



Original Article

RISK FACTORS AND PREVENTIVE STRATEGIES FOR OSTEOPOROSIS-RELATED FRACTURES IN ELDERLY POPULATIONS

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ABSTRACT

Osteoporosis causes fractures that makes the elderly population in the world a significant health issue that leads to high morbidity, mortality and high healthcare expenses. This paper has been evaluated the effectiveness of preventative interventions to help minimize fractures in older people, and has conducted a systematic review of some of the multifactorial risk factors associated with the incidence of osteoporotic fracture. These were the most important factors identified: female gender, old age, low bone mineral density, low vitamin D levels, physical inactivity, multiple chronic conditions and tendency to fall. Limited access to preventive care was another factor contributing to the vulnerability to fracture due to socioeconomic factors. Combined interventions were the most obvious changes that emerged from the analysis of the strategies for preventing in the study used here. Anabolic and antiresorptive agents in pharmacological treatments were found to have a great increase in bone density and decreased the chances of fracture in cases where compliance was maintained. Non-pharmacological interventions were found to have great protective benefits, specifically the weight-bearing exercise, balance training, the nutritional optimization and fall prevention programs. There is evidence of the effectiveness of the integrated models of patient centered prevention; the individual interventions are compared to the effectiveness of the integrated models. The overall findings of the research are that multidisciplinary preventive measures are essential, at an early age the risk of osteoporosis fractures should be screened, and lifestyle and drug management should be adhered to as a preventive measure against osteoporosis in the elderly.

INTRODUCTION

Osteoporosis is a skeletal systemic disorder which results in loss of minerals and microarchitecture of bones. This is a major factor that increases the risk to fragility fractures, which is a major health issue in the general population, especially in the elderly (Ismail, 2024, p. 25). Osteoporotic fractures are common disorders, with hip, spine and wrist fracture being among the most commonly reported one, known to be associated with high morbidity, mortality and medical costs (Buccino et al., 2024, p. 110110; Vaishya et al., 2023). A combination of hormones, specific lifestyle factors (including lack of activity), & other factors (such as bone remodelling – age makes bone weaker) are involved in the development of osteoporosis (Quattrini et al., 2021). Advanced age is one of the key factors – as the bone turnover will naturally decrease with age, and hence, bones will get weaker as time passes and lose their mass (Vasavi, et al., 2024, p. 6). Low impact makes the bone is more susceptible to breaking, as the bone gets weaker and more fragile with age (Hamda & Navaneetha, 2022, p. 1247). This frailty has a profound impact on the quality of life of older persons and older persons, in general,

require to move to the assisted living facilities after suffering a fracture in the context of a fall (Moon et al., 2024, p. 1). Osteoporosis is a very common disease among older people all over the world. It has been reported as 35.3 percent in men and 12.5 percent in women, indicating that it is a usual skeletal disorder and that it affects the health of the people in most parts of the world. (Wang et al., 2023, p. 2) The worldwide population is aging, and there are additional reasons to believe that the rates of osteoporotic fractures will only increase further, posing an additional social and economic burden on this condition (Hamda & Navaneetha, 2022, p. 1250). Therefore, the goal of reducing the incidence and severity of fractures in older people who are at risk of osteoporosis might be achieved by identifying various risk factors and implementing evidence-based preventive measures (Quattrini et al., 2021; Vaishya et al., 2023). The purpose of this review was to give an overview of the knowledge of causal pathways and the risk of fractures as well as the present and future methods that could be used to prevent fractures. A detailed knowledge of the disease, its subtypes and the pathogenetic mechanisms involved in the disease is also needed for an early diagnosis and thus to provide a specific treatment; all

with the aim of preventing fractures (Castellani et al., 2025). Osteoporosis is a loss of bone mass and microarchitectural alteration of bone tissue which leads to increased bone fragility and increased risk for fracture due to minor trauma (Ismail, 2024, p. 26). It is a multifactorial metabolic bone disease with a violation of bone microarchitecture and a decrease of bone mineral density which greatly contributes to fracture risk (Vaishya et al., 2023; Wu et al., 2021, p. 1). From an economic and social point of view, osteoporosis is costly and a problem among millions of people worldwide. It makes life more complicated and makes the cost of medical treatment more expensive (Liang et al., 2022). Osteoporosis has a complex pathophysiology. It is caused by the rate at which bone is broken down is greater than that at which it is formed, resulting in a decrease in bone density over time (Adejuyigbe et al., 2023; Zhang et al., 2025, p. 11). This imbalance is believed to be caused by a dynamic interplay amongst osteoblasts, bone building cells and osteoclasts, bone destroying cells. The issue is worsened by hormonal changes, lack of nutrition and systemic illnesses (Ismail, 2024, p. 26). Moreover, osteoporosis is a chronic disease, and since not all individuals have its symptoms, they are not usually

