OBESITY AND SKELETAL FRAGILITY IN AGING POPULATIONS: REVISITING THE OBESITY-OSTEOPOROSIS PARADOX

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ABSTRACT

Introduction: Obesity and osteoporosis are increasingly coexisting conditions in aging populations. Traditionally, obesity has been viewed as protective against osteoporosis, given that a higher body mass index (BMI) is linked to increased bone mineral density (BMD). However, emerging evidence calls this assumption into question, suggesting that excess adiposity may negatively impact skeletal health, even in the presence of elevated BMD. ***Methods:*** This narrative review synthesizes current evidence regarding the relationship between obesity and skeletal health in middle-aged and older adults. Relevant literature was identified through structured searches in PubMed, Scopus, and Web of Science, focusing on studies evaluating obesity, body composition, bone mineral density, fracture risk, and sarcopenic obesity. ***Results:*** Although obesity is generally associated with higher BMD, accumulating epidemiological evidence indicates that fracture risk in obese individuals is heterogeneous and often site-specific. Visceral adiposity, chronic low-grade inflammation, and bone marrow adiposity may impair bone microarchitecture and bone quality despite preserved bone density. In addition, sarcopenic obesity, characterized by excess fat mass combined with reduced muscle mass, has emerged as an important contributor to skeletal fragility through increased fall risk and impaired functional mobility. ***Discussion and Conclusion:*** These findings highlight a complex interaction between adipose tissue, muscle, and bone metabolism. Reliance on BMI or BMD alone may therefore underestimate fracture risk in obese individuals. Obesity should not be considered uniformly protective against skeletal fragility in aging populations. A comprehensive evaluation of body composition, bone quality, and functional status is essential for accurate fracture risk assessment in aging populations.

Keywords: Osteoporosis; Bone mineral density; Fracture risk; Visceral adiposity; Sarcopenic obesity

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I. INTRODUCTION

Obesity and osteoporosis are two major global health challenges that increasingly coexist in the context of rapid population aging worldwide. Over the past several decades, the prevalence of obesity has risen substantially across the globe. According to the World Health Organization, more than one billion people worldwide are currently living with obesity, and this number continues to increase in both developed and developing countries. At the same time, osteoporosis remains one of the most common metabolic bone diseases affecting middle-aged and older adults, and it represents a leading cause of fragility fractures, reduced quality of life, disability, and increased mortality among elderly individuals. Osteoporotic fractures are now recognized as a significant burden on global healthcare systems, particularly as life expectancy continues to rise [1],[2].

Osteoporosis has long been regarded as a prototypical disease of aging, characterized by decreased bone mass, deterioration of bone microarchitecture, and increased skeletal fragility. Recent international epidemiological syntheses suggest that the prevalence of osteoporosis ranges from approximately 10–30% among women aged 40 years and older and around 10% among men, with prevalence increasing markedly with advancing age and being consistently higher in women. Another global meta-analysis further emphasizes that osteoporosis represents one of the most prevalent metabolic bone disorders in older adults. Although its distribution varies across geographic regions, osteoporosis is widely recognized as a major contributor to pain, reduced mobility, diminished quality of life, and increased risk of fragility fractures. In geriatric clinical practice, the consequences of osteoporosis extend far beyond fractures themselves. Fragility fractures frequently trigger a cascade of adverse outcomes, including functional decline, loss of independence,

increased need for long-term care, and elevated mortality, particularly in patients with hip fractures and vertebral fractures [3].

Traditionally, obesity has been considered a protective factor against osteoporosis. This concept largely stems from epidemiological observations indicating that individuals with higher body mass index (BMI) tend to have higher bone mineral density (BMD) than those with normal or lower BMI. Increased body weight imposes greater mechanical loading on the skeleton, thereby stimulating bone formation through biomechanical pathways. In addition, adipose tissue has increasingly been recognized as an active endocrine organ that regulates bone metabolism through several hormonal mechanisms. These mechanisms include the peripheral conversion of androgens to estrogens within adipose tissue, which may contribute to reduced bone loss, particularly in postmenopausal women [4].

From this perspective emerged the so-called "obesity paradox" in osteoporosis: individuals with obesity may exhibit higher BMD but do not necessarily have a lower risk of fractures. Accumulating evidence in recent years suggests that the relationship between obesity and fracture risk is complex and influenced by anatomical site, sex, and metabolic context.

