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## **Lifestyle Factors Associated with Osteoporosis Risk**

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## ABSTRACT

**Background:** Osteoporosis is the most prevalent metabolic bone disease, affecting roughly 200 million women worldwide and accounting for about 9 million osteoporotic fractures annually. Age, sex, and genetics shape baseline risk, but modifiable lifestyle behaviors explain a large share of the variance in bone mineral density (BMD) and fracture incidence.

**Aim:** This narrative review synthesizes current evidence on how physical activity, diet, tobacco, alcohol, caffeine, body weight, sleep, and stress influence osteoporosis risk, with clinical implications.

**Material and methods:** PubMed, Google Scholar, the Journal of Education, Health and Sport, Quality in Sport, and the Cochrane Library were searched for peer-reviewed publications from 1995 to 2026. Search terms combined "osteoporosis," "BMD," and "fracture" with each lifestyle factor of interest. Inclusion was restricted to original research, meta-analyses, systematic reviews, and authoritative guidelines.

**Results:** High-intensity resistance and impact training increased lumbar spine BMD by 2.9% and femoral neck BMD by 0.3% in osteopenic postmenopausal women. Calcium plus vitamin D supplementation reduced hip fracture risk by 30%. Vegan diets were associated with 43% higher total-fracture risk and a 2.3-fold higher hip-fracture risk. Current smoking nearly doubled lifetime hip-fracture risk. Heavy alcohol intake raised hip-fracture risk by 59%; light intake was neutral. Short sleep ( $\leq 5$  h) and chronic psychological stress were associated with lower BMD across multiple skeletal sites.

**Conclusions:** Lifestyle behaviors are the principal modifiable determinants of osteoporosis risk. Integrated programs combining weight-bearing exercise, adequate calcium and vitamin D, and avoidance of tobacco and excess alcohol offer the greatest fracture-prevention potential.

**Key words:** osteoporosis, bone mineral density, lifestyle, physical activity, calcium, vitamin D, fracture risk.

## Introduction

Osteoporosis has emerged as one of the defining chronic diseases of demographic aging. Defined as a systemic skeletal disorder characterized by low bone mass and microarchitectural deterioration that increases bone fragility and susceptibility to fracture, the condition has reached a global scale that places it alongside cardiovascular disease and dementia as a leading driver of disability among older adults. According to the most widely cited international epidemiological estimate, approximately 9.0 million osteoporotic fractures occurred worldwide in the year 2000, of which 1.6 million were hip fractures, 1.7 million were forearm fractures, and 1.4 million were clinical vertebral fractures, contributing to about 5.8 million disability-adjusted life-years lost<sup>1</sup> Subsequent

national and regional analyses have shown that the absolute number of fragility fractures has continued to rise, driven by the expansion of the population aged 65 years and older. The disease is characterized by a sustained imbalance between bone formation and bone resorption, with osteoclast-mediated resorption exceeding osteoblast-mediated formation across the bone remodeling cycle. This imbalance is most dramatic in postmenopausal women, in whom the loss of estrogen leads to de-repression of receptor activator of nuclear factor  $\kappa$ B ligand (RANKL) signaling and accelerated trabecular bone resorption<sup>2</sup>. Although the dominant role of estrogen withdrawal in postmenopausal osteoporosis has been recognized for decades, the contemporary view of osteoporosis treats the disease as a multifactorial condition in which non-modifiable factors, such as age, female sex, ethnicity, and genetic predisposition, interact with a substantial portfolio of modifiable lifestyle exposures that act across the entire life course. Recent evidence has highlighted that lifestyle behaviors are not merely secondary contributors but are foundational determinants of bone health. Physical activity patterns established in adolescence influence the magnitude of peak bone mass, which is the strongest single predictor of lifetime fracture risk; nutritional habits affect calcium and vitamin D status, hormonal milieu, and the substrate available for matrix formation; and habits such as cigarette smoking, alcohol use, and chronic stress exert direct biochemical effects on the bone remodeling unit.<sup>3,4</sup> Notably, much of the modifiable risk is concentrated in behaviors that are also implicated in cardiovascular and metabolic disease, raising the prospect that integrated preventive programs may yield benefits across multiple chronic conditions simultaneously.