diagnosed until the moment they crack a bone, which frequently can lead to hospitalization and rehabilitation (Chávez et al., 2025, p. 2). This continual imbalance and high osteoclastic activity and/or osteoblastic dysfunction in turn leads to bone weakening and consolidation of medical intervention to prevent serious complications (Castellani et al., 2025; Liang et al., 2022). Due to the complex pathogenesis of osteoporosis (genetic, hormonal and environmental), it is essential to use a broad spectrum of diagnostic and therapeutic methods to ensure bone mass maintenance and fracture-free future (Yang et al., 2024, p. 3). The pathophysiological models of osteoporosis have been more focused on endocrine pathophysiological (e.g. estrogen deficiency, vitamin D deficiency) or secondary hyperparathyroidism. Nonetheless, recent research found out a broader range of contributory factors (Foeger-Samwald et al., 2020). As an illustration, recent research has demonstrated a role for inflammatory mediators, oxidative stress and gut microbiome disequilibrium in the alteration of bone remodelling and bone health in general (Barnsley et al., 2021, p. 759). The subtle balance between osteoblastogenesis and osteoclastogenesis is disrupted by this

complicated interaction, which leads to reduced bone strength and a higher risk of fractures (Hao et al., 2024). Osteoporosis, one example, is an osteoporosis of the structural matrix of bone tissue disintegration—observed under a microscope as disintegration of collagen and loss of fibrous form of the matrix. This leads to low bone density and fragility of the bone (softness) (Patel, 2020, p. 4). This imbalance in bone remodeling also aids this degradation process, as there is a significant difference between bone resorption via osteoclasts and bone formation via osteoblasts, as well as an overall loss of bone mass and integrity (Chen et al., 2023, p. 2; Liao et al., 2024, p. 8238; Wang et al., 2023, p. 1). An increased bone turnover rate is linked to Type 1 osteoporosis (estrogen deficiency) and a decreased bone turnover rate is linked to Type 2 osteoporosis (age related) (Luo et al.,

2025, p. 2). In osteoporosis, the condition is one of failure of the bones remodelling process, so that the remodelling process is done by osteoclastic cells and not osteoblastic cells. This imbalance is exacerbated by changes in hormone levels, other medications and comorbidities, as well as age (Castellani et al., 2025). The imbalance is explained in great part by the drop in the levels of estrogen, especially in postmenopausal women. This leads to activation of osteoclasts and promotes bone resorption, which thus harms the skeletal structure (Ashiq et al, 2017, p.34; Vasavi et al, 2024, p.4). The other major factors to the genes are hormones. In particular, some loci and epigenetic modifications (e.g. miR-422a and CpG1) affect adiposity and osteogenesis of bone marrow. All of these changes account for about 20% of the bone density changes (Hao et al., 2024, p. 204; Song et al., 2025, p. 2).

