Journal of Aging Research and Healthcare

Open Access - Peer-Reviewed - ISSN 2474-7785

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Review Article

Obesity and Type 2 Diabetes Impacts on Late Life Bone Fragility, Falls, and Fractures: A Narrative Review and Commentary

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Published citation: Ray Marks (2026) **Obesity and Type 2 Diabetes Impacts on Late Life Bone Fragility, Falls, and Fractures: A Narrative Review and Commentary**. *Journal of Aging Research and Healthcare* - 5(4):1-13. https://doi.org/10.14302/issn.2474-7785.jarh-26-6471

Article DOI: 10.14302/issn.2474-7785.jarh-26-6471

Abstract

Sound bone health and the ability to respond effectively to unexpected mobility obstacles that heighten falls risks are crucial for mediating many life affirming activities at all ages, but may be impacted adversely over the course of time by multiple factors, such as excess body weight and its link to type 2 diabetes. To better understand this issue which does not seem to be effectively addressed, the **PUBMED, PubMed Central, and GOOGLE SCHOLAR** data bases were sourced for relevant data published between January 1 2015 and November 1 2026 on these topics. These data show while a pervasive idea has been that obesity may be beneficial in some respects to bone mineral density attributes and fracture protection, several current publications tend to portray a negative impact of obesity on bone health via multiple proposed mechanisms and that continued clinical vigilance of vulnerable individuals both young and old are those with pre diabetes or actual existing type 2 diabetes who may have a high risk of sustaining falls and bone fractures.

Keywords:
Bone; Bone Mineral Density; Falls; Fractures; Obesity; Type 2 Diabetes

Article Information	
Running Title	aging bone, obesity and type 2 diabetes
Received Date	21 September 2026
Accepted Date	23 September 2026
Published Date	29 September 2026

Article Information (continued)

Online	29 September 2026
Publication	Volume 5; Issue 4; Pages 1–13
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Background

Bone fractures, as well as bone diseases such as osteoporosis, plus sarcopenia- denoting an age related muscle mass decline- as well as an increasing falls risk are enormously disabling public health challenges that prevail in most aging societies and at increasing rather than decreasing rates [1], [2], [3], [4], [5]. Among the many bone fracture causes that specifically prevail among most older adult populations is a possible role for obesity, a term denoting the presence of excess body fat, rather than being overweight due to the high presiding volumes of muscle mass, bone mass, or water, and one rapidly increasing in the United States and elsewhere that can thereby literally cause excess disability among millions of aging citizens, as well as children, even if initially modifiable.

In addition, the risk of falling may be heightened in aging adults who are overweight with a type 2 diabetes health profile, and in addition to fostering declines in functionality and independence as well as heightened morbidity and mortality rates, secondary injuries and recurrent falls incidents that may lead to one or more bone fractures and possibly other comorbid health conditions [5].

In turn, the presence of a fracture may exacerbate rather than diminish, a possible limited ability to function physically, thereby potentially expediting the acquisition or retention of excess body fat, as well as secondary bone structure deficits, muscle mass declines, despite the false belief that obesity has little impact on bone attrition. Moreover, type 2 diabetes-often caused by obesity is also a well known osteoporotic and falls risk factor [3].

Indeed, while the presence of excess body weight may impact bone mineral density favorably, bone fractures and the multiple attributes of bone destruction that preside in sizeable numbers of older adults as well as youth categorized as being obese, are strongly linked to the inability to offset falls and their injurious consequences, especially in the obese type 2 diabetes case, where chronic pain, depression, poor cardiovascular health, and possible resultant suboptimal motivation to move or exercise may weaken their ability to protect bones from succumbing to excess or unexpected mechanical forces or perturbations in their own right [4].