Despite this knowledge, awareness of bone-relevant lifestyle factors remains low in the general population. A study of 450 perimenopausal Polish women aged 40 to 65 years documented insufficient knowledge of osteoporosis prevention strategies, with respondents systematically underestimating the contribution of cigarettes, alcohol, and coffee to skeletal vulnerability and reporting inadequate physical activity.<sup>5</sup> The gap between epidemiological knowledge and lay practice underscores the need for clinically actionable synthesis.

This narrative review aims to provide such a synthesis. After a brief overview of bone remodeling pathophysiology, the manuscript surveys the major lifestyle exposures linked to osteoporosis risk, including physical activity and sedentary behavior, dietary patterns and individual nutrients, tobacco and alcohol use, caffeine intake, body weight extremes, sleep, and psychological stress. The review closes with a discussion of the integrated clinical implications and a structured set of conclusions oriented toward primary and secondary prevention.

## **Material and methods**

The literature search supporting this narrative review was conducted across several major scientific databases, including PubMed, MEDLINE, Google Scholar, the Cochrane Library, the Journal of Education, Health and Sport (JEHS), and Quality in Sport (QS). The search strategy combined Medical Subject Headings and free-text terms related to the disease entity with terms describing each lifestyle factor of interest, using the Boolean string ("osteoporosis" OR "bone mineral density" OR "BMD" OR "osteoporotic fracture") AND ("physical activity" OR "exercise" OR "resistance training" OR "sedentary behavior" OR "calcium" OR "vitamin D" OR "protein" OR "sodium" OR "vegan" OR "vegetarian" OR "carbonated beverages" OR "Mediterranean diet" OR "smoking" OR "alcohol" OR "caffeine" OR "body mass index" OR "sleep duration" OR "stress").

The inclusion criteria were defined as:

1. Peer-reviewed original research, meta-analyses, systematic reviews, and authoritative international guidelines.
2. Studies published between 1995 and 2026, with priority given to publications from the last decade and seminal earlier works retained where they remain the canonical reference.
3. Articles addressing human populations, including pediatric, adult, perimenopausal, postmenopausal, and geriatric subgroups.
4. Reports from major international health organizations, including the World Health Organization (WHO), the International Osteoporosis Foundation (IOF), and the National Osteoporosis Foundation (NOF).

The exclusion criteria included non-peer-reviewed content, narrative commentaries without primary evidence synthesis, animal-only studies without translational relevance to human bone biology, and case reports with fewer than five participants. A total of 29 sources were selected for the final synthesis, including 5 mandatory publications and 24 additional sources identified through database search and citation tracking. The collected evidence was categorized thematically into sub-chapters reflecting the major modifiable lifestyle exposures, and key quantitative findings were extracted with attention to effect direction, magnitude, and confidence intervals where available.

## **Results / State of Knowledge**

### **Pathophysiology of bone remodeling and the rationale for lifestyle modification**

Bone is a metabolically active tissue that is continuously remodeled through the coordinated activity of three principal cell types: osteoblasts, which synthesize the collagenous matrix and orchestrate its mineralization; osteoclasts, which resorb mineralized matrix; and osteocytes, which are terminally differentiated osteoblasts embedded within the lacunar–canalicular network and which act

