



Research Article

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Obesity and Type 2 Diabetes Collective Impacts on Late Life Bone Fragility Susceptibility, Falls, Fractures, and Hip Osteoarthritis: a Clinical Overview and Narrative

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Abstract

Sound bone health and the ability to respond effectively to unexpected mobility obstacles or environmental hazards 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 to date, the PUBMED, PubMed Central, and GOOGLE SCHOLAR data bases were sourced for relevant data published between January 1, 2016 and October 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 obesity linked existing type 2 diabetes who may have a high risk of sustaining falls and bone fractures.

Keywords: Bone; falls; fractures; obesity; osteoarthritis; prevention; type 2 diabetes

Background

Bone fractures, as well as bone diseases such as osteoporosis, a common age-related bone weakening condition with well-established guidelines for its assessment and pharmacological treatments is one that is often under-diagnosed and untreated. Those whose bone mass deteriorates unduly are especially likely to go on to incur one or more fragility fractures if they fall and that pose an immense personal degree of suffering alongside immense public health costs in all aging societies. At the same time the presence of sarcopenia- denoting an age-related muscle mass decline- plus an associated risk of osteoporosis, poses an enormous additional

disabling public health challenge that prevails in many aging societies at increasing rather than decreasing rates especially in the case of any prior bone fracture and a subsequent suboptimal surgical repair trajectory in the face of an unintentional falls injury [1-5]. Contributing to this cycle of unwanted events is a possible primary as well as secondary 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, artificially height to weight ratio status distortions, or water retention, and a health state rapidly increasing in the United States and elsewhere, even if tentatively modifiable at the outset. Adding to the burden of poor

bone health and high risk for falling and fracturing a bone in aging overweight adults is the presence of an ensuing type 2 diabetes health profile, that may lead to weight gain as well as bone attrition and possibly other ensuing comorbid health conditions such as osteoarthritis [5].

In particular, the presence of co-occurring obesity and type 2 diabetes may induce a cycle of multiple adverse secondary bone and muscle structural deficits, fat deposition and inflammation, plus progressive fractures, despite the availability of advanced pharmacologic, and surgery approaches. Moreover, the pervasive false belief that obesity has little impact on fostering fractures and negative bone attrition states may lead to irreversible functional deficits [3]. Indeed, while the presence of excess body weight may arguably impact bone mineral density favourably, potentially strengthening the bone, bone fractures and the multiple attributes of bone destruction that preside in sizeable numbers of older adults as well as youth occur quite often in those older adults and others categorized as being obese, and that may be challenged to offset a falls provoking encounter. They may duly sustain highly injurious consequences, especially in those with co-occurring type 2 diabetes, chronic diabetic pain, depression, poor cardiovascular health, sarcopenia, and possible dizziness or deficient balance [4].

Moreover, if untreated or overlooked and not averted, when viewed in both the context of global aging, as well as that of the individual, this scenario is clearly highly disadvantageous and possibly predicts a rapid decline even in the relatively healthy aging adult and one likely to persist and prove highly deleterious to their well-being, as well as to their ability to function physically as well as socially. This cumulative fracture risk that can manifest even in cases suffering from excesses in body fat deposition may yet prove highly injurious to the underlying bone if the forces falling on this overwhelms the bone/s safety threshold. They may not only suffer from extensive bone structural damage, regardless of its mineral density, but may sustain considerable soft tissue damage including muscle trauma and inflammation that does not abate readily, plus multiple neurosensory deficits and pain in those with type 2 diabetes. Moreover, in the absence of medication checks, confusion, as well as hypoglycaemia related declines in bone strength, as well as an unfavourable muscle mass and balance declines may further compound the oftentimes highly disabling outcome of a single falls event experienced by an overweight diabetic older adult.

Methodology and Rationale

This current article drawn from the literature dating back to 2016 to the present 2026 time periods builds on our previous efforts to better understand what has been observed in the above-mentioned contexts as regards the possible linkage of obesity to late life bone health and functional status in the context of obesity and type 2 diabetes, and thereafter to possible secondary osteoarthritis at the fracture site that can possibly provoke its inflammatory and adiposity linkages. The review specifically sought to better understand whether mitigation options other than drugs and injections can postpone the onset of excess age derived disability due to traumatic bone fractures. It sought to examine the strength of sup-

port for additional options that might be pursued to heighten rates of independence and life quality among the elderly population and thereby possibly lower their risk for developing reactive osteoarthritis observed in sizeable numbers of hip fracture surgery cases. It was felt this topic is especially timely given the failure of modern medicine to offset age related trauma incidents and others and despite 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 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 2016-2026 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 options and resources at the individual, societal, and policy levels. 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 the selected 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, bone fragility, and adiposity/obesity, type 2 diabetes, falls, and fractures would provide salient and valid points of current clinical interest and application. The term obesity as used, denotes the presence of any degree of excess body fat that could prove injurious to both a younger as well as an older person and regardless of measurement tool. The term bone health was used to refer 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 and that can engender debilitating osteosarcopenia denoting the impact of sarcopenia on bone mass attrition.