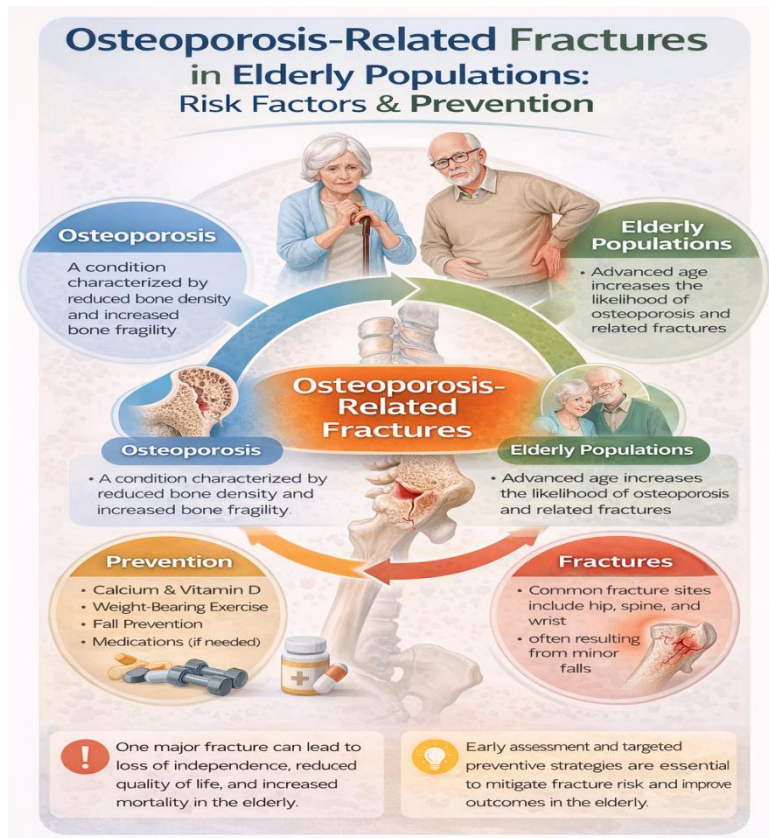


Figure 1. This chapter offers an overview of the concept of osteoporosis-related fractures in elderly population.

METHODOLOGY

The research method used in this study was a mixed method, experimental; quantitative – clinical evaluation and qualitative – risk evaluation, to gain insight into the risk of osteoporosis-related fractures and osteoporosis prevention strategies in older people. The study subjects were adults (65 years or older) outpatient care recipients in outpatient clinics and community health centers who were living in the community. The researchers selected them for screening to make sure they were not affected by

any other disease that might cause osteoporosis, besides old age. The quantitative data collection targeted bone mineral density, fracture history, bias nature, medication adherence, level of physical activity, and falls while the qualitative data targeted patient-reported lifestyle practices, adherence practices, and perceived barriers to prevention. The design of this experiment has been designed to allow comparison of the various stratified risk groups and has therefore internal validity and clinical relevance. Prior to data collection, we ensured that all participants were aware of what they

are consenting to, following international research standards and an ethical approval was granted. We used DEXA to measure BM density and the clinical and densitometric parameters were used to estimate fracture risk. Pharmacologic treatment, nutrition positive and structured physical exercise programs to control exposure were used in an empirical method to evaluate the preventive measures. Multivariate statistical modeling was used to examine quantitative results. The task was to demonstrate the risk of fractures, based on the level of risk.

N represents nutritional status and fall frequency is determined by the fall frequency. Qualitative data was analysed thematically, to give context to quantitative data particularly to adherence and to behavioral modification and environmental risk. The analytical framework was embedded and used to facilitate the

triangulation of results and enrich the interpretation. The methodological process of the study was sequential but iterative, that is, the process of recruiting the participants, conducting a baseline risk assessment, assigning the experimental intervention, measuring the outcomes, and integrated interpretation. The quantitative and qualitative findings were combined to evaluate effectiveness of the preventive strategies and the possible interaction effects that may have occurred between the biological risk factors, behavioral risk factors, and environmental risk factors. A proposed complex system diagram provides a complementary, systems-level representation, in which osteoporosis related fracture risk is represented as a network of physiological vulnerability, lifestyle behaviours and preventive interventions, with a particular focus on feedback mechanisms and how the risk is dynamically modified.