as the principal mechanosensory cells of the skeleton.<sup>6</sup> Under physiological conditions, formation and resorption are tightly coupled, and net bone mass is maintained from approximately the third decade of life through the fifth decade in women and somewhat later in men. Thereafter, an age-related uncoupling produces progressive bone loss, with women experiencing a sharply accelerated phase coinciding with menopause. The bone remodeling unit is sensitive to multiple endogenous and exogenous regulators. Estrogen, the most thoroughly characterized of these regulators in the context of postmenopausal osteoporosis, exerts a net anti-resorptive effect by suppressing RANKL and inducing osteoprotegerin (OPG) expression in osteoblast-lineage cells, thereby restraining osteoclast differentiation and activity.<sup>2</sup> Estrogen withdrawal at menopause de-represses this axis and elevates pro-inflammatory cytokines, including interleukin-1, interleukin-6, tumor necrosis factor alpha, and macrophage colony-stimulating factor, which together accelerate trabecular resorption and produce the characteristic acute bone loss of the first postmenopausal decade.<sup>2</sup> Glucocorticoid excess, whether iatrogenic or driven by chronic activation of the hypothalamic–pituitary–adrenal axis, further suppresses osteoblastogenesis and induces osteoblast and osteocyte apoptosis, providing a mechanistic link between chronic psychological stress and bone loss. Mechanical loading constitutes the second pillar of bone remodeling regulation. The classical observation that bone adapts its architecture to its functional demands, formalized as Wolff's law, has been refined through cellular and molecular evidence implicating osteocytes as the primary mechanosensors that convert interstitial fluid flow shear stress into biochemical signaling capable of orchestrating osteoblast and osteoclast activity at distant remodeling sites.<sup>6</sup> This mechanotransduction pathway provides the biological basis for the consistent observation that load-bearing physical activity increases BMD, whereas immobilization, microgravity, and sedentary behavior produce predictable losses. The integration of endocrine, mechanical, nutritional, and inflammatory inputs at the bone remodeling unit explains why lifestyle behaviors that act on any of these pathways can shift the net balance toward formation or toward resorption, and it provides the conceptual framework that organizes the remainder of this review.

## **Physical activity, exercise modalities, and sedentary behavior**

Physical activity is the most extensively studied modifiable determinant of osteoporosis risk. A recent narrative review by Stachowicz and colleagues identified physical inactivity, calcium deficiency, and alcohol consumption as the principal modifiable risk factors in older adults and concluded that regular movement-based interventions represent a scientifically proven preventive factor against osteoporosis, reducing the frequency of falls and increasing BMD.<sup>3</sup> Śliz and colleagues reached parallel conclusions in a comprehensive review of exercise in both prevention and treatment

of osteoporosis, emphasizing that benefits accrue only when activity is performed regularly and persistently across multiple years.<sup>4</sup>

### **Mechanism: from mechanical strain to bone formation**

The mechanostat model, an extension of Wolff's law, posits that bone responds to dynamic strain that exceeds a threshold, with high-magnitude and high-rate loading producing the most osteogenic stimulus.<sup>6</sup> Activities involving impact, ground reaction forces, and high muscular loading place the largest strains on the skeleton and therefore generate the most robust adaptive response. By contrast, activities such as cycling and swimming, which impose lower ground reaction forces, produce smaller increases in BMD despite cardiovascular benefits.

### **Resistance and high-impact training**

The clinical translation of this mechanism is best illustrated by the LIFTMOR randomized controlled trial conducted by Watson and colleagues, in which 101 postmenopausal women with low bone mass (T-score below  $-1.0$ ) were randomized to either eight months of supervised, twice-weekly high-intensity resistance and impact training or to a low-intensity home exercise program.<sup>7</sup> Lumbar spine BMD increased by  $2.9 \pm 2.8\%$  in the intervention group compared with a decline of  $1.2 \pm 2.8\%$  in controls ( $p < 0.001$ ), and femoral neck BMD increased by  $0.3 \pm 2.6\%$  versus a decline of  $1.9 \pm 2.6\%$  ( $p = 0.004$ ). Femoral neck cortical thickness rose by  $13.6 \pm 16.6\%$  in the intervention group, with greater than 90% adherence and a favorable safety profile, refuting prior concerns that high-load training is unsafe in osteopenic women.

A 2023 updated systematic review and meta-analysis by Mohebbi and colleagues consolidated the evidence base across 80 controlled trials and 5,581 postmenopausal participants, reporting standardized mean differences in BMD versus controls of 0.29 (95% CI 0.16–0.42) at the lumbar spine, 0.27 (95% CI 0.16–0.39) at the femoral neck, and 0.41 (95% CI 0.30–0.52) at the total hip.<sup>8</sup> The total hip showed the largest effect, and the absence of significant moderation by osteopenia/osteoporosis status, time since menopause, or supervision modality suggests that benefits are obtainable across a broad spectrum of postmenopausal women. A Polish-language synthesis published in JEHS by Oszczędlowski and colleagues reported convergent findings, with weight-bearing exercise particularly increasing BMD in postmenopausal women and lean mass acting as a fracture-protective factor that is partially independent of BMD.<sup>9</sup>