This situation, when viewed in both the short term and the long-term, and in the face of an increasingly apparent aging society with limited health resources, is likely to prove highly deleterious to the well being of many in general, as well as to their bone health, in particular, if protective efforts continue to be ignored, overlooked, misinterpreted, poorly understood, or understated. Moreover, even if the belief that having a large body size protects bone, rather than exposing it to damage, this belief appears inconsistent with the actual effect of excess body weight on bone during an impactful fall and possible associated muscle mass losses and others that render the falls excessively injurious in the case of bone fragility. Of specific concern is that efforts to mitigate obesity alone but that fail to appreciate its possible heightened link to falls and fracture impacts due to multiple interacting factors may fail to reduce the costs of one or more recurrent injurious falls and may continue to observe cases who have suffered from obesity in spite of pharmacologic mediated weight loss anti-obesity interventions. Moreover, in the type 2 diabetic case, that disease alone is one often subjected to the vicarious impact of medication induced hypoglycemia related declines in bone strength as well as unfavorable muscle mass and balance declines.

This current article updates our previous efforts to better understand what has been observed and concluded as regards the possible linkage of obesity to late life bone health status in the context of type 2 diabetes, and what it teaches us to be mindful of. It specifically examines if a case can be made for

arguing against blanket acceptance of the widespread notion that obesity protects an older adult and others from sustaining one or more bone fractures, especially at the hip, along with possible bone micro fractures that underpin painful disabling obesity linked osteoarthritis as well as type 2 diabetes.

Since the risk factors for adult obesity of poor diet and physical inactivity are well studied, as is the link between obesity and cardiovascular diseases, such as type 2 diabetes, this present review specifically sought to examine their potential theoretical or clinically relevant association that may persist even though weight loss medication strategies appear effective alongside those designed to control glucose levels. The time period 2015-2026 was chosen as the main focus, as it was felt this body of works may reflect some past as well as updated issues and insights that have emerged in the past decade and may have a bearing on health services allocation and delivery needs in the future as well as healthy aging policy directives.

In particular, this report investigates the recent evidence base broadly in support of a preventive rather than a management perspective using various drugs or injections to eliminate obesity and/or insulin resistance. The review does not examine some of the other leading causes of bone impairments in older or younger adults and does not examine studies in depth for their possible flaws or shortcomings. It was assumed that recent peer reviewed studies housed in **PUBMED, PubMed Central,** and **GOOGLE SCHOLAR** data bases using the key words: *bone health and adiposity/obesity, type 2 diabetes, falls, and fractues* would provide salient and valid points of current interest. The term obesity was used to denote any degree of excess body fat that could prove injurious to both a younger as well as an older person. The term bone health referred to the ability of the skeleton to withstand stress, and provide for pain free functional independence and optimal mobility and stability functions. The term sarcopenia refers to a decline in muscle mass and an increase in muscle fat mass, age or disease related, or both. Osteosarcopenia is the term denoting the impact of sarcopenia on bone mass attrition.

Research Question 1:

Is the combination of falls and bone attrition with age of concern in the context of the obese older adult suffering from type 2 diabetes?

Research Question 2:

Are there indications that more holistic preventive care approaches across the lifespan as well as more study in this realm will prove fruitful?

Underlying assumption

Sarcopenia, a health condition characterized by an age-related decline in muscle mass and strength, and a replacement of muscle tissue by fat, is being shown to raise the risk of falls, fractures, disability, and mortality in its own right and is frequently exacerbated by the presence of an obesity linked high muscle fat ratio that reduces bone protection and function [5]. Unsurprisingly, type 2 diabetes when induced by or co-occurring with obesity may well exacerbate an emergent sarcopenic state and its oftentimes multiple adverse health and wellbeing ramifications, thus contributing to the growing clinical challenges of high numbers of aging adults where these conditions when they exist or co exist are associated with an increased risk of injurious falls [6]. However, this is not a linear uni-directional associations or one readily reversed because weight reduction driven by artificial appetite suppression or lifestyle methods may not induce or reverse a predictable and desirable bone or muscle status in absence of continuous efforts to pursue health eating and/or exercise practices, plus any needed calcium, protein and vitamin D supplementation. Rather, a sole reliance on anti-obesity strategies may potentially compromise muscle health as well as bone health inadvertently and progressively [6], [7], [8], [9], [10].