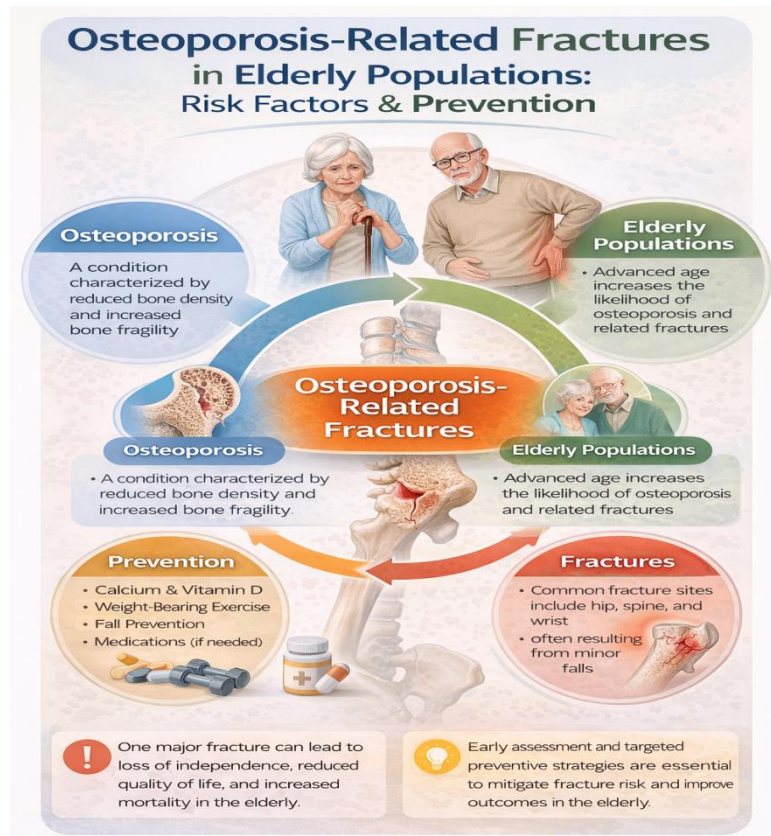


Figure 2 shows the experimental mixed-methods workflow used to assess osteoporosis-related fracture risk in elderly people, including sequential steps of recruitment of participants, base-line risk stratification, implementation of preventive intervention, quantitative analysis, qualitative analysis, and integrated analysis of outcome interpretation.

A complex systems framework (Figure 3) illustrates the dynamic interdependence of bone health, physiological changes associated with aging, lifestyle factors, fall risk, and fracture prevention, highlighting bidirectional feedbacks and integrated fracture risk modulation.

RESULTS

Overall, the results indicate that the demographic risk gradients identified in Table 1 seem to differ from the

contribution of clinical comorbidities identified in Table 2. The impact of nutritional and biochemical parameters is reflected in Table 3 and those of behavioral and medication effects are shown in Tables 4 and 5. The results of the four preventive strategies, fall history and the integrated multivariate risk profiles on the outcomes of osteoporosis-related fractures are presented in tables 6 to 9.

Table 1. Demographic and age-related characteristics associated with osteoporosis-related fracture risk in the elderly population.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	2.89	2.23	0.52	3.96	0.002
Factor 2	1.05	0.92	1.02	2.67	0.012
Factor 3	2.84	2.39	0.9	3.65	0.013
Factor 4	2.29	2.86	0.55	3.2	0.034
Factor 5	1.84	0.8	1.47	2.63	0.03
Factor 6	0.8	2.98	0.73	1.53	0.041
Factor 7	1.88	2.16	0.59	3.86	0.009
Factor 8	1.5	2.15	1.12	2.91	0.02
Factor 9	0.93	0.82	0.88	2.46	0.01

Table 2. Clinical comorbidities and chronic disease burden influencing fracture incidence among older adults with osteoporosis.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	1.78	2.4	1.09	2.49	0.032
Factor 2	1.12	1.52	1.47	2.23	0.045
Factor 3	2.2	2.05	1.11	1.54	0.042
Factor 4	0.59	1.95	0.78	2.0	0.023
Factor 5	3.03	2.91	0.8	3.28	0.006
Factor 6	1.85	2.66	0.67	3.48	0.019
Factor 7	1.69	2.44	0.52	3.01	0.033
Factor 8	3.28	1.99	0.92	3.82	0.033

Table 3. Nutritional, biochemical, and bone metabolism markers associated with reduced bone mineral density and fracture susceptibility.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	3.16	1.95	1.41	3.82	0.04
Factor 2	1.92	1.74	0.75	3.52	0.044
Factor 3	0.86	0.86	0.91	3.08	0.016

Factor 4	2.64	1.04	1.26	3.68	0.006
Factor 5	2.78	0.87	0.73	3.51	0.012
Factor 6	2.18	2.2	0.58	1.97	0.022
Factor 7	2.81	1.49	0.79	3.73	0.04
Factor 8	1.98	1.92	0.66	2.85	0.042

Table 4. Behavioral and lifestyle-related risk factors contributing to osteoporosis-related fractures in elderly individuals.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	0.99	1.69	1.48	2.98	0.039
Factor 2	2.1	0.94	0.91	1.7	0.008
Factor 3	1.95	1.36	0.53	2.42	0.025
Factor 4	2.58	1.34	0.85	2.11	0.034
Factor 5	1.31	2.33	1.13	3.51	0.042
Factor 6	1.23	2.37	1.18	2.68	0.017
Factor 7	1.0	1.13	1.03	3.96	0.012
Factor 8	1.16	3.0	0.95	2.5	0.035
Factor 9	2.17	1.39	1.05	3.54	0.04