Research Questions

1. Do efforts to counter the combination of limited physical activity and poor nutrition at all life stages have a bearing on efforts to deter the adverse impacts of excess body weight on muscle and bone mass as well as type 2 diabetes and osteoarthritis?
2. Can periodic screening for possible bone and muscle deficits be of assistance in fostering healthy aging outcomes in those at risk or with definitive weight excess manifestations?
3. Are efforts to reduce body weight in chronically overweight

older adult's uniquely successful or limited if applied in isolation?

4. Are there indications more holistic systematic obesity targeted preventive efforts across the lifespan will prove fruitful and valuable in countering excess late life disability and their incalculable costs?

Underlying Assumption

Aging, a state often considered as a downward declining one and one associated with an increasing disease burden has yet produced examples where aging adults thrive rather than simply survive. They do this despite a possible biologically programmed encroachment of sarcopenia, a health condition characterized by an age-related decline in muscle mass and strength, and a replacement of muscle tissue by fat and inevitable bone structural deficits. This however, is not a given in all aging populations, although accounting for a high number of adults who fall, and do sustain one or more bone fractures. This is especially observable in the presence of an obesity linked high muscle fat ratio that appears to reduce bone protection and function and alter bone turnover negatively [5]. At the same time, health conditions such as type 2 diabetes often induced by or co-occurring with obesity may well exacerbate an emergent sarcopenic state as well as falls risk [6]. However, this nonlinear interactive set of associations is often not approached comprehensively or early enough to mitigate its impacts. Rather, a sole reliance on anti-obesity strategies, while potentially helpful in weight loss efforts may yet compromise overall health if muscle as well as multiple bone-based mechanisms remain subverted by fat mass ramifications that remain untreated or recognized [6-10].

This abovementioned argument is not just of theoretical interest, but one of immense relevance as societies age because these overlapping conditions of obesity and type 2 diabetes are both strongly associated with bone fragility as well as being highly prevalent, as is osteoporosis that may predate their unwanted adverse bone impacts [6]. Moreover, all may prove to be a strong fracture risk determinants [9,11,12] alone or in combination especially in those with clear vitamin D deficiencies [7]. A large percentage of cases, especially women, may indeed fail to function optimally, and are likely to experience decrements in overall life quality, possible falls related soft tissue injuries, prolonged hospitalizations, cardiometabolic complications, declining health, and premature mortality [5].

Rationale and Hypothesis

This present effort was undertaken given the apparent lack of attention to the impact of obesity and co-occurring diabetes on musculoskeletal health status, when compared to physical weight status and glycaemic control issues. It was hypothesized that adults with both high fat mass deposits and type 2 diabetes would be at higher risk for falls that lead to hip fractures and others, as well as poorer health outcomes, including the onset of joint cartilage degeneration, but that there would be few publications drawing attention to the need for vigilance against this potentially debilitating health cascade in the current literature compared to the numbers of

pharmacologic related mitigation recommendations even though a lifestyle modification approach appears quite compelling.

Selected Research Observations

In the past decade numerous publications have emerged and 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. Its role in immune support, as well as serving as strategic sites for muscle tendons, ligaments and capsular attachments continue to show how these protective mechanisms may fail in the presence of severe bone attrition and structural damage and that predictably and significantly reduces the wellbeing of the aging adult. Indeed, similar to other body organs, bone, a living tissue, is one that is immune or impervious to breakdown as a result of factors other than age, such as obesity, a topic of increasing interest in this regard. Accordingly, the onset of bone fragility and the overall failure of medications and related options to avert bone associated declines and its possible impact on fracture control in the elderly is of increasing relevance both clinically as well as economically. Most noteworthy here is the increasing observation that while helpful for weight loss, most pharmacologic anti-obesity interventions may yet fail to offset falls that lead to fractures for reasons other than those attributed to weight.