This abovementioned argument is not just of theoretical interest, but one of immense relevance as societies age because these overlapping conditions are strongly associated with bone fragility [6], a strong fracture risk determinant in its own right [9], [11], as well as in cases with type 2 diabetes [12] and those with clear vitamin D deficiencies [7].

Osteosarcopenic obesity **,** which may stem from as well as further compromise physical function, metabolic health and overall life quality also increases the risk of injurious falls, one or more bone fractures and soft tissue injuries, possible prolonged hospitalizations, cardiometabolic complications

and premature mortality [5]. In women transitioning through menopause, declining estrogen levels that accelerate fat accumulation and muscle loss may be further compromised, while also heightening susceptibility to osteoporosis, hypertension, dyslipidemia, type 2 diabetes and certain cancers that are often worsened in the face of obesity [1]. A parallel vitamin D deficiency may likewise hasten progressive bone deterioration and muscle and adipose dysfunctions, and the osteosarcopenic obese phenotype is strongly correlated with an increasing risk of fragility fractures, injurious falls, functional and metabolic declines, plus higher morbidity and mortality rates [7].

Predominant research observations

Numerous publications over time that have attested to the key role of bone and bone health as a means of protecting vital body organs, and affording static and dynamic support to the human when upright, plus immune support, as well as serving as strategic sites for muscle tendons, ligaments and capsular attachments continue to show how lesions or deficits in bone significantly reduce the well being of the aged. Indeed, similar to other body organs, bone is a living tissue, but is not one immune or impervious to breakdown as a result of factors other than age. In terms of our examination of the specific theme of bone health and its association with obesity, a topic of increasing interest in this regard, and more so now in the face of the onset of bone fragility and the overall failure of medications and related options to avert this. We specifically elected to ascertain what link exists between obesity and bone health and its possible impact on fracture control in the elderly. Of interest was the validity of the premise that the impact of obesity on bone mineral density deposits is one favoring a bone protective response against sustaining a fracture in the case of a fall, although this is despite the negative influence of this belief and the strong belief in the application of anti-obesity medications or injections that control appetite to afford significant weight losses that thereby restores health including bone health. However, this does not accord with the data wherein bone fragility may well be magnified in the face of co existing type 2 diabetes, an osteoporotic mediator in its own right, and one where the control of glucose via pharmacologic approaches may independently impact bone mass negatively and progressively [12].

In addition, bone tissue houses important minerals and ions that can be released or stored in a controlled manner to provide constant concentrations in body fluids as required, and this relatively dynamic organ that undergoes significant turnover compared to other organs in the body may yet be affected negatively by multiple factors such as obesity, and its dual association with type 2 diabetes, even when these conditions appear to be effectively controlled or minimized using standard pharmacologic approaches. Indeed, despite an intuitive beliefs to the contrary, obesity and osteoporosis, a disease that is associated with progressive bone loss and fracture risk are not only both common chronic related disabling conditions that appear to be increasing over time rather than decreasing, but can readily occur in tandem, thereby engendering high rates of morbidity and mortality, especially in older adults with an osteoporosis diagnosis.

There is also increasing evidence that the relationship between obesity and bone may not be as protective against fractures as previously believed because not only may bone homeostasis in general be altered in multiple ways by the presence of excess body fat that can impair bone integrity and bone remodeling long before efforts to control weight are implemented but even after that [6]. It also appears that obese cases who may well be weaker and at risk for falls at higher rates than the non obese [6] and obesity in the form of visceral fat in particular, appears to harm bones collectively over time, possibly raising the risk of incurring osteopenia/osteoporosis especially in the older poorly nourished or inactive adult unless apprehended, and countered in a timely effective manner [7].

Thus, even if it is accepted that the presence of any excess body weight can help to build bone, there are several studies that highlight pathways whereby excess weight can yet pose an unanticipated bone fracture or bone damage risk at all ages. This is due in part to a possible obesity associated array of adverse inflammatory impacts on bone genes and bone cell physiology, as well as having diverse negative metabolic influences [8], for example in cases with type 2 diabetes who are obese [9], as well as a possible ensuing high muscle fat content, weakness, and reduced muscle mass [6].

