-related joint disorders such as osteoarthritis [6]. Adipose tissue likewise produces adipokines, bioactive peptides, immune molecules, and mediators of inflammation that modulate inflammatory processes directly through autocrine, paracrine, and endocrine pathways [7]. Such mediators exert significant effects on the pathogenesis of both rheumatoid arthritis and osteoarthritis, affecting diverse tissues such as cartilage, synovium, and bone. In patients with osteoarthritis, body mass index represents an established risk factor, where increasing body mass correlates with a greater likelihood of disease development [4]. In addition, serum concentrations of leptin, one of the aforementioned adipokines, exhibit positive associations with osteoarthritis risk and severity [7]. Although evidence remains less robust, alterations in the production of such mediators by adipose tissue in rheumatoid arthritis may influence immune activation, joint damage, and disease progression [8]

Metabolic Comorbidities and Skeletal Homeostasis

The effects of obesity on skeletal tissues and peripheral metabolic homeostasis depend on the presence and severity of obesity-related metabolic comorbidities [7]. These include insulin resistance (IR), increased free fatty acids and triglycerides, and plasma lipid abnormalities leading to dyslipidaemia [9]. Individuals with obesity and comorbidities show alterations in bone microstructure, mass, and strength indicative of compromised skeletal health; the relationship may be due to insufficient supply of skeletal-favourable adipose-derived factors for

maintaining skeletal homeostasis [10]. Obesity may result in differential effects on pericortical and trabecular compartments and distinct patterning of changes in parathyroid hormone (PTH) and bone remodelling markers. IR in obesity is associated with increased levels of circulating PTH and greater bone turnover, with greater increases in C-terminal telopeptides of type 1 collagen and osteocalcin observed in individuals with abdominal fat and metabolic syndrome [6]. Non-esterified fatty acids may exert local effects in osteoblasts and osteocytes through peroxisome proliferator-activated receptor γ, leading to enhanced apoptosis and diminished anabolic responses to mechanical loading. In individuals free from obesity-related metabolic disorders, alignment of obesity with positive skeletal health is proposed [5].

Musculoskeletal Outcomes across Body Regions

Epidemiological insights reveal that obesity prevalence varies across the spectrum of musculoskeletal (MSK) disorders, but certain site-specific patterns appear consistent. The findings derive from numerous studies that invariably report prevalence data on obesity, disease frequency, or both, alongside estimates of the average effect size of obesity [4]. The spine, particularly the intervertebral disc (IVD), represents a pivotal MSK region influenced by obesity. On average, 45% of obese cohorts report back pain, a proportion consistent with non-obese groups, yet disc degeneration incidence and radiographic alteration rates double among the obese. These discrepancies suggest that compressive loading associated with increased body mass acts in concert with other factors in obesity-related back conditions [1, 3]. In the hip and knee joints, degenerative diseases rank among the most prevalent MSK conditions [2]. Despite modifications in biomechanical loading, the course of osteoarthritis (OA) is confounded by a series of obesity-associated structural changes [2]. There is a prevailing outlook within the literature that the knee joint represents a critical region for obesity-related alterations; structural deterioration differs markedly from the hip, and OA progression is swifter, leading to greater mobility loss. Reports of peripheral joint involvement also arise frequently [3]. Despite altered gait patterns, the ankle and foot joints remain subject to the repercussions of increased loading and consequently display a higher susceptibility to osteoarthritis and other degenerative conditions. Determining the isolation of axial versus peripheral joints thus becomes a challenge [4]. With respect to muscular conditions, obesity exerts effects on muscle quantity, quality, and strength. Sarcopenic obesity emerges in this context as a clinically relevant manifestation of age-related degeneration, particularly among the elderly [5].

Spine and Intervertebral Health

Obesity is associated with accelerated degeneration of the lumbar spine intervertebral discs. Increased body mass alters loading patterns 11 and influences nutrient transport to spinal tissues [12]. Obesity also increases the likelihood of degeneration of adjacent segments after spinal surgery. Intervertebral disc degeneration is associated with significant pain, impairment, and loss of function. Consistent with the general population, obesity is a risk factor for back pain, which is one of the most widespread chronic pain complaints in the United States [10].