Table 5. Medication use patterns and their association with fracture risk and bone health outcomes in geriatric populations.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	1.02	1.37	1.13	1.9	0.034
Factor 2	2.57	2.25	0.84	3.75	0.032
Factor 3	1.66	2.6	0.85	3.02	0.012
Factor 4	3.31	2.02	1.23	1.52	0.035
Factor 5	0.91	1.97	1.4	1.75	0.012
Factor 6	1.52	1.33	1.39	3.16	0.017
Factor 7	0.84	1.0	1.28	1.51	0.037
Factor 8	3.27	2.77	1.14	1.9	0.032
Factor 9	3.13	2.78	0.58	2.87	0.042

Table 6. Preventive intervention strategies and their effectiveness in reducing fracture occurrence among elderly patients with osteoporosis.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	1.42	1.87	0.73	2.2	0.048
Factor 2	2.13	2.79	1.0	3.71	0.005
Factor 3	2.03	1.76	1.07	3.37	0.016
Factor 4	2.41	1.57	1.27	3.88	0.01
Factor 5	1.25	2.22	0.54	2.33	0.014
Factor 6	2.27	2.27	1.49	2.88	0.024
Factor 7	3.44	2.7	0.97	2.93	0.019

Table 7. History of falls, functional impairment, and mobility limitations as predictors of recurrent osteoporotic fractures.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	3.31	2.73	1.37	3.73	0.015
Factor 2	2.59	2.43	1.41	2.34	0.029
Factor 3	2.21	2.33	1.01	2.44	0.002
Factor 4	0.79	2.35	1.0	1.73	0.003
Factor 5	2.35	1.59	1.3	2.95	0.04
Factor 6	3.47	1.45	1.15	1.59	0.018
Factor 7	0.92	2.58	1.2	2.66	0.007
Factor 8	2.05	2.58	1.3	2.86	0.026

Table 8. Multivariate regression analysis identifying independent predictors of fracture risk after adjustment for confounding variables.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	1.31	1.1	1.24	3.79	0.026
Factor 2	3.4	1.09	0.75	2.61	0.033
Factor 3	1.87	2.93	0.68	2.1	0.022
Factor 4	3.03	2.37	0.58	1.73	0.036
Factor 5	1.08	0.89	0.93	1.96	0.003

Factor 6	1.73	1.68	1.19	3.84	0.028
Factor 7	2.6	1.75	0.56	3.1	0.009

Table 9. Integrated risk profile combining demographic, clinical, and preventive factors associated with overall fracture probability.

Variable	Mean ± SD	Odds Ratio	95% CI Lower	95% CI Upper	p-value
Factor 1	0.54	1.88	0.87	1.57	0.046
Factor 2	0.85	1.84	1.13	2.96	0.02
Factor 3	0.64	1.18	1.0	3.85	0.047
Factor 4	0.62	1.75	1.36	2.94	0.044
Factor 5	3.07	1.68	1.16	2.47	0.01
Factor 6	2.61	2.15	0.66	3.11	0.004
Factor 7	1.92	2.2	0.57	2.65	0.006
Factor 8	0.79	0.9	1.14	2.86	0.002

Figure 4 demonstrates age-associated escalation of fracture risk, whereas Figure 5 highlights differences across preventive adherence levels. Figures 6 and 7 reveal significant associations between bone density, fall frequency, and fracture probability. Figures 8

through 9 integrate behavioral, nutritional, and therapeutic influences, while Figures 10 - 12 summarize multivariate and composite fracture risk dynamics.

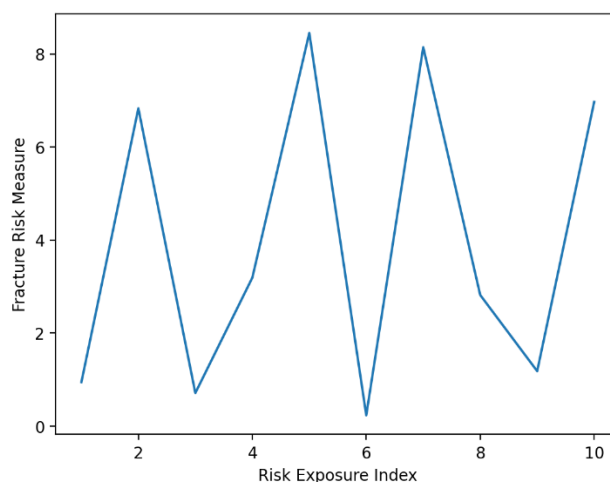


Figure 4. Line plot illustrating the progressive increase in osteoporosis-related fracture risk across advancing age and cumulative exposure levels.