Despite the growing body of literature investigating the relationship between obesity and bone health, substantial gaps in knowledge remain. Many previous studies have relied on BMI as a surrogate marker of obesity. However, BMI cannot distinguish between fat and lean mass, nor does it adequately reflect body fat distribution. Therefore, a comprehensive synthesis of existing scientific evidence is necessary to clarify better the relationship between obesity and osteoporosis in middle-aged and older adults.

Accordingly, this review aims to synthesize current scientific evidence on the relationship between obesity and skeletal health, with particular emphasis on BMD, fracture risk, fat distribution, and obesity's role in skeletal fragility.

II. MATERIALS AND METHODS

2.1. Study design

This study was conducted as a narrative review to synthesize and critically discuss current evidence on the relationship between obesity and osteoporosis in middle-aged and older adults. Unlike systematic reviews, narrative reviews allow a broader conceptual analysis and integration of heterogeneous evidence; however, to ensure transparency and methodological rigor, a structured literature search and selection strategy was applied.

The review focused on epidemiological, clinical, and mechanistic studies investigating the association between obesity, body composition, and bone health outcomes, including BMD, bone microarchitecture, and fracture risk in aging populations.

2.2. Literature search strategy

A comprehensive literature search was performed in several major biomedical databases, including PubMed/MEDLINE, Scopus, and Web of Science. Additional relevant articles were identified through manual searches of reference lists of key publications and relevant review articles.

The search strategy combined keywords and Medical Subject Headings (MeSH) related to obesity and bone health. The primary search terms included: "obesity", "osteoporosis", "bone mineral density", "fracture risk", "aging", "older adults", "body composition", "visceral fat", "sarcopenic obesity".

The search primarily focused on studies published between 2000 and 2025, with priority given to research from the past 10–15 years to capture recent advances in understanding obesity–bone interactions.

2.3. Eligibility criteria

Studies were included in the review if they met the following criteria: (i) Studies conducted in human populations, particularly middle-aged or older adults (generally ≥40 years); (ii) Studies investigating the association between obesity, adiposity, or body composition and bone health outcomes such as BMD, osteoporosis, bone quality, or fracture risk; (iii) Observational studies (cross-sectional, cohort, or case-control studies), clinical studies, systematic reviews, or meta-analyses; (iv) Articles published in peer-reviewed journals and written in English.

Studies were excluded if they met one or more of the following conditions: (v) Animal or in vitro studies; (vi) Studies focusing exclusively on pediatric populations; (vii) Conference abstracts without sufficient methodological detail; (viii) Editorials or opinion papers lacking original data or comprehensive analysis.

2.4. Data synthesis

The selected studies were analyzed and synthesized using a thematic approach to facilitate integration of evidence from heterogeneous study designs. The literature was categorized into several major domains reflecting key aspects of the relationship between obesity and skeletal health.

The thematic domains included: (i) The association between obesity and BMD; (ii) The relationship between obesity and fracture risk; (iii) The influence of body fat distribution on bone health; (iv) The role of sarcopenic obesity in skeletal fragility; (v) Biological mechanisms linking adipose tissue, muscle, and bone metabolism.

This approach allowed the integration of epidemiological, clinical, and mechanistic evidence to provide a comprehensive overview of the complex interactions between obesity and osteoporosis in aging populations.

III. RESULTS

3.1. Obesity and bone mineral density

A substantial body of epidemiological and clinical evidence consistently demonstrates a positive association between obesity and BMD. Across adult populations, individuals with higher BMI generally exhibit higher BMD at major skeletal sites, particularly the lumbar spine, femoral neck, and total hip. This association has been observed in multiple cross-sectional and cohort studies and appears to be particularly pronounced at weight-bearing skeletal sites where mechanical loading is greatest.

The most widely accepted explanation for this relationship is the mechanical loading effect. Increased body mass imposes greater mechanical stress on the skeleton, thereby stimulating bone formation through mechanotransduction pathways and promoting osteoblast activity. Consequently, higher body weight may contribute to increased bone mass and partially explain the positive correlation between BMI and BMD reported in numerous observational studies.

In addition to biomechanical influences, endocrine mechanisms related to adipose tissue may also contribute to this association. Adipose tissue functions as an active endocrine organ that secretes hormones and cytokines capable of influencing bone remodeling. Peripheral aromatization of androgens to estrogens in adipose tissue may reduce bone resorption, particularly in postmenopausal women, thereby contributing to the preservation of bone mass.