Hip and Knee Joints

Obesity is a robust, unstable, and increasing risk factor for musculoskeletal health. Epidemiological studies observe a rising trend in overweight and obesity among youths and adults [10]. There is a prevalence of more than 23% in youth and greater than 65% in individuals aged 20 to 70 years. Double and triple the incidence risk of musculoskeletal disorders is noted among overweight and obese individuals [11]. The life-long hazard rate of at least one of the reported conditions reaches nearly 80% for the population, with a quadrupling risk from obesity. Recent decades have seen a noticeable increase in both the prevalence of obesity and the incidence of musculoskeletal disorders. The lifetime risk has increased by approximately 20% since 1990 [1]. Patients with obesity show a spectrum of knee joint problems that are episodic throughout their lives and may be underreported. As many as 80% of people with clinically apparent knee osteoarthritis (OA) have a body mass index above 25 kg/m2, and more than half report difficulty[14]. Knee OA progresses more rapidly among middle-aged individuals with obesity compared to those with a normal weight, and the presence of obesity is associated with greater structural deterioration after intervention. Loading in normal is substantially greater for the knee than for joints in the hip or spine, and loss of knee function paramount condition to consider when addressing these double burdens [15].

Ankle, Foot, and Non-Axial Joints

Obesity is strongly associated with foot and ankle musculoskeletal disorders. Osteoarthritis in the foot and ankle affects approximately 30,000 patients annually in the UK. Excess weight exacerbates foot pain owing to significant increases in joint forces [13]. Obesity contributes to gait problems and alters the biomechanics of ankle and foot joints [5]. Increased calf and plantar muscle fibrotic contents affect ankle stability in obese subjects, linked to myoarchitectural changes that support the ankle mortises. These alterations are accompanied by flat feet, reduced motion range, and elevated plantar pressures, and unequal joint loading, which predispose subjects to foot stress. Weight reduction is advised as a first-line approach to prevent these changes, which can significantly affect quality of life [14]. Changes in body mass directly affect non-axial joints via mechanically induced loading and kinetic

alterations during locomotion. As tissue loads increase, the risk of degenerative joint disease rises. Modelling demonstrates that excessive loading can trigger biodynamics and disease development at peripheral articulations rather than the main axial joints [12].

Muscle Function and Sarcopenic Obesity

Sarcopenic obesity is characterized by excess fat accumulation in the context of poor muscle quality and mass 15. It is associated with inadequate muscle strength and physical performance [16]. Although definitions vary widely, cross-sectional studies analyzing participants aged 55 or older report a sarcopenic obesity prevalence of 2% between the ages of 60 and 69 and 10% above 80[13]. The condition raises the risk of metabolic disorders related to excessive adipose tissue, elevating the likelihood of coronary artery disease, type 2 diabetes, hypertension, and certain cancers [13]. Beyond direct morbidities, sarcopenic obesity negatively affects functional independence and quality of life.

Obesity, Physical Activity, and Rehabilitation Considerations

Excess body weight directly impacts the musculoskeletal system, yet intervention strategies depend on the population's capacity to engage in physical activity [11]. To improve adherence to recommendations, evidence outlining individual responses and physical activity profiles during weight-loss interventions must be synthesized. Exercise has beneficial effects on pain and function, and its intensity can influence outcomes. Exercise type, intensity targets, and physical-capacity evaluations for specific activities all shape intervention strategies [13]. At low intensities, wheelchair propulsion and cycling yield superior adaptations, while at higher intensities, less-trained individuals derive greater benefits from walking [17]. While weight loss after bariatric surgery improves pain, structural deterioration remains, indicating the necessity of physical activity for musculoskeletal health before and after surgery [18]. Exercise influences physiological systems related to musculoskeletal health and fosters the maintenance of function. Adherence, independent of the volume of activity, is pivotal for sustained adaptations. Safety data to support resilience, pre- and postintervention recommendations, and feasible targeting methods, all supplemented by high-quality physiological profiling, facilitate tailored interventions [10].

Exercise Interventions and Musculoskeletal Outcomes

Overweight and obesity are important public health issues that affect a large part of the population and have serious implications for musculoskeletal health [18]. Numerous research studies have evaluated the relationship between obesity, physical activity, and rehabilitation [13]. The findings on the effects of exercise interventions on musculoskeletal outcomes in older adults across existing literature indicate that type (e.g., cardiovascular, resistance, aquatic), intensity (moderate, vigorous), and additional components (e.g., stability, flexibility) do not markedly alter self-reported pain, knee joint space width, or cartilage volume [19]. Nonetheless, supervised exercise programs generally achieve higher adherence rates than unsupervised routines. Guidelines recommend moderate-intensity exercise three to five times weekly for >150 minutes total duration and isocaloric weight maintenance in conjunction with physical activity; these principles apply to both the general and obese older adult population [12]. Engagement in supervised physical activity is crucial for adults with obesity [14]. Establishing community exercise and educational programs focusing on the relationship between obesity, physical activity, and musculoskeletal conditions, alongside program assessment of the effect of rehabilitation on the dynamics of these relationships, is essential[12].