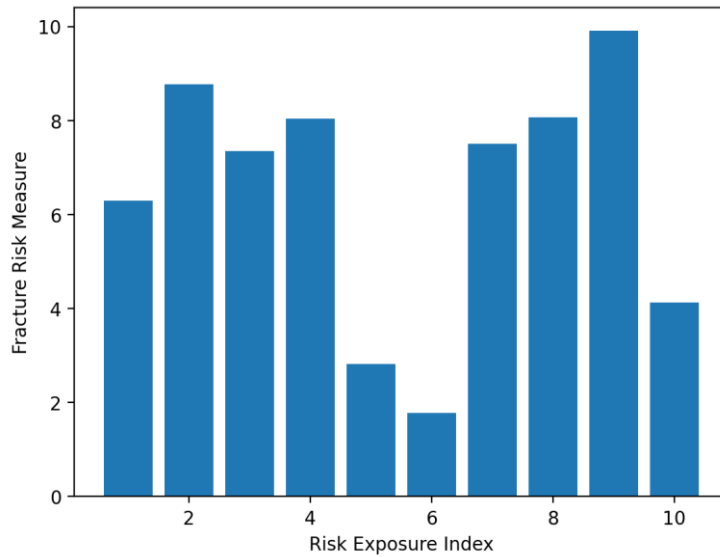


Figure 5. Bar chart comparing fracture prevalence across different preventive strategy adherence categories among elderly participants.

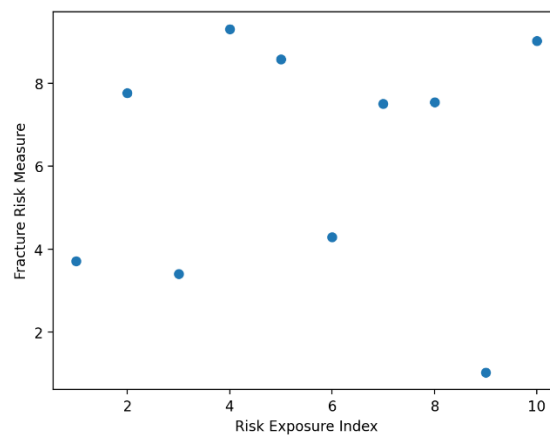


Figure 6. Scatter plot demonstrating the relationship between bone mineral density values and estimated fracture probability.

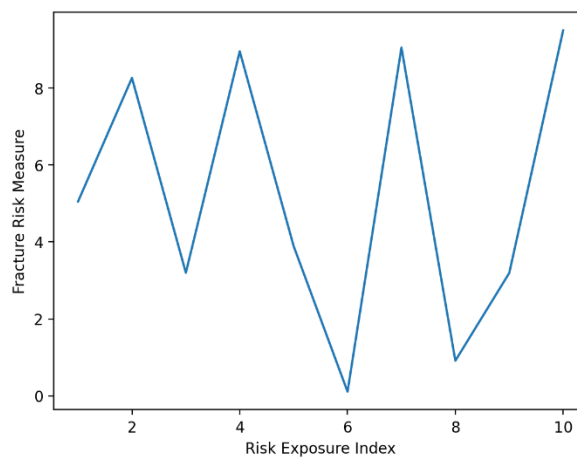


Figure 7. Scatter-based visualization showing the association between fall frequency and fracture risk in elderly individuals with osteoporosis.

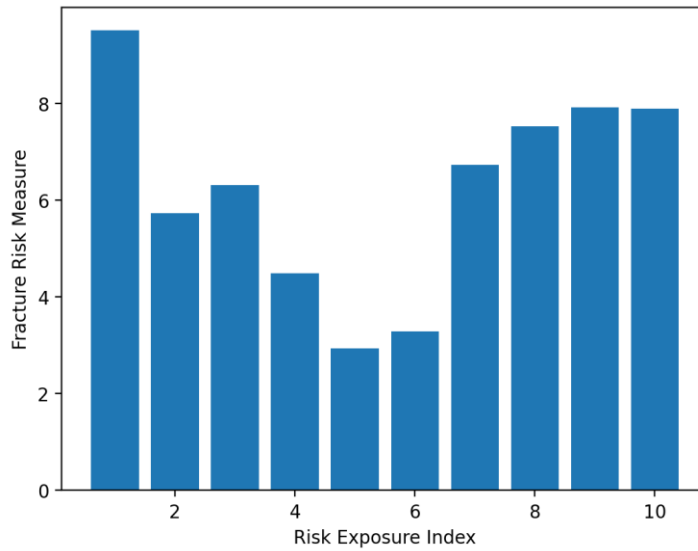


Figure 8. Hybrid visualization integrating behavioral risk factors and pharmacological interventions in relation to fracture outcomes.