Piñar-Gutierrez et al. [10] recount that the recent scientific evidence that has emerged in this regard and that shows an increased risk of fractures among cases deemed to obese and who have an especially high degree of visceral located fat is surely one of ongoing concern because this is increasingly common. One

reason for this may be that muscles in that region and others may be that may be infiltrated by excess fat may limit bone protection if a fall occurs as a result of possible deficient muscle reflex and suboptimal impact attenuation responses. Moreover, those who also suffer from obesity associated inflammatory responses and those who use diabetes medications that induce confusion, may foster a state of chronic repetitive joint injuries that may hasten the onset of osteopenia in its own right if sedentary behaviors become more entrenched in response to heightened pain and fears of falling [11].

Indeed, it appears multiple obesogenic-associated metabolic disturbances not only impact bone homeostasis negatively, but can affect bone quality detrimentally over time if the obese person is sedentary or less inclined to move than normal weight similar age individuals. They may thus exhibit various degrees of bone fragility, that progressively impact bone cellular processes as well as various alterations in local bone homeostasis and systemic metabolism [7] and metabolic disorders [12], especially if undetected or only detected when the condition is basically irreversible [13].

Hsu et al. [14] found that the risks of an individual acquiring osteoporosis, osteopenia, or nonspine fractures was significantly higher for subjects with a higher percentage of body fat independent of body weight, physical activity, and age. As such it appeared that having a high fat mass could impose a negative effect on bone mass that is different from that of normal weight-bearing. Other research has revealed that beneficial improvements in bone mineral density do occur in men with diabetes [15] or overweight status, along with an enhancement in bone strength, but other bone attributes were not examined. On the other hand, Lopez-Gomez et al. [17] found postmenopausal women who were deemed obese tended to show lower levels of bone formation markers than those deemed to be of a healthy weight. In addition, other data indicate bone quality in the obese case may not be sufficient to successfully avert a fragility fracture because other factors are involved [16]. For example, a role for muscle and possible sensory response changes cannot be discounted, along with a high risk of developing severe co-morbid health conditions that play a role in influencing bone quality [18]. Bone as well as various body systems may also serve as a 'fat-depot' that may mediate a range of obesity associated low-grade inflammatory responses that may not only alter the expression of multiple life negating cellular processes, but harmful molecules and chemicals that can adversely affect bone metabolism, and result in its attrition [19].

Another explanation is that since adipocytes and bone cells known as osteoblasts are derived from the same stem cell source, the presence of obesity may foster more adipocyte differentiation than osteoblast differentiation. These increased numbers of bone adipocytes are found to release a number of pro-inflammatory and immunomodulatory molecules that up-regulate the formation and activation of osteoclasts, a bone cell that removes excess bone, thus favoring bone fragility. As well, the persistent impact of excess body weight, while possibly favoring bone mineral increases, may yet exceed the overall mechanical threshold of the bone, especially in females if other obesity associated health conditions prevail that heighten fracture risk, such as dynapaenic abdominal obesity and/or altered bone blood flow [20], [21] despite any favorable body weight increases in bone mineral density [22]. Rather, this potentially greater risk of an injurious fall and chances of a bone fracture at one or more bone sites in the obese subject may involve an array of complex metabolic as well as neuromuscular, cognitive, and circulatory factors as well as related biomechanically mediated impacts and their interaction with bone viability as well as vulnerability acquired in the face of certain medications [24], [25], [26], poor diets, and adults exhibiting abdominal obesity [4], [20], [27].