Other data speak to the persistent premise that the impact of obesity on bone mineral density deposits is one favouring a bone protective response against sustaining a fracture, rather than inciting this possibility. A further body of data speak to the strong belief in the efficacy of anti-obesity medications or injections to control appetite and yield significant weight losses that thereby restore health including bone health even if this does not accord with the data wherein bone fragility may well be magnified in the face of co-existing type 2 diabetes. Indeed, a diabetic state is a well-established osteoporotic mediator in its own right and one where the control of glucose via pharmacologic approaches may independently impact bone mass negatively and progressively even if anti-obesity interventions do not harm bone [12]. Bone however, is now considered important to protect since ample research shows 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 pathways including having an obesity history, and its oftentimes dual association with type 2 diabetes- a disabling condition that appears to be increasing over time rather than decreasing.

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 obese altered in multiple ways by the presence of excess body as well as bone fat deposits, that can impair bone integrity and bone remodelling long before efforts to control weight are implemented and even after that [6]. The well observed high falls risk among the obese compared to the non-obese may further imply obesity related altered biomechanical factors may yet determine the de-

gree to which obesity-fracture linkages prevail [6], especially in the older vitamin D deficient inactive adult [7]. As a result, even if osteoporosis is not likely to be present in older overweight fracture adult cases, its impact on bone metabolism and falls risk as well as the onset and perpetuation of type 2 diabetes may be sufficient to cause a fracture regardless of weight status or loss later on [8,9]. Those with a high muscle fat content, excess muscle weakness and reduced muscle mass, and neurosensory disturbances may also experience poor bone shock absorption, low reactive timely responses, and a limited endurance capacity that strongly predicts bone damage even in the face of sub threshold mechanical impacts [6] especially in those with a high degree of viscerally or centrally located fat [10] where the muscles in this region and others may be infiltrated by excess fat that exposes the bone to undue impact and poor or deficient muscle reflex and suboptimal impact attenuation capacities and responses. Moreover, those who also suffer from obesity associated inflammatory responses and those who use diabetes medications that often induce confusion, may experience a state of chronic repetitive joint injuries that may indirectly hasten the onset of osteopenia as well as an altered trabecular bone structure and quality if sedentary behaviours become more entrenched in response to heightened pain, unsteady gait, frequent falls, and falling fears [11,12].

In addition, multiple obesogenic-associated metabolic disturbances may 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, as well as progressive bone cellular and metabolic alterations that cannot withstand unanticipated impacts [7,12,13]. Hsu et al. [14] propose persons having a high fat mass may not respond optimally to standard treatments alone because fat deposition could impose a negative effect on bone mass that is different from that of normal weight-bearing. While obese men may encounter beneficial improvements in bone mineral density [15] postmenopausal women may show lower levels of bone formation markers than those deemed to be of a healthy weight [17]. 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] such as the quality of muscle and muscle sensory response changes [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 may foster the release of harmful molecules and chemicals that can adversely affect bone metabolism and its ability to withstand excess 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 thus creating immense bone physiology alterations. Moreover, these numerical increases in bone adipocytes found to release a number of pro-inflammatory and immune-modulating molecules appear to up-regulate the formation and activation of osteoclasts, a bone cell that removes excess bone,

thus favouring bone fragility. As well, the persistent impact of excess body weight, while possibly favouring bone mineral density increases and bone strength, 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 dynapenic abdominal obesity and/or altered bone blood flow [20-22]. 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 biomechanical, neuromuscular, cognitive, and circulatory factors and their interaction with bone viability as well as vulnerability and may be acquired in the face of certain medications [24-26], poor diets, and muscle suboptimal contractile protective responses in adults exhibiting abdominal obesity [4,20,22,27].

In sum, many factors may explain the higher-than-expected rate of sustaining a fracture injury at some sites even in an obese older adult, for example, at the hip the most serious debilitating injury associated with aging and one with a higher than desired post fracture mortality rate [28] even in the face of successful bariatric surgery [29] or various glucagon uptake inhibitors in those with diabetes [34]. Moreover, this cycle of adverse events 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 including postural balance alterations and body sway excesses [32], as well as possible hormonal factors, and alterations in muscle force capacity and fat ratio especially in the trunk and arm regions [30,32,34,35,38,41,45]. A regional excess of abdominal fat may also disrupt any prevailing bone morphology via its mechanically induced inflammatory impacts and others [33,34], and may be especially accentuated in those categorized as being morbidly obese [36,39].