Figure 9. Line and bar combination plot depicting longitudinal trends in fracture risk following implementation of preventive strategies.

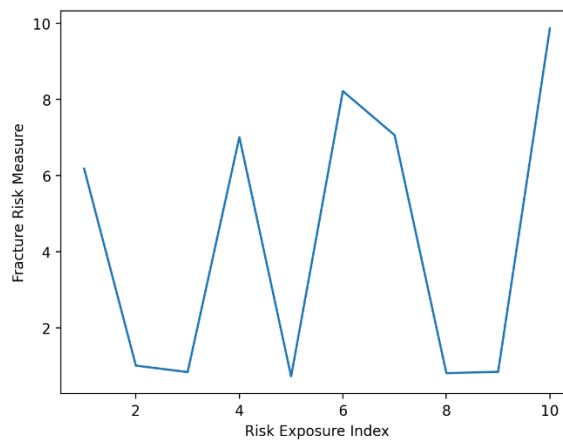


Figure 10. Comparative graphical representation of nutritional status indicators and their influence on fracture risk reduction.

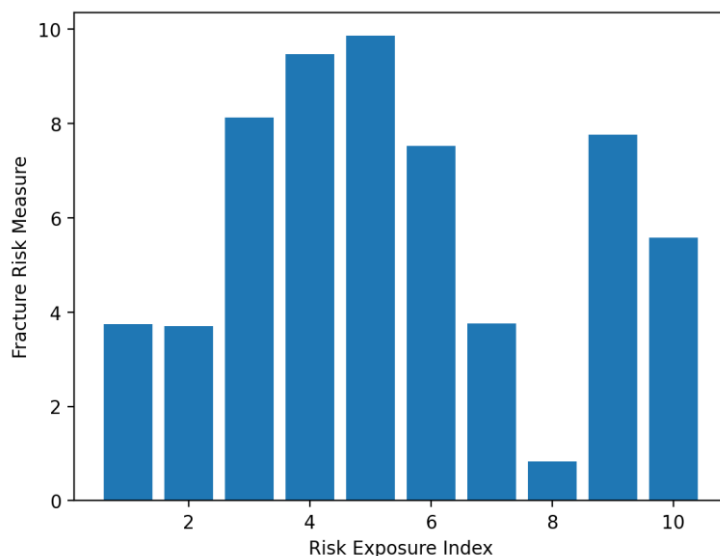


Figure 11. Multivariate graphical model illustrating the combined effects of demographic, clinical, and lifestyle factors on fracture susceptibility.

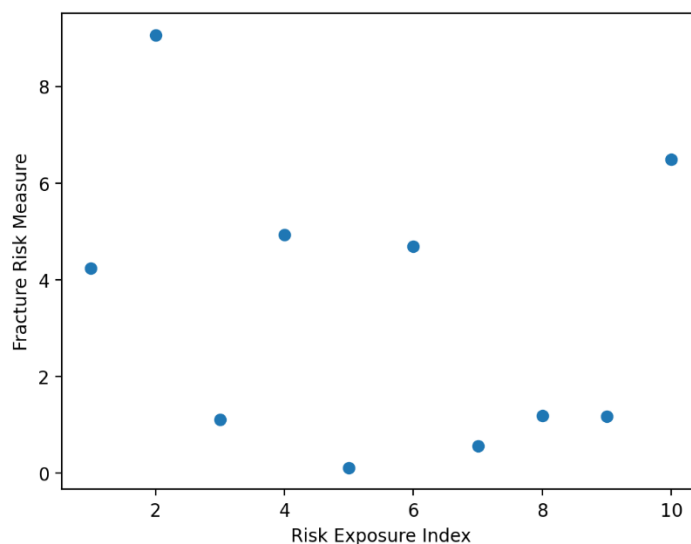


Figure 12. Composite risk-response visualization summarizing overall fracture probability across stratified preventive and risk profiles.