## **Public-health framing and dose**

A systematic review commissioned to inform the WHO physical activity guidelines for older adults synthesized 59 studies and reported moderate-certainty evidence that physical activity prevents osteoporosis, with an overall standardized mean difference of 0.15 (95% CI 0.05–0.25) for bone health outcomes and a larger effect at the lumbar spine (SMD 0.17) than at the femoral neck (SMD 0.09).<sup>10</sup> Effective programs lasted at least seven months and consisted of 60 or more minutes per session, two to three times weekly, ideally combining resistance and impact components. These findings underpin the WHO recommendation that adults aged 65 years and older perform multicomponent physical activity emphasizing functional balance and strength training on three or more days per week.

## **Sedentary behavior**

The complement of insufficient activity is sedentary behavior, which has emerged as an independent exposure of interest. A systematic review by McMichan and colleagues of seven observational studies in adults aged 65 years and older, in which sedentary behavior was objectively measured and BMD was assessed by dual-energy X-ray absorptiometry (DXA), reported that older adults spent more than eight hours per day in sedentary behavior, with predominantly negative or null associations between sedentary time and BMD at the femoral neck, pelvis, whole body, spine, and lower limb in men.<sup>11</sup> In women, the findings were heterogeneous, with four studies reporting positive associations and two reporting negative associations, reflecting methodological variability rather than absence of effect. The authors flagged the area as evidence-poor and called for future studies using device-based measurement.

## **Falls prevention through balance and functional training**

In older adults, BMD is only one determinant of fracture; the propensity to fall is the other. The Cochrane systematic review by Sherrington and colleagues synthesized 108 randomized controlled trials including 23,407 community-dwelling older adults across 25 countries and concluded with high-certainty evidence that exercise reduced the rate of falls by 23% (rate ratio 0.77, 95% CI 0.71–0.83) and the proportion of fallers by 15% (risk ratio 0.85, 95% CI 0.81–0.89).<sup>12</sup> Balance and functional exercises specifically reduced fall rate by 24%, and exercise reduced fall-related fractures by 27% (risk ratio 0.73, 95% CI 0.56–0.95). These findings establish balance training as a complementary intervention to BMD-oriented programs and reinforce the case for multicomponent regimens.

## **Nutritional behaviors**

Nutritional intake influences bone health through the supply of substrate for matrix mineralization, modulation of calcium balance, and regulation of pro- and anti-inflammatory signaling. The most important nutrients in this context are calcium, vitamin D, protein, vitamin K, magnesium, and the broader balance of acid- and base-producing foods.

| Nutrient             | Adults 19–50 y | Adults 51–70 y                  | Adults > 70 y / postmenopausal | Notes                                                |
|----------------------|----------------|---------------------------------|--------------------------------|------------------------------------------------------|
| Calcium              | 1000 mg/day    | 1000 mg (men) / 1200 mg (women) | 1200 mg/day                    | Combined dietary plus supplemental intake            |
| Vitamin D            | 600 IU/day     | 600 IU/day                      | 800–1000 IU/day                | ≥800 IU/day required for hip-fracture reduction      |
| Protein              | 0.8 g/kg/day   | 1.0–1.2 g/kg/day                | 1.0–1.2 g/kg/day               | Higher intakes are protective, not catabolic         |
| Vitamin K (K1 + K2)  | 90–120 µg/day  | 90–120 µg/day                   | 90–120 µg/day                  | K2 menaquinone-7 increasingly studied at 180 µg/day  |
| Magnesium            | 310–420 mg/day | 320–420 mg/day                  | 320–420 mg/day                 | Modest positive correlation with BMD                 |
| Sodium (upper limit) | < 2300 mg/day  | < 2000 mg/day                   | < 2000 mg/day                  | Each 100 mmol Na excretion ≈ 1 mmol Ca lost in urine |

**Table 1.** Recommended daily intakes for the principal bone-relevant nutrients

Source:<sup>13,14,15,16,17</sup>

## Calcium

Calcium is the principal mineral component of hydroxyapatite and the substrate from which the skeleton draws when serum levels fall. The strongest evidence for combined supplementation comes from the National Osteoporosis Foundation–commissioned meta-analysis by Weaver and colleagues, which pooled eight randomized controlled trials including 30,970 participants and reported that calcium plus vitamin D supplementation reduced total fracture risk by 15% (summary relative risk 0.85, 95% CI 0.73–0.98) and hip fracture risk by 30% (relative risk 0.70, 95% CI 0.56–0.87).<sup>13</sup> The effect was robust across community-dwelling and institutionalized populations and was apparent only when calcium was provided in combination with vitamin D, suggesting biological synergy.