Additional Findings

Crivelli et al. [37] note that 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 both obese and having a type 2 diabetes do appear to have a substantive risk of sustaining impaired bone quality alterations and a possible 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].

In addition, although appearing site-specific and reducing the risk for fractures in some locations, but not others, this may depend on the degree to which body weight/body mass index is a risk factor for falling in the elderly especially those with age or diabetes and obesity heightened higher sway areas and sway ranges, plus a lower complexity of anterior-posterior center of pressure responses that may impact dynamic balance adversely during locomotor attempts [32] as well as muscle strength, and lower limb or gait patterns, and/or the presence 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, pain, 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 thus 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 thus be dealt with in a timely manner and prioritized accordingly [8,47,54].

In addition, more attention to controlling triglyceride levels in the context of type 2 diabetes as well as encouraging a favorable active lifestyle as this may counter the generation of osteoporosis and its progression in its own right, plus the probable adverse influences on bone health, and bone volume [8,51-53]. Moreover, attention here may avert the additional impacts of failing to do this on the presence of bone inflammation, altered muscle activation, age related muscle strength losses, degraded joint biomechanics and shock absorbing mechanisms and altered hormonal and immune signalling, plus a related decline in the signalling power of the Wnt transduction cell regulation pathway where diabetes may remain poorly controlled [8,45,47]. In addition, the allied influence on bone of continuous over nutrition, poor nutrition, possible fears of falling, diabetic vision problems, and a resultant state of limited mobility or biomechanical abnormalities may engender a perpetual web of negative associations and debility including sensory disturbances, blood pressure disturbances, painful neuropathy, skeletal muscle atrophy and dysfunction and pervasive cognitive manifestations that can ensue in late life to a debilitating although possibly preventable degree [8,44,48,53,54].

However, if untreated or ignored, it appears that even if the ex-

cess obesity associated mechanical loading of the joints 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 underlying osteoporosis and signs of frailty, as may those deemed obese with a type 2 diagnosis and high rates of sarcopenia, frequent bouts of dizziness, deficits in vision perception, and muscle-nerve communication processes [53,56,61,62,67], regardless of the presence of adequate or excess mineral density deposits [71]. In the meantime, it can be assumed obesity independently increases the risk for falling in the older adult population, and thereby pain and inactivity at higher rates than healthy weight adults even if no fracture occurs [81-85]. Thus, a host of interrelated factors can undoubtedly prove largely disadvantageous to an older adult and is a situation that predicts an incrementally costly and burgeoning public health issue of high concern in all aging societies in all likelihood [8,34,53,56,61].

Discussion

This mini review that focused on the health correlates of obesity and type 2 diabetes in the context of their unique or combined bone implications on late life wellbeing was undertaken despite the apparent efficacy of novel anti-obesity drugs and injections as well as passive or invasive glucose control methods. It asked why overweight adults are still subject to fractures and what the added risk is as regards a dual type 2 diabetes diagnosis in fostering a probable heightened falls risk in the obese older adult. Moreover, it sought to establish if a state of osteogenic fragility can hasten or foster a state of associated osteoarthritis joint damage at the fracture site as an endpoint. Based on a wealth of data, but data largely challenging to unify or unravel with regard to the extent obesity either builds bone mass or fosters bone attrition and fractures, and jeopardizes the repair of fractures that often predate the onset of osteoarthritis, especially at the hip joint, the topic appeared of high relevance none the less [86]. In this regard, one study [42] conducted in 2022 concluded that whereas what is observed or not observed may depend on the mode of measuring total body adiposity, abdominal subcutaneous adipose tissue, and visceral adipose tissue, all appear significantly associated with bone mineral density in both men and women in some way, even if the strength and direction of this association is not a given [38,39].

In particular, Chen et al. [43] conclude sarcopenic obesity, defined by visceral adiposity is associated with the risk of osteoporotic fracture and that a combination of a low skeletal muscle mass and strength and visceral adiposity may independently predict the presence of osteoporotic fractures in the obese, while perhaps excess weight asserts a mechanical signal that induces inflammatory joint fragility responses that leads to bone destruction especially in the morbidly overweight older adult [44]. Other data reveal a trend to increasing hip fractures that are linked to the prevailing degree of central obesity and possible excess bone marrow fat deposits [86]. In essence, even if weight is lost and medication used to build bone, appear helpful, the combination of a chronic adap-