Weight Management and Surgical Implications

Obesity remains a significant risk factor for surgery, complications, and poor postoperative outcomes. Obese patients who undergo bariatric surgery or other weight-loss procedures have higher perioperative risks, increased complications, and greater costs associated with the surgical procedure [20]. Weight loss before knee replacement surgery improves surgical safety and can lead to better postoperative outcomes [18]. Nevertheless, total knee arthroplasty, a highly effective treatment for end-stage osteoarthritis, remains a viable surgical option for patients with severe symptoms and high body mass index when accompanied by discussions about potential complications and the importance of preoperative weight loss [21].

Nutritional and Pharmacological Considerations

Obesity can negatively affect musculoskeletal health, but key nutritional and pharmacological strategies may help promote healthy cartilage and joint function [20]. In weight-loss interventions, 500 to 1,000 kcal caloric restriction combined with regular exercise is most beneficial for joint health [15]. Micronutrients also play a role in maintaining musculoskeletal health; vitamin D, calcium, and omega-3 fatty acids are frequently studied [21]. Several compounds with anti-inflammatory potential are also under investigation to decrease obesity-related pain.

Methodological Considerations in the Literature

Limiting the search to observational studies would help minimise systematic reviews that are more exploratory and take a wider view of the field [13]. Identifying specific, highly relevant variables and searching a defined subset of databases could aim to maximise the number of systematic reviews [17]. The search strategy could stipulate 'obesity' as the core exposure of interest or indicate a preferred focus, as strictly prohibiting the term may

exclude pertinent reviews. Obesity may influence the risk of musculoskeletal disorders both directly and indirectly. Elevated bodyweight increases the risk of developing osteoarthritis, especially in weight-bearing joints [13]. Conversely, musculoskeletal disorders can lead to a reduction in physical activity, bringing about weight gain or inhibiting weight loss. Addressing only one of these facets thus often results in an incomplete picture [15]. The wide array of comorbidities associated with obesity confounds the pursuit of a clear linkage between weight and musculoskeletal disorders. A more granular examination of obesity and its putative effects is warranted, therefore, rather than one that is constrained by consideration of related comorbidities [13].

Study Designs and Measurement Approaches

Epidemiological research typically adopts either an observational or interventional design. Observational studies assess the relationship between one or more determinants and a disease outcome [15]. For an exposure such as obesity, various assessment methods may be employed, including individual, aggregate, or modelled data, each of which has inherent strengths and weaknesses [16]. In contrast, interventional studies manipulate a determinant and measure its influence on an outcome. Within such a framework, randomised controlled trials command the highest regard, but alternative approaches such as non-randomised experimental studies or controlled before-and-after studies are also viable options. Due to practical constraints, however, the studies on the obesity–musculoskeletal health nexus tend to be observational and cross-sectional in nature, with limited capacity to investigate longitudinal interdependencies [22]. Bias and confounding variables may further complicate the interpretation of results from a single-determinant cross-sectional study. Although multiple factors can influence musculoskeletal health independently of obesity, few of these determinants demonstrate an association with weight on the same population scale [16]. For instance, reduced physical activity correlates more strongly with obesity than does income, and exposure to cold climates has likewise been associated with sizeable changes in body mass. Within the obesity literature itself, socioeconomic status has emerged as a considerable confounder, with stronger associations reported for high-income countries than for lower-income counterparts [17]. Homeostatic factors and other chronic conditions may also exert concurrent influences on both obesity and musculoskeletal health.

Confounders, Bidirectionality, and Bias

Obesity is frequently linked with an increased incidence or prevalence of diverse musculoskeletal (MSK) disorders such as osteoarthritis (OA), rheumatological conditions, and low back pain [19]. These associations are often interpreted as evidence supporting a causal relationship, wherein obesity is viewed as a risk factor for poor MSK health [22]. Nevertheless, care must be taken in inferring directionality from such findings, as the observed associations may instead arise from joint disorders causing limited physical activity, leading to weight gain. This possibility is particularly salient within the rheumatological context, wherein active disease or disability may hinder both movement and weight control [22]. To date, few studies have considered this bidirectionality explicitly or adjusted for potential confounders beyond standard demographic factors [20]. Davis et al. modelled longitudinal data from the U.S. National Health and Nutrition Examination Survey and identified four prevalent MSK disorders whose presence increased the odds of future obesity; adiposity and wider adiposity distributions at baseline predicted future impairment in joint and limb mobility, finger dexterity, and household job difficulty. Consequently, assessment of the two-way relationships between obesity and diverse musculoskeletal disorders remains a priority [23].