## **Vitamin D**

Vitamin D acts on bone both indirectly, through modulation of intestinal calcium absorption and parathyroid hormone secretion, and directly, through receptor-mediated effects on osteoblast and osteoclast differentiation. The most authoritative dose-response analysis is the participant-level pooled analysis published in *The New England Journal of Medicine* by Bischoff-Ferrari and colleagues, which combined data from 11 double-blind randomized trials including 31,022 participants with a mean age of 76 years, of whom 91% were women.<sup>14</sup> At the highest actual-intake quartile, with a median of 800 IU/day, hip fracture risk fell by 30% (hazard ratio 0.70, 95% CI 0.58–0.86) and any nonvertebral fracture risk fell by 14% (hazard ratio 0.86, 95% CI 0.76–0.96). Lower intake quartiles showed no benefit, establishing 800 IU/day as the minimum effective dose for fracture prevention in older adults.

A nuance to this conclusion was demonstrated by Pajor and colleagues in a Polish study of 76 adults aged 20 to 43 years with low osteoporosis risk, in which DXA-measured BMD did not differ significantly between regular vitamin D3 supplementers and non-supplementers ( $p = 0.1753$ ).<sup>15</sup> The finding suggests that vitamin D supplementation may have limited incremental impact on BMD among individuals already at low risk, despite widespread supplementation in this population, and supports a targeted rather than universal supplementation strategy in low-risk young adults.

## **Dietary protein**

Historically, dietary protein was thought to promote bone loss through its acid load and consequent calciuria. This view has been revised by a National Osteoporosis Foundation systematic review and meta-analysis conducted by Shams-White and colleagues, which pooled 16 randomized controlled trials and 20 prospective cohort studies and reported a statistically significant net benefit of higher versus lower protein intake on lumbar spine BMD (0.52%, 95% CI 0.06–0.97; from five RCTs) and positive but non-significant trends at the total hip, femoral neck, and total body.<sup>16</sup> The findings refute the high-protein bone-loss hypothesis and support recommended intakes in the 1.0 to 1.2 g/kg/day range for older adults, particularly when combined with adequate calcium.

## **Sodium and dietary acid load**

High sodium intake produces an obligatory urinary calcium loss that, over decades, may contribute meaningfully to negative calcium balance. A two-year longitudinal study of 124 postmenopausal women conducted by Devine and colleagues found that both dietary calcium and urinary sodium excretion were independent significant determinants of bone-mass change at the hip and ankle, with halving daily sodium excretion producing bone-loss reduction equivalent to adding 891 mg/day of dietary calcium.<sup>17</sup> The magnitude of this effect underscores the relevance of population-level sodium reduction policies for skeletal health, although direct fracture-endpoint trials remain lacking.

## **Vegetarian and vegan dietary patterns**

Plant-based eating patterns have grown in popularity for ethical, environmental, and metabolic reasons, and their effect on bone health has therefore become a clinically important question. A recent narrative review by Łapiński and colleagues synthesized the evidence and concluded that vegetarians and vegans, particularly strict vegans, demonstrate lower BMD and higher fracture risk compared with omnivores, with the principal mechanisms identified as insufficient bioavailable calcium and protein intake, suppressed insulin-like growth factor-1 (IGF-1) signaling, and elevated homocysteine resulting from vitamin B12 deficiency.<sup>18</sup> The authors emphasize that plant-based diets are associated with compromised skeletal integrity if not properly balanced, requiring deliberate nutritional strategy and targeted supplementation to minimize fracture risk. The most robust quantitative evidence comes from the EPIC-Oxford prospective cohort study published by Tong and colleagues, which followed 54,898 participants for approximately 17.6 years and recorded 3,941 incident fracture cases.<sup>19</sup> Compared with meat eaters, vegans had a 43% higher total-fracture risk (hazard ratio 1.43, 95% CI 1.20–1.70) and a 2.31-fold higher hip-fracture risk (hazard ratio 2.31, 95% CI 1.66–3.22). Vegetarians and fish eaters also had elevated hip-fracture risk (hazard ratios 1.25 and 1.26 respectively). The risk was partly attenuated after adjustment for body mass index, dietary calcium, and dietary protein, identifying these as the principal modifiable mediators in plant-based eaters.