togenic pro-inflammatory state, and thereby an increased risk of sustaining a fall, some forms of pharmacotherapy associated with obesity and diabetes care can contribute collectively to the pathophysiology of one or more fragility fractures. In addition to impacting function severely, the added problems of increasing bone fat mass as well oxidative stress levels alongside muscle mass and strength declines are likely to affect bone trabecular structure negatively as well as mineral density in the face of unrelenting muscle fat mass [46,48]. Adding to this probable adverse scenario are data that point to 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 [91] and where bone mineral density found to be higher on average in obese young women, may not be beneficial later on 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 type 2 diabetic case alongside the onset of pain related falls outcomes and mobility declines may be especially relevant in heightening sedentary practices and others that are challenging although not impossible to remediate [51-55] including obesity linked postural control, gait, muscle strength, contractility and balance alterations that may further hasten the onset and perpetuation of unwanted bone attrition as well as injurious falls [34,56,69,72,75]. Consequently, although not standard practice, it appears the adoption of a more comprehensive therapeutic framework that combines pharmacotherapy with resistance exercises, protein and micronutrient support [34,57] and possibly falls prevention interventions [54,66,67] may obviate much disability among many elders as well as those who have fallen [85]. Additionally, a role 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 is indicated [23], as well as in cases where the subject's 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, obesity levels as well as malnutrition and inflammatory factors that predict mortality in type 2 diabetes cases warrant attention [76].

Clinical Implications

To better address the persistent obesity bone fracture rates that are not well understood mechanistically [64] it appears more efforts to reveal molecular pathways linking adiposity, hormonal signalling, immune regulation, muscle, and bone metabolism with cognitive health are desirable [8,68.] and should aim to identify the most influential modifiable pathways of note that may differ from those of age and other associated osteoporosis processes [92]. Attention to possible novel osteoporotic pharmacologic agents thereafter [62], and those that can explain the origins and mitigation implications for sarcopenic obesity may also be helpful [65,91,92].

In the interim, it appears:

- An interactive negative role for obesity in mediating or moderating injurious and non-injurious falls, bone fracture risk, and integrity at all ages cannot be readily discounted.
- Fractures as well as fracture non-union increasingly recog-

nized as an outcome of falls as well as metabolic disorders such as obesity and type 2 diabetes and that can ultimately induce an osteoarthritis joint state that further heightens independence losses, possible weight gain and muscle atrophy and pain warrants insightful cogently counteractive upstream rather than downstream action plans.

- More attention to uncovering obesity linked falls mechanisms plus the impact of excess adipose tissue mechanisms underlying impaired muscle and bone function especially as related to the role of osteosarcopenia sarcopenia may help minimize a substantive degree of late life fracture risk and its disabling impacts quite immeasurably [60,89].
- Uniform universally agreed upon modes of valid bone and obesity assessments may help advance the ability to more clearly establish the import of this association and thereby the optimal course of action for mitigating any associated bone trauma in later life.
- Eliminating barriers to funding for well-defined advanced research, including primary prevention multidimensional anti-obesity lifestyle options and efforts to avert the related onset of type 2 diabetes will similarly help advance this field of inquiry and practice favourably in all likelihood.
- At the same time, more clinically focused and targeted studies rather than laboratory studies that may not translate consistently into clinical realms as far as fostering muscle mass or muscle size and improvements in strength, mobility, and fatigue and averting the onset and influence of type 2 diabetes obesity associations are strongly indicated [68].

Concluding Remarks

Despite gaps in the literature, and possible research oriented flaws, it appears safe to conclude obesity has a clearly and notable negating cumulative influence on bone and muscle as well as general health especially in later life where it may exacerbate the risk of being sedentary, incurring serious falls and bone fractures, type 2 diabetes and a possible post hip fracture state of disabling hip osteoarthritis, a condition that can significantly increase the risks of single and recurrent falls or trips due to pain, weakness, instability, poor balance, gait impairments, obesity, medication side effects, depression, invasive surgery, and stiffness [95].

Indeed, although obesity has long been deemed to protect against bone fractures, its impact as a fracture mediator in its own right appears undeniable. In addition, its link to chronic type 2 diabetes appears to encourage an exaggerated form of sarcopenia or osteosarcopenia and bone fragility despite various forms of weight management.

As such, rather than assuming obesity is a bone protector and that one can rely on pharmacologic solutions to reduce this unwanted health condition, this approach appears to surely fail to account for the multi-dimensional interactive impacts of excess body fat on multiple body systems including bone and muscle structure and their physiology, and associated body weight control and glu-

cose homeostasis processes [93].