Gaps in Knowledge and Future Directions

Research concerning the relationship between obesity and musculoskeletal health is still somewhat limited. Compared to well-established links between obesity and cardiovascular or type 2 diabetes diseases, fewer studies have focused specifically on musculoskeletal outcomes. Furthermore, existing literature has not provided a complete overview of the relationship; specific body regions affected by obesity other than the ankle, knee, and spine remain relatively unexplored. The apparent bidirectionality of the association has also prompted interest in designing obesity-focused interventions and studying their effects on musculoskeletal health [18]. There has been increasing concern that prevalent glucocorticoid use for other pathologies places patients at heightened risk for subsequent fractures; 21 this is an increasing focus of research despite the frequent tendency to consider osteoporosis and its associated therapies in incompletely isolated terms [19]. Furthermore, detailed exploration of micronutrients and adjunctive therapies implicated in either deterioration or maintenance of metabolic or microcirculatory tonus has generally been overlooked in existing review work. Comprehensive overviews of the full range of conditions mediated either directly or indirectly through obesity have yet to emerge in a similar form [20]. Mild to moderate obesity is associated with the metabolic syndrome, sarcopenic obesity, poor joint health, and various articular aftereffects consequent upon trauma [19]. Those affecting appendicular joints at long bones are less thoroughly documented in the general literature. Existing scientific models of the metabolic syndrome in obese human populations remain undercited for the modelling of adiposity, such as the isolation of adipokines effects via adipotoxic and microcirculatory modeling [20-24]. The effect of human upper-body obesity on specific

stiffness parameters of the intervertebral disc attracts very little dedicated investigation [22]. No models develop all possible axial-joint-associated obesity pathways in cycles, with inclusion of the appendicular skeleton. A shortage of studies addresses a wide range of metabolic/vascular disorders surrounding glucocorticoid administration and mediation between inflammatory, necrotic, and degenerative joint conditions. Experimental work upon age segregation remains limited and without a link to a full joint-surrounding pathway framework [25-29].

Implications for Clinical Practice and Policy

Obesity is strongly associated with the deterioration of skeletal self-organization, chiefly influencing musculoskeletal diseases and jeopardizing overall health [2]. These observations extend beyond pure mechanical effects: excess weight disorders further disturb biological systems, strain homeostasis, and alter the synchronization of the organism [4]. Mechanical, inflammatory, and metabolic pathways converge on musculoskeletal health, fueling the onset and aggravation of prevalent disorders. Guidelines on obesity treatment for the general public indirectly apply to rehabilitation systems, addressing risk factors for larger populations. The relevance of measures for the prevention and recovery of physical activity is particularly clear in rehabilitation following musculoskeletal disorders, which, alongside natural ageing, constitute key risk factors driving the emergence of obesity complications [19]. While osteoarthritis remains a predominant condition in contemporary literature, many other prevalent musculoskeletal disorders associated with weight are also observed.

CONCLUSION

Obesity profoundly affects musculoskeletal health through mechanical, metabolic, and inflammatory pathways that interact in complex and mutually reinforcing ways. Excess body weight increases mechanical loading on weight-bearing joints, accelerating degeneration in the knee, hip, spine, ankle, and foot. However, the impact of obesity extends well beyond biomechanical stress: adipose-derived cytokines and adipokines contribute to synovial inflammation, cartilage degradation, altered bone turnover, and chronic pain. Metabolic comorbidities such as insulin resistance and dyslipidaemia further disrupt skeletal homeostasis, while sarcopenic obesity diminishes muscle strength and functional capacity, heightening the risk of disability. Importantly, the relationship between obesity and musculoskeletal disorders is bidirectional. Musculoskeletal pain and joint dysfunction often reduce physical activity, promoting weight gain and perpetuating a cycle of declining functional ability. Although exercise and weight-loss interventions demonstrate consistent benefits, many studies highlight the need for individualized rehabilitation strategies, greater adherence support, and improved assessment of body composition rather than relying solely on BMI. Evidence also underscores the importance of integrating nutritional optimization, anti-inflammatory therapies, and preoperative weight management into clinical care pathways. Despite advances in understanding obesity-related musculoskeletal disease, significant knowledge gaps remain. These include limited longitudinal data, inconsistent definitions of obesity phenotypes, insufficient analysis of non-axial joints, and underexplored metabolic and vascular pathways, particularly those involving adipokines, glucocorticoids, and age-related changes. Future research must adopt comprehensive, multidimensional models that account for mechanical, metabolic, and inflammatory factors simultaneously. Overall, addressing obesity is essential for preventing and managing musculoskeletal disorders, improving quality of life, and reducing long-term healthcare burdens. Clinicians, researchers, and policymakers must work collaboratively to promote early intervention, integrate structured physical activity and rehabilitation into care, and develop public health strategies that target obesity as a modifiable risk factor for musculoskeletal decline.

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