According to Rikkonen et al [28], the presence of obesity is associated with the earlier than expected rate of sustaining a hip fracture injury, a serious debilitating injury associated with a higher than desired post fracture mortality rate and chances of developing complications following a fracture that may yet persist even in the face of successful bariatric surgery [29] or various glucagon uptake inhibitors in those with diabetes [34]. Moreover, this cycle may persist even in the face of semaglutide anti obesity interventions factors as a result of the unaffected or altered biomechanical factors associated with a high falls risk [32], as well as possible hormonal factors, and alterations in muscle protective responses, muscle strength capacity and fat ratio that can undermine inherent protective bone impact functions and hasten the emergence of osteoporotic fractures especially in the presence of excess or increasing muscle fat mass in the trunk and arm regions and despite weight reduction efforts [30], [32], [34], [35], [38], [41], [45].

Importantly, if a falls event occurs unexpectedly, the presence of obesity, especially that located in the abdominal area may yet disrupt any prevailing bone morphology via its mechanically induced inflammatory impacts and others [33], [34], and may be especially accentuated regardless of body mass distribution in those categorized as being morbidly obese [36], [39].

As argued by Crivelli et al. [37] when viewed as a whole, it seems fair to conclude both visceral and subcutaneous fat may be detrimental to bone health in pre- and postmenopausal women, as well as men and in both cases severe obesity may increase the risk for incurring a fall and subsequent fracture as well as poor healing trajectories thereafter, even in young adults.

Moreover, in view of what has been observed relative to the fat muscle ratio variable in elderly persons arm and trunk muscles [38], the belief that obesity confers protection against bone fractures must be challenged especially if other conditions affecting people with obesity, such as type 2 diabetes that can foster osteosarcopenia- a debilitating condition characterized by concurrent bone loss and muscle wasting, prevails [39].

Obesity is also associated with a possible vitamin D deficiency [7], as well as impaired muscle function, and sarcopenic phenotypes, and possibly thereby with a further fracture risk [41] and damage to both the bone itself and/or the surrounding soft tissue and its structures. Indeed, contrary to expectations, individuals diagnosed as being obese and having type 2 diabetes do appear to have substantive impaired bone quality alterations and an elevated risk of fragility fractures, despite their higher and/or unchanged bone mineral density [49] and often even in the face of efforts to alter bone cell metabolism [34].

Although the effect of obesity on fracture risk appears site-specific with a reduced risk for several fractures (e.g., hip, pelvis, and wrist) and increased risk for others (e.g., shoulder, ankle, upper leg, elbow, vertebrae, and rib), this may be linked to the degree to which body weight/body mass index a risk factor for falling in the elderly [31] may prevail due to their higher sway area and sway ranges, plus a lower complexity of anterior-posterior center of pressure responses that may impact dynamic balance adversely [32].

Obese older adults with a type 2 diabetes and who may have a high falls risk may also have a greater risk of upper leg, foot, shoulder, and total fractures occurrences than expected depending on falls location and age, but especially if they have abdominal fat excesses, exhibit dynapenia, subnormal lower limb or gait biomechanics, and/or have painful foot ulcers [40, 47. 50].

In this regard, Gkastaris et al. [2] conclude the interaction between obesity and bone metabolism even though complex and not fully understood to date must take note of the possible effects of obesity on the multiple skeletal attributes other than bone that might lead to one or more bone fractures for neuromechanical reasons, as well as possible increases in bone marrow fat content [40], alongside subcutaneous fat and muscle fat increases, muscle strength, mass and reactivity declines especially in the presence of one or more metabolic diseases such as chronic type 2 diabetes that can impact bone metabolism, bone vascular and energy associated mechanisms, muscle endurance, and cognitions highly adversely [41], [45], [54].

In particular, obesity correlates that are linked to multiple chronic inflammation influences, excessive oxidative stresses, as well as possible impaired glucose control and altered bone metabolism and marrow adiposity, plus dizziness, may especially impair bone quality directly or indirectly regardless of bone mineral density and insightful efforts to offset this adverse and potentially serious reactive cycle appear essential to prevent these mechanisms from unduly undermining bone micro architecture and engendering an unwanted progressive state of bone fragility [33], [45]. The role for muscle power and strength declines observed in type 2 obese diabetes sufferers [44] as well as the possible persistent obesity associated gait abnormalities should also be dealt with in a more timely manner and prioritized accordingly, not ignored [46], [47], [54].