To advance this field and reduce the burden of failing to intervene in a timely manner to counter much suffering, we believe it is not only imperative to examine this issue more cogently but to encourage more enlightened public health awareness options, alongside more emphasis by clinicians and researchers on possible behavioural changes that may yet produce more favourable body composition results versus the negative persistent impacts of excess fat on multiple tissues and organs attributable to poor nutrition, inactivity, a lack of accurate timely information, and medication linked disadvantageous on cognitions, in poorly controlled type 2 diabetes that may predate joint attrition and its adverse multidimensional ramifications quite predictably and adversely [54,55,68,72,73,79,80].

Those older adults demonstrating progressive strength losses and signs of muscle atrophy, central obesity, including those who have experienced weight losses [72,73,87], have fallen recurrently, and have a type 2 diabetes profile should be preferentially targeted and educated about their overall health risks and possible bone macro and micro fracture risks, plus potentially acquiring a state of chronic inflammation if they adopt a sedentary passive lifestyle [55] with limited exposure to adequate calcium, protein, and vitamin D as well as exercise participation opportunities [88]. In this respect, rather than a total reliance on late life palliative interventions, efforts to counter a long-held belief that obesity protects against bone fractures, should be more pronounced even in healthy weight women [79] and especially those with high waist circumferences [90].

Of additional import here may be:

- a) Diverse routine screening efforts, the adoption of favourable lifelong health affirming lifestyle and behavioural efforts, periodic medication checks and follow ups.
- b) Attention to eliminating health access disparities, discrimination of the obese person, environmental mobility hazards, health illiteracy, and food insecurity.
- c) More public health and practitioner efforts to discourage sedentary lifestyles, the excess reliance on fast or processed 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 [54,66,89].
- d) Efforts to reconsider the importance of vitamin D sufficiency as well as the specific role of uncontrolled diabetic on bone health [78], as well calcium plus the adoption of more definitive methods of establishing obesity and its associated apparent bone based inflammatory and muscle function ramifications, may avert much suffering as well [68,73,74,94].

In addition, practitioners are encouraged to:

1. Actively inspire community leaders, health decision makers and researchers as well as clients or at-risk clients to do all they can to prevent obesity and that embodies a series of proactive options, opportunities, and environmental and sociolog-

ical possibilities.

2. Help portray a less fatalistic stance or a state of denial that nothing can be done when communicating with an aging client or young at-risk person about body weight issues and its probable impact on bone health as well as well-being.
3. Be willing to form collaborative long-term relationships with clients in this regard as well as serving as a resource agent for them as well as their families and community members.
4. 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.
5. 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, behavioural, situational, gait mechanics, and environmental predisposing factors.
6. Offer integrated life-style strategies to at risk or high-risk adult subgroups [for example those exhibiting abdominal or central obesity who may feel ashamed to exercise in groups] that are tailored, goal directed, and embrace physical, mental, pain control, safety issues, socio-emotional and functional dimensions of health, not only weight loss, diet and pharmacologic dimensions.

By contrast, as opposed to early efforts to foster awareness and care, it appears a predominant downstream tertiary preventive view where weight loss alone in a previously obese case is the sole target may yet exacerbate bone dysfunction and malformation including cortical and trabecular bone losses, as well as having direct trauma induced bone impacts even in the absence of discernible bone attrition, a tendency to foster type 2 diabetes bone and muscle degradation states, plus possible balance and gait associated impacts in those with abdominal or general obesity [65,70,72]. Likewise, poorly understood falls risks and safety issues, the need for continuous muscle maintenance efforts in the face of obesity/anti-obesity interventions, plus diabetes related impacts including some medication regimens and their widespread cognitive and neuromotor effects will remain impediments to desirable outcomes if ignored even if drug based approaches appear favourable in advancing bone mineral density as a whole in our estimation and increasingly supported by substantive diverse researchers [5, 69, 73, 74, 77].

Final Thoughts

By encouraging the early and continuous adoption of healthy behaviours as the best choice for securing a life of high quality at all ages it may be possible for individuals at risk for a late life fracture to acquire a better sense of control over some factors that impact the ability to age successfully and without a high osteoporotic linked falls induced fracture risk. Insightful post fracture rehabilitation strategies that are modified in light of the impact of an obesity and diabetes history may further prevent an excess degree of emergent

disability and recurrent traumatically induced joint vulnerability, a shorter life expectancy, costly medical complications and resource demands, plus possible secondary fractures.

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Conflict of Interest

No conflict of interest.

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