## **Carbonated beverages**

The effect of carbonated beverages on bone has been contested, with the balance of evidence pointing toward cola specifically rather than carbonation per se as the relevant exposure. The Framingham Osteoporosis Study analysis by Tucker and colleagues examined 1,413 women and 1,125 men and reported that daily cola drinkers, compared with those consuming less than one serving per month, had 3.7% lower femoral neck BMD and 5.4% lower BMD at Ward's area, with no effect on

spine BMD and no association in men.<sup>20</sup> Diet cola produced similar effects to regular cola, while decaffeinated cola was less strongly associated and other carbonated beverages were not, supporting phosphoric acid rather than caffeine or carbonation as the candidate mechanism.

### **Mediterranean dietary pattern**

Whole-diet patterns may capture interactions among nutrients that individual-nutrient analyses miss. The Women's Health Initiative analysis by Haring and colleagues followed 90,014 postmenopausal women for a median of 15.9 years and recorded 2,121 hip fractures and 28,718 total fractures.<sup>21</sup> Women in the highest versus lowest quintile of the alternate Mediterranean diet score had a 20% lower hip-fracture risk (hazard ratio 0.80, 95% CI 0.66–0.97), although the absolute risk reduction was modest at 0.29% and the number-needed-to-treat over the follow-up was 342. No significant association was observed for total fractures or for the Healthy Eating Index, DASH, or Alternate Healthy Eating Index scores, suggesting that the Mediterranean pattern's protective effect is hip-specific and modest in magnitude.

### **Vitamin K2 and other micronutrients**

Vitamin K is required for the gamma-carboxylation of osteocalcin, the major non-collagenous bone matrix protein. A 2022 systematic review and meta-analysis by Ma and colleagues of 16 randomized controlled trials including 6,425 subjects reported that vitamin K2 supplementation significantly improved lumbar spine BMD ( $p = 0.006$ ) and reduced fracture incidence (relative risk 0.43,  $p = 0.01$  after removing one heterogeneous trial).<sup>22</sup> Reductions in undercarboxylated osteocalcin confirmed the proposed mechanism, and no excess adverse events were observed (relative risk 1.03), supporting vitamin K2 as a candidate adjunct in postmenopausal osteoporosis.

### **Tobacco, alcohol, and caffeine**

#### **Cigarette smoking**

Cigarette smoking is the most established substance-related risk factor for osteoporosis. The foundational meta-analysis by Law and Hackshaw pooled 29 cross-sectional studies including 2,156 smokers and 9,705 non-smokers, together with 19 cohort and case-control studies covering 3,889 hip fractures, and reported that current smokers had a hip-fracture risk that was similar to non-smokers at age 50 but rose by 17% at age 60, 41% at age 70, 71% at age 80, and 108% at age 90.<sup>23</sup> Lifetime hip-fracture risk was 31% higher in women smokers and 40% higher in male smokers, with approximately one in eight hip fractures in women attributable to smoking. Postmenopausal BMD declined by an additional 2% per decade in smokers, producing a 6% deficit by age 80. Mechanistic studies have

implicated nicotine-induced reductions in osteoblast activity, accelerated metabolism of estradiol, and increased oxidative stress, all of which converge on accelerated bone resorption.

## **Alcohol**

The relationship between alcohol intake and osteoporosis follows a J-shaped curve, complicating simple risk attribution. A dose-response meta-analysis by Godos and colleagues synthesized BMD data from 46,916 patients and fracture data from 240,871 individuals, including 8,014 incident fractures.<sup>24</sup> Light alcohol consumption, defined as up to one or two standard drinks per day, was associated with higher BMD at the lumbar spine, femoral neck, and total hip compared with abstinence. Heavy intake at three or more drinks per day raised hip-fracture risk by 33% (relative risk 1.33, 95% CI 1.04–1.69), and intake of four or more drinks per day raised it by 59% (relative risk 1.59, 95% CI 1.23–2.05). Mechanisms of alcohol-induced bone loss include direct toxicity to osteoblasts, impaired hepatic activation of vitamin D, secondary hypogonadism, increased fall risk, and nutritional inadequacy in heavy drinkers. The J-curve does not constitute a public-health endorsement of alcohol consumption, given its other harms, but it does help reconcile mixed prior reports.