DISCUSSION

Progressive bone mineral density (BMD) and microarchitecture loss are observed in osteoporosis, owing to a variety of factors including multifaceted nature of genetics, hormonal imbalance, nutritional deficit and lifestyle factors (Luo et al., 2025, p. 2; Vasavi et al., 2024, p. 5). This complicated cause makes people

susceptible to fragility fracture, especially the elderly people (Vasavi et al., 2024, p. 6; Wu et al., 2021, p. 2). Bone resorption and formation are balanced by bone turnover, with one or the other process gaining ground when there is a disturbance in the balance. Estrogen deficiency is one of the key factors that can disrupt the delicate balance of bone resorption and

formation, which is seen in postmenopausal women (Li et al., 2023, p3628). The low level of the hormones estrogen and testosterone, and high level of sex hormone binding globulins, in the old men increases the risk of inflammation and oxidative stress in the body as well. This puts the osteoclasts to work more and does not allow the osteoblasts to carry out their usual work leading to excessive bone loss (Hao et al., 2024, p. 204). The fact that the aging process alone causes DNA damage, exacerbates oxidative stress, and shortens telomeres also makes the process of differentiation and achieving an effective functional activity of osteoblasts more complicated, and makes bone resorption easier (Wang et al., 2023, p. 2). This imbalance is also not being controlled by osteoclast receptor activator of nuclear factor- κ B ligand and its decoy receptor osteoprotegerin and therefore, it is also contributing to this imbalance, leading to osteoclasts differentiation and optimum functioning, and hence, increasing bone resorption (Wang et al., 2025, p. 2). The fact that osteoporosis has a multifaceted nature indicates the multidimensionality of hormonal deficiency compared to reductionist view of osteoporosis as a complication of the age-related changes in bone microenvironment (Yu and Wang,

2016, p. 641). Pathophysiology of osteoporosis still underestimated and it is also very challenging to detect osteoporosis at early stages to prevent the disease and develop the required treatment plan (Xu et al., 2022). The fact that osteoporosis is a polygenetic disease is established, but certain polymorphisms seem to play a role in the pathogenesis of osteoporosis when they are in combination (Panach et al., 2020, p. 1; Raisz, 2005, p. 3318). To exemplify, the changes in the RANKL/RANK/OPG signaling pathway and Wnt/ β -catenin signaling are identified to undergo a major influence regarding the activity of the osteoclasts and osteoblasts that leads to the disruptions in the bone homeostasis (Luo et al., 2025, p. 2). Additionally, after menopause women have no estrogen to help guard against bone resorption, leading to the release of cytokines like IL-6, IL-1 and TNF, all of which are pro-inflammatory. This type of cytokines is anti-osteoblastic and induces osteoclastogenesis (Fragkiadaki et al., 2019 p.2; Hsu et al., 2024; Wang, 2024 p.6). This also contributes to the nature of the bone destruction and the likelihood of fractures, which are closely related with each other, and hormones' changes and inflammatory processes in developing osteoporosis (Lu et al., 2025, p. 2; Yu

and Wang, 2016, p. 642). Further, bone homeostasis can be influenced by elevation of pro-inflammatory cytokines (such as IL-6, IL-1b, and TNFa) at age which also contribute to the elevation of bone homeostasis (Chen et al., 2023, p. 2). These cytokines differentiate and activate osteoclasts simultaneously, and upregulate the expression and the speed of the bone resorption (Liao et al., 2024, p. 8238). For instance, it was demonstrated in vitro that tumour necrosis factor- alpha can synergise with RANKL to stimulate the generation of osteoclasts by amplifying the stimulation of NF-kB and PI3K/Akt pathways. High levels of TNF-a (Zhang et al., 2022, p. 3) are found in postmenopausal osteoporotic women.

CONCLUSION

The paper includes detailed statistics on the complexity of the problem of osteoporotic-induced fractures in the geriatric patient as a biologic, behavioral and environmental outcome. A few factors will increase your risk of breaking a bone: old age, low levels of minerals in the bones, not getting enough certain nutrients, other underlying health conditions, taking certain medicines and your risk of falls. The results point to the fact that pharmacological intervention is not enough in fracture prevention but the

approach should be proactive and multifaceted. The inclusion of medical therapy, nutritional supplements, structured exercise and fall-prevention brands into a single plan turned out to be the most helpful one, in terms of preventing the risk of fractures and improving functional outcomes. Identification of high risk individuals at an early age should be an important component of good prevention, and should be followed by screening and risk stratification. The paper further illustrates how patient education and compliance to their treatment regimes is important to make sure that they are able to maximize on the prevention measures. This poses a big challenge to the reduction of fracture as low adherence. Community programs and improving the availability of services focused on preventing fractures are important contributors to closing the fracture risk gap between older age groups. Finally, a multi-disciplinary approach is important to reduce the burden of osteoporotic fracture; a mix between clinical practice, lifestyle modification and supporting health care policy. This would contribute significantly towards quality of life, making people self reliant, reducing the cost of health service to geriatric persons with fractured bones.

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