However, emerging evidence suggests that the relationship between obesity and BMD may be more nuanced when body composition is considered. Several studies indicate that lean mass may be a stronger determinant of BMD than fat mass, highlighting the important role of muscle–bone interactions in skeletal health. These findings suggest that the positive association between BMI and BMD may partly reflect the contribution of greater muscle mass rather than adiposity alone.

3.2. Obesity and fracture risk

Despite the generally positive association between obesity and BMD, fracture risk among obese individuals is not consistently reduced. Increasing epidemiological evidence suggests that the relationship between obesity and fracture risk is heterogeneous and strongly site-specific.

Large population-based studies have shown that obesity may be associated with a reduced risk of fractures at some skeletal sites, particularly the hip, but an increased risk at other locations such as the humerus, ankle, and vertebrae. These findings have led to the concept of the "obesity paradox" in skeletal health, whereby individuals with obesity may exhibit higher BMD yet remain vulnerable to fragility fractures.

Several mechanisms have been proposed to explain this paradox. First, obesity is frequently associated with reduced mobility, impaired balance, and altered gait mechanics, which may increase the likelihood of falls. Second, increased body mass may alter fall biomechanics, influencing the distribution of impact forces and leading to site-specific fracture patterns.

In addition, metabolic disturbances associated with obesity may adversely affect bone microarchitecture and bone quality despite preserved BMD. Chronic low-grade inflammation, insulin resistance, and dysregulated adipokine signaling have all been implicated in the disruption of bone remodeling processes. These factors may compromise bone strength independently of BMD, thereby contributing to the observed discrepancy between BMD and fracture risk in obese individuals.

3.3. Body fat distribution and bone health

Recent advances in body composition research suggest that fat distribution, rather than total body fat alone, plays a critical role in determining skeletal outcomes. Visceral adipose tissue (VAT), which accumulates within the abdominal cavity, is metabolically active and strongly associated with systemic inflammation.

VAT secretes pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), which can stimulate osteoclastogenesis and promote bone resorption. Chronic low-grade inflammation associated with visceral adiposity may therefore contribute to the deterioration of bone microarchitecture and reduced skeletal strength.

Several studies have demonstrated that increased visceral fat is associated with alterations in bone metabolism and reduced bone quality even in the presence of normal or elevated BMD. Furthermore, visceral adiposity is closely linked to metabolic disorders such as type 2 diabetes mellitus, a condition characterized by increased fracture risk despite normal or increased BMD.

In contrast, subcutaneous adipose tissue appears to exert distinct metabolic effects and may have a less detrimental impact on bone metabolism than visceral fat. Some evidence suggests that subcutaneous fat may exert neutral or modestly protective effects on bone through mechanical loading and endocrine mechanisms. These findings highlight a key limitation of BMI as an indicator of obesity-related skeletal risk. Because BMI does not distinguish between fat distribution patterns or body composition components, it may inadequately capture the complex relationship between adiposity and skeletal fragility.

3.4. Sarcopenic obesity and skeletal fragility

An emerging concept that has received increasing attention in recent years is sarcopenic obesity, defined as the coexistence of excess adiposity and reduced skeletal muscle mass or strength. This phenotype is particularly relevant in aging populations because skeletal muscle plays a critical role in maintaining both skeletal integrity and functional mobility.

Epidemiological studies indicate that individuals with sarcopenic obesity experience worse clinical outcomes than those with obesity or sarcopenia alone, including impaired physical performance, increased disability, and a higher risk of falls.

The interaction between muscle and bone is mediated through both mechanical and biochemical mechanisms. Muscle contractions generate mechanical loading that stimulates bone formation, while muscle-derived signaling molecules (myokines) influence bone remodeling processes. Consequently, loss of muscle mass and strength may reduce skeletal loading and contribute to bone fragility.

When obesity coexists with sarcopenia, the potential mechanical benefits of increased body weight may be offset by impaired mobility and reduced muscle strength. Individuals with sarcopenic obesity often demonstrate slower gait speed, poorer balance, and a higher incidence of falls compared with individuals with obesity alone. These functional impairments may substantially increase the risk of fragility fractures in older adults.

Taken together, these findings underscore the importance of considering the integrated fat–muscle–bone axis when evaluating skeletal health in aging populations. Comprehensive assessment of body composition, muscle strength, and functional performance may therefore provide a more accurate evaluation of fracture risk than reliance on BMI or BMD alone.