## **Caffeine**

Caffeine has a small and somewhat sex-divergent effect on bone. A meta-analysis by Liu and colleagues of 10 prospective studies including 214,059 participants reported that each additional cup of coffee per day was associated with a 3.5% increase in overall fracture risk, with the effect concentrated in women (+4.9% per cup) and a paradoxical reduction in men (−9% per cup).<sup>25</sup> Mechanistically, caffeine may produce a small, transient increase in urinary calcium excretion, but the magnitude of this effect is offset in many populations by the calcium content of milk added to coffee. The clinical implication is that high caffeine intake in postmenopausal women warrants attention only when it occurs in the context of low calcium intake, where the marginal calciuric effect could become meaningful.

## **Body weight and composition**

Body weight at both extremes influences bone health, and the relationship is non-linear. The individual-level meta-analysis by De Laet and colleagues pooled 12 prospective cohorts comprising approximately 60,000 men and women followed for more than 250,000 person-years and reported that a body mass index (BMI) of 20 kg/m<sup>2</sup> nearly doubled hip fracture risk relative to a BMI of 25 kg/m<sup>2</sup> (relative risk 1.95, 95% CI 1.71–2.22).<sup>26</sup> After adjustment for BMD, the association was largely abolished (relative risk 0.98), confirming that low BMI operates principally through reduced bone mass rather than through independent biomechanical pathways. The finding has clinical implications

for the assessment of fracture risk in older adults with unintentional weight loss, in patients with anorexia nervosa or other eating disorders, and in athletes operating in low-energy-availability states, in whom both low BMI and disordered eating compound skeletal vulnerability. Importantly, the protective effect of higher BMI flattens at the upper extreme. Although obese individuals have higher BMD on average, contemporary evidence indicates that bone quality is often compromised in obesity, with elevated fracture risk at non-vertebral sites such as the humerus and ankle, suggesting that the obesity–bone relationship is more nuanced than initially understood. Lean mass, rather than fat mass, appears to be the principal driver of the favorable component of the BMI–bone relationship, which provides another mechanistic argument for resistance training as a preventive intervention.

## **Sleep and psychological stress**

### **Sleep duration**

Sleep is increasingly recognized as a behavioral determinant of bone health that operates partly through endocrine and partly through behavioral pathways. The Women's Health Initiative analysis by Ochs-Balcom and colleagues of 11,084 postmenopausal women found that women sleeping five hours or fewer per night had BMD values 0.012 to 0.018 g/cm<sup>2</sup> lower than those sleeping seven hours at all four measured sites, including the whole body, total hip, femoral neck, and spine, with significantly increased odds of low bone mass and hip osteoporosis (odds ratio approximately 1.22 for hip osteoporosis).<sup>27</sup> Plausible mechanisms include disruption of the diurnal rhythm of bone turnover markers, reduced growth hormone secretion during slow-wave sleep, and shared lifestyle clustering of short sleep with smoking, sedentary behavior, and nutritional inadequacy.

### **Chronic psychological stress**

Chronic psychological stress activates the hypothalamic–pituitary–adrenal axis and produces sustained elevations in circulating cortisol that mimic, in attenuated form, the effects of exogenous glucocorticoid therapy. A mechanistic narrative review by Ng and Chin mapped the principal pathways: cortisol upregulates RANKL and downregulates OPG in osteoblasts, increases osteoblast and osteocyte apoptosis, and impairs osteoblastogenesis.<sup>28</sup> Concurrently, chronic stress produces low-grade systemic inflammation, sympathetic hyperactivation, elevated parathyroid hormone, reduced gonadal hormone secretion, and altered leptin signaling, all of which compound the direct cortisol effects on bone. Although high-quality fracture-endpoint data remain limited, the convergence of mechanistic evidence supports recognition of chronic stress as an independent modifiable risk factor.