In addition more attention to controlling triglyceride levels in the context of type 2 diabetes is warranted due to their adverse influence on fat distribution patterns as well as bone mineral density levels and bone volume [51]. The presence of bone inflammation, altered muscle activation, dynapenia, and excess body weight influences on joint loading, plus a type 2 diabetes declining signaling power of the Wnt transduction cell regulation pathway potentially implicated in this regard also warranted future attention [45], [47].

In addition, the allied influence on bone of continuous over nutrition, poor nutrition, fears of falling, and a resultant state of physical inactivity and limited mobility warrants clarification as does the role of obesity associated musculoskeletal health issues, including skeletal muscle atrophy and dysfunction [44], [48], [53], [54]. Indeed, it appears that even if mechanical loading has positive effects on bone mineral content, this may not suffice to counter fractures in the obese case who has fallen and where low-grade systemic inflammation is probably present alongside a degraded degree of dynamic stability and muscle reactivity [49], [50] and possible presence of extensive frailty [66] in those with health conditions such as osteoporosis or others requiring steroid interventions [61]. Moreover, a lab study has revealed aging related obesity may not only impact bone structure adversely, but may suppress vital muscle-nerve communications that thereby contribute to- rather than resolve- type 2 diabetes functional deterioration impacts [62]. In addition linkages to falls due to dizziness are often present but not duly perceived as injurious [67]. Obesity in its own right has a negative effect on muscle force generation and slow muscle contractile kinetic responses that could injure bone regardless of its mineral density [71].

Additionally, chronic diseases such as type 2 diabetes may deplete bone mass inadvertently in its own right while negatively impacting muscle function and joint protection as well as postural control. This interrelated array of health issues, far from being of only modest concern in many cases and not well understood and poorly palliated at present, that is challenging to reverse, surely predicts a costly burgeoning public health concern in all aging societies in the future if weight control interventions as well as falls and osteoporosis prevention attempts are unsuccessful in offsetting late life bone fragility and a host of progressive neural and biodynamic deficits [34].

Discussion

This mini review that focused on the negative health correlates of obesity and type 2 diabetes attempted to examine this topic in the context of bone implications so as to better establish how adiposity interactions may be harmful in multiple ways to one's wellbeing even in the face of novel anti-obesity drugs and injections as well as passive or invasive glucose control methods. It asked why are overweight adults subject to fractures, and what is the added risk here of a dual type 2 diabetes condition.

Based on a wealth of data, but data largely in its infancy in many respects and challenging to unravel and aggregate with regard to the extent obesity either builds bone mass or fosters bone attrition and fractures, it seems more emphasis is now being placed on explaining the non intuitive relationship between obesity and bone fragility and a bone state not protected from fracturing especially in the presence of type 2 diabetes [41]. In this regard, one study [42] conducted in 2022 concluded that whereas the measure of total body adiposity, abdominal subcutaneous adipose tissue, and visceral adipose tissue were all significantly associated with bone mineral density in both men and women, the strength and direction of this association was not a given but dependent on the subjects sex, body mass index classification, and menopausal status (women) and possible site specific obesity falls linkages and body mass distribution effects [38], [39].

Yet Chen et al. [43] conclude that sarcopenic obesity, defined by visceral adiposity is associated with the risk of osteoporotic vertebral fracture and that a combination of a low skeletal muscle index, low muscle strength and visceral adiposity are independently associated with osteoporotic fractures, while perhaps excess weight asserts a mechanical signal that induces inflammatory responses that underpin joint fragility that leads to bone destruction especially in the overweight older adult [44].