Key epidemiological and mechanistic studies examining the relationship between obesity and skeletal health are summarized in Table 1.

Table 1. Key epidemiological and mechanistic studies examining the relationship between obesity and skeletal health.

Author (Year)	Title	Exposure	Outcome	Key Findings
Rosen CJ et al. (2006)	Mechanisms of disease: Is osteoporosis the obesity of bone?	Adipose tissue and bone interaction	Bone metabolism	Proposed the conceptual link between adiposity and bone remodeling, highlighting the role of adipocytes, bone marrow fat, and endocrine signaling in skeletal metabolism
Zhao et al. (2007)	Relationship of obesity with osteoporosis	BMI, fat mass	BMD	Positive correlation between BMI and BMD
Binkley et al. (2009)	Beyond FRAX: it's time to consider "sarco-osteopenia"	Sarco-osteopenia	Fracture risk	Interaction between sarcopenia and bone fragility
Premaor et al. (2010)	Obesity and fractures in postmenopausal women	BMI	Fracture risk	Obesity is associated with an increased risk of ankle and humerus fractures
Compston et al. (2011)	Obesity is not protective against fracture in postmenopausal women: GLOW	BMI	Fracture incidence	Obesity is not protective against fractures
Schwartz et al. (2011)	Association of BMD and FRAX score with risk of fracture in older adults with type 2 diabetes	BMD, diabetes	Fracture risk	Higher BMD in diabetes, but increased fracture risk
Shapses et al. (2012)	Bone metabolism in obesity and weight loss	Obesity	Bone metabolism	Obesity influences bone through endocrine and mechanical pathways
Johansson et al. (2014)	A meta-analysis of the association of fracture risk and body mass index in women	BMI	Fracture risk	High BMI reduces hip fracture risk but increases others
Tagliaferri et al. (2015)	Muscle and bone, two interconnected tissues	Muscle mass	Bone metabolism	Muscle–bone interaction is important for skeletal health
Scott et al. (2016)	Associations of sarcopenic obesity and dynapenic obesity with bone mineral density and incident fractures over 5-10 years in community-dwelling older adults	Sarcopenic obesity	BMD, fractures	Sarcopenic obesity increases fracture risk
Bredella (2017)	Sex differences in body composition	Body composition	Bone health	Fat distribution influences skeletal outcomes
Batsis et al. (2018)	Sarcopenic obesity in older adults: Aetiology, epidemiology and treatment strategies	Sarcopenic obesity	Aging outcomes	Combined obesity and sarcopenia increase disability risk
Cruz-Jentoft et al. (2019)	Sarcopenia: revised European consensus on definition and diagnosis	Sarcopenia	Muscle loss	Updated definition of sarcopenia
Clynes et al. (2020)	The epidemiology of osteoporosis	Osteoporosis epidemiology	Fractures	Major burden in aging populations
Gkastaris et al. (2020)	Obesity, osteoporosis and bone metabolism	Obesity	Bone metabolism	Complex interaction between adiposity and bone