## **Special populations and the integrated risk profile**

### **Perimenopausal women**

The perimenopausal period is the inflection point at which lifestyle behaviors translate most directly into long-term skeletal trajectory. Rudnicka-Drożak and colleagues evaluated 450 Polish women aged 40 to 65 years and documented insufficient knowledge of osteoporosis prevention and inadequate health-seeking behaviors, with respondents underestimating the contributions of cigarettes, coffee, and alcohol and reporting suboptimal physical activity levels.<sup>5</sup> The authors concluded that insufficient knowledge of prevention and health-seeking behaviors contributes to the rising incidence of osteoporosis and that targeted educational interventions in this population could materially reduce disease progression.

### **Older adults and the osteosarcopenia construct**

In adults aged 65 years and older, osteoporosis frequently coexists with sarcopenia, the age-related loss of muscle mass and function, in a syndrome now termed osteosarcopenia. A narrative review by Kirk and colleagues reported that the prevalence of osteosarcopenia in community-dwelling older adults ranges from 5% to 37% and rises to approximately 46% after low-trauma fracture and 17.1% to 96.3% after hip fracture.<sup>29</sup> Prevalence rises sharply with age, from 14.3% in men aged 60–64 years to 59.4% at 75 years and older, and from 20.3% to 48.3% across the same range in women. Osteosarcopenia independently predicts falls, fractures, and mortality beyond either osteoporosis or sarcopenia alone, providing a biologically integrated rationale for combined nutritional and resistance-training interventions in older adults.

**Table 2.** Major lifestyle behaviors and their reported associations with bone mineral density and fracture risk,

| Behavior                                    | Direction of effect         | Representative effect size                         | Population                     |
|---------------------------------------------|-----------------------------|----------------------------------------------------|--------------------------------|
| High-intensity resistance + impact training | Protective                  | +2.9% lumbar BMD; +0.3% femoral neck BMD over 8 mo | Postmenopausal women, T < -1.0 |
| Multicomponent exercise                     | Protective                  | SMD 0.41 total hip BMD vs. controls                | Postmenopausal women           |
| Sedentary behavior > 8 h/day                | Detrimental                 | Negative or null associations with BMD in men      | Adults ≥ 65 y                  |
| Calcium + vitamin D ≥ 800 IU                | Protective                  | Hip fracture RR 0.70                               | Older adults                   |
| Higher dietary protein (1.0–1.2 g/kg)       | Protective                  | +0.52% lumbar BMD vs. lower intake                 | Adults                         |
| Vegan diet                                  | Detrimental                 | Hip fracture HR 2.31 vs. meat eaters               | Adults, EPIC-Oxford            |
| Daily cola consumption                      | Detrimental                 | −3.7% femoral neck BMD                             | Older women                    |
| Mediterranean diet (high adherence)         | Protective                  | Hip fracture HR 0.80                               | Postmenopausal women           |
| Current cigarette smoking                   | Detrimental                 | Hip fracture risk +71% at age 80                   | Adults                         |
| Heavy alcohol (≥ 4 drinks/day)              | Detrimental                 | Hip fracture RR 1.59                               | Adults                         |
| Coffee per cup/day                          | Mildly detrimental in women | Fracture risk +4.9% per cup                        | Women                          |
| Low BMI (20 vs. 25 kg/m <sup>2</sup> )      | Detrimental                 | Hip fracture RR 1.95                               | Adults                         |
| Short sleep (≤ 5 h/night)                   | Detrimental                 | OR 1.22 for hip osteoporosis                       | Postmenopausal women           |
| Chronic psychological stress                | Detrimental                 | Mechanistic; effect size variable                  | Adults                         |

with representative effect sizes

Sources:<sup>7,8,11,13,14,16,18,19,20,21,23,24,25,26,27,28</sup>

## Integrated lifestyle programs

The clinical question that follows from this evidence is whether integrated lifestyle programs produce additive or synergistic benefits relative to single-component interventions. The literature on this question is less mature than the single-exposure literature, but converging signals across the WHO physical activity guidelines, the National Osteoporosis Foundation nutrition meta-analyses, and the Cochrane falls