In turn, the combination of a chronic pro-inflammatory state, an increased risk of falls, and some forms of pharmacotherapy can contribute to the pathophysiology of a fracture even if . bisphosphonates and denosumab significantly reduce the risk of fractures in cases with both obesity and type 2 diabetes, as may teriparatide interventions. The implementation of novel diagnostic tools, including those that can readily assess trabecular bone scores, bone mineral density, body mass index ratios, dietary phosphorus, calcium, and vitamin D levels, and microRNAs while possibly helping to predict as well as explain the risk for bone fragility borne by the obese aging adult as well as how bone metabolism is impacted by inflammation, and degraded Wnt signaling in the obese adult with type 2 diabetes, while of high potential, is not well studied or integrated to any degree in this body of research though [39], [47], [58].

In addition the role of muscle strength losses or dynapenia, and their combination that may affect bone density in a sex-dependent manner, in the presence of excess muscle fat mass also appears inconclusive [48]. Also confusing is counter evidence that the obese subject can be found to have a lower bone quality rather than a higher one, regardless of their obesity phenotype [1] and where bone mineral density found to be higher on average in obese young women, may not be beneficial later in life in the face of any persistent vitamin D deficiency [50]. Moreover, the lack of attention to the role of a declining falls efficacy in the obese, alongside pain, and mobility may be especially relevant in explaining sedentary practices and others that are remediable factors that remain generally unaddressed and likely increase the web of age associated bone fracture causation [51], [52], [53], [54], [55] if overlooked in this regard [34].

The allied role of negative cognitions in the face of various degrees of sarcopenia, type 2 diabetes and obesity plus obesity linked balance alterations may further generate the onset and perpetuation of bone attrition, plus injurious falls if unaddressed or acknowledged [34], [56], [69], [72], [74] alongside the application of a therapeutic framework that combines pharmacotherapy with resistance exercise, protein and micronutrient support [34], [57] and possibly falls prevention interventions [54], [66], [67]. Additionally, Aemetscu et al. [23] stress the need for routine fracture risk assessments especially in adults with type 2 diabetes and chronic metabolic syndrome plus abdominal obesity and those categorized as morbidly obese where their bone micro architecture is found to be significantly lower compared to others with less profound obesity as well as healthy weight comparable controls [63].

In addition, to better address the persistent obesity bone fracture rates that are not well understood mechanistically [64] it appears proteomic analyses that reveal molecular pathways linking adiposity, hormonal signaling, immune regulation, muscle, and bone metabolism, offer potential therapeutic targets that should be explored [8], [68] alongside possible novel osteoporotic pharmacologic agents [62] and those that can explain the origins and mitigation of sarcopenic obesity [65].

Key conclusions

Despite the obvious limitations of this umbrella overview of obesity and bone as this pertains to older adults with type 2 diabetes and their wellbeing, we conclude:

1. An interactive negative role for obesity in mediating or moderating bone fracture risk, bone density, and bone integrity prevails at all ages and cannot be readily discounted by those who seek to foster healthy aging across the lifespan.

2. Fractures as well as fracture nonunion are increasingly recognized as an outcome of metabolic disorders such as obesity and type 2 diabetes that demand attention, even though the molecular mechanisms underlying impaired fracture risk as well as healing remain incompletely understood [60].

It is further concluded that the parallel between diabetes, metabolic disease and bone, and a possible interactive impact of excess weight on bone metabolism, as well as a declining ability to absorb load in a timely well modulated manner plus a potent array of inflammatory factors may explain the paradox of bone fractures in the face of obesity associated increases in bone density and should be studied further.

In addition, efforts across the lifespan are imperative to avert childhood and adult obesity and to address this issue more forcefully and consequentially as regards a life time of suffering for many as well as untold public health costs in the face of inaction and the limitations of pharmacologic or weight loss interventions alone. Indeed, it appears weight loss alone in a previously obese case may exacerbate bone dysfunction and malformation including cortical and trabecular bone losses unexpectedly, rather than alleviating adverse obesity linked bone structural and possible balance and gait associated impacts in those with abdominal obesity or obesity in general [65], [70], [72].