Author (Year)	Title	Exposure	Outcome	Key Findings
Hou et al. (2020)	Obesity and bone health: A complex link	Obesity	Bone health	Adipose tissue influences bone remodeling
Rinonapoli et al. (2021)	Obesity and bone: A complex relationship	Obesity	Bone metabolism	Chronic inflammation affects bone quality
Salari et al. (2021)	Global prevalence of osteoporosis among the world older adults: A comprehensive systematic review and meta-analysis	Osteoporosis prevalence	Global burden	High prevalence in older adults
Turcotte et al. (2021)	Association between obesity and risk of fracture, bone mineral density and bone quality in adults: A systematic review and meta-analysis	Obesity	BMD and fracture	Obesity is linked to higher BMD but variable fracture risk
Kupisz-Urbańska et al. (2022)	Fracture risk in obesity: a narrative review	Obesity	Fracture risk	Fracture risk varies across skeletal sites
Piñar-Gutierrez et al. (2022)	Obesity and bone health: A complex relationship	Obesity	Bone health	Fat distribution influences bone fragility
Xiao et al. (2022)	Global, regional prevalence, and risk factors of osteoporosis according to the World Health Organization diagnostic criteria: A systematic review and meta-analysis	Global osteoporosis	Prevalence	Global osteoporosis prevalence is increasing
Arjunan et al. (2023)	Osteoporosis and obesity	Obesity	Osteoporosis	Obesity contributes to skeletal fragility
Chandran et al. (2023)	Prevalence of osteoporosis and incidence of related fractures in developed economies in the Asia Pacific region: A systematic review	Osteoporosis prevalence	Fractures	Osteoporosis burden rising in Asia-Pacific
Liu et al. (2023)	Obesity and risk of fracture in postmenopausal women: A meta-analysis of cohort studies	Obesity	Fracture risk	Obesity is associated with increased fracture risk
Gruneisen et al. (2024)	Fat as a friend or foe of the bone	Adipose tissue	Bone metabolism	Fat acts as both a protective and a harmful factor
Guo et al. (2024)	Obesity induces osteoimmunology imbalance: Molecular mechanisms and clinical implications	Obesity	Osteoimmunology	Obesity disrupts immune–bone interaction
Martiniakova et al. (2024)	Links among obesity, type 2 diabetes mellitus, and osteoporosis: Bone as a target	Obesity, diabetes	Bone metabolism	Metabolic diseases affect bone health
Luo et al. (2025)	The obesity paradox in osteoporosis risk among older adults is mostly driven by women: A population-based prospective study	Obesity	Osteoporosis risk	The obesity paradox is driven mainly by women

IV. DISCUSSION

The present review highlights the complex and multifactorial relationship between obesity and skeletal health in middle-aged and older adults. Although obesity is commonly associated with increased BMD, accumulating evidence suggests that higher bone mass does not necessarily translate into reduced fracture risk. This apparent discrepancy reflects the important distinction between bone density and bone quality, indicating that elevated BMD in obese individuals may not fully represent the structural integrity or mechanical strength of bone [5].

Several biological mechanisms may explain this paradox. Obesity is characterized by chronic low-grade inflammation, altered adipokine signaling, and metabolic disturbances such as insulin resistance, all of which can influence bone remodeling. These metabolic factors may impair bone microarchitecture and reduce bone strength despite preserved or increased BMD [4].

In addition, differences in adipose tissue distribution appear to play a critical role. Visceral adipose tissue, which is strongly associated with systemic inflammation and metabolic dysfunction, may exert more detrimental effects on skeletal metabolism than subcutaneous fat. Consequently, BMI alone may inadequately capture the relationship between adiposity and skeletal fragility [5].

The interaction between muscle and bone further complicates the obesity–osteoporosis relationship, particularly in aging populations. Sarcopenic obesity, characterized by excess adiposity combined with reduced muscle mass or strength, represents a high-risk phenotype associated with impaired mobility, increased fall risk, and greater skeletal fragility [6]. These findings emphasize that skeletal health in obese individuals cannot be understood solely by BMD measurements but requires consideration of body composition and functional status.

From a clinical perspective, reliance on BMD alone may lead to underestimation of fracture risk in obese patients. Current risk assessment tools, including Fracture Risk Algorithm (FRAX), incorporate BMI but do not fully account for variations in fat distribution, muscle mass, or bone quality [7]. Therefore, a more comprehensive approach that integrates body composition, muscle strength, and fall-risk assessment may improve fracture-risk assessment in older adults with obesity.

Weight management also represents an important consideration. Although weight loss improves cardiometabolic health, it may accelerate bone turnover and contribute to bone loss if not accompanied by adequate nutritional intake and resistance exercise. Thus, obesity treatment strategies in older adults should aim not only to reduce excess adiposity but also to preserve musculoskeletal health.

Several limitations should be acknowledged. As a narrative review, this study did not include a formal risk-of-bias assessment or quantitative synthesis of evidence. Furthermore, heterogeneity in the definitions of obesity and sarcopenic obesity across studies may contribute to variability in reported findings.

Overall, the current evidence suggests that obesity exerts complex and sometimes opposing effects on skeletal health. While increased body mass may contribute to higher BMD, metabolic alterations, fat distribution, and muscle loss may compromise bone quality and increase fracture risk. Understanding the integrated interaction between adipose tissue, muscle, and bone is therefore essential for improving fracture risk assessment and prevention strategies in aging populations.

V. CONCLUSION

Obesity should not be considered uniformly protective against osteoporosis in aging populations. Future research should prioritize longitudinal studies examining body composition, bone quality, and metabolic factors to understand better the mechanisms linking obesity and skeletal fragility.

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