At the same time, unaddressed falls risks and a failure to encourage an active lifestyle can undoubtedly further heighten the risk for exponentially increasing health costs as well as immense human suffering both physical and mental as well as immunological and metabolic that is not necessarily inevitable, but somewhat modifiable [5], [69], [73], [74]

Practice implications

While an individualized multidimensional approach to preventing obesity is desirable as far as mitigating serious falls and bone fractures in later life in the context of weight induced type 2 diabetes, and a bone loading mediator in its own right, practitioners cannot be expected to yield favourable outcomes without very careful and comprehensive analyses of the most likely physiological mechanisms underlying bone structural and falls mechanisms in the context of both obese manifestations as well as chronic type 2 diabetes based on isolated thematic non inclusive study approaches and their exaggerated focus on vicarious forms of weight management rather than behaviour changes that may yet underlie a bone fracture risk. Indeed, the persistent impacts of a life of poor nutrition, inactivity, alongside obesity linked disadvantageous cognitions and possible neuromechanical aberrations, in the face of aging and poor obesity management presumably contribute enormously to the public health care burden associated with one or more fractures in later life, especially if ignored or treated too late and without due insight [54], [55], [68], [72], [73].

In particular, the myth that overweight older adults or youth or those with type 2 diabetes are protected against fractures or bone loading alterations, rather than at risk for injury may not only fail to address salient modifiable bone fracture risk factors such as muscle strength losses or those yet uncovered [72], [73], but may allow for more risky rather than protective behaviors or diminished confidence beliefs in the older adults self-generated abilities to protect against both excess bone structural deficits as well as falls [55]. In addition the imperative to assure adequate calcium and vitamin D exposure/ intake as well as exercise participation, health education, food access, outdoor and indoor safety issues, and concerted efforts to foster weight loss and control glucose levels largely pharmacologically may fail if not encouraged or continuous or duly recognized as salient.

In this regard, He et al. [59] imply that rather than an depending on or seeking an isolated pharmacologic solution to weight management, a weight loss-targeted lifestyle intervention may better protect against unwanted bone mineralization and structural alterations as well as falls particularly in the face of an induced rapid weight loss strategy that may yet be harmful to bone and/or fail to return it to its control state if introduced solely as a tertiary rather than as a primary prevention strategy [65].

Childhood and adult obesity prevention efforts, rather than late life palliative interventions as well as bone sparing even in diabetic cases appears to offer a more universal chance of saving lives and is in our view increasingly imperative to overcome obesogenic external socially derived highly negative health influences as well as nutrition based bone deficits in our view, and should not neglect the allied importance of health disparities, nutrition access, efforts to discourage sedentary lifestyles, fast foods, excess screen time, advertising of non nutritious foods, and limited vitamin D exposure as this pertains to bone and muscle health as well as cognitive health and sarcopenic obesity-falls linked attributes [54], [66]. In particular, the specific role of uncontrolled diabetic osteopathy which is poorly studied and why or how obesity may affect some forms of fracture and not others also warrants possible targeted consideration, as do the methods of establishing obesity and its associated apparent bone based and muscle function ramifications, that may well be somewhat reversible [68], [73], [74].

In addition to the above public health implications noted here we urge the practitioner to also:

- Actively aspire to inspire health decision makers and researchers more effectively as regards the immense need for obesity preventive that embodies a series of proactive options, opportunities, and environmental and sociological possibilities that may allay late life fracture associated disabilities that stem from obesity rather than aging alone.
- Portray a less fatalistic stance that nothing can be done when communicating with an aging client or young at risk person.
- Be willing to form collaborative relationships with governments and health agencies concerned with healthy aging in this regard as well as serving as a resource agent for community members.
- Educate the public as well as youth and administrators about what they can do to save costs and lives by remaining active and controlling their food choices and intake.
- Conduct regular pre clinical screening tests for obesity and osteoporosis risk on asymptomatic citizens likely to be at risk for falls, motor as well as cognitive dysfunction, and type 2 diabetes including

non biological social factors, behavioral, situational, gait mechanics, and environmental predisposing factors.

Funding

None

Competing Interests

The authors have declared that no competing interests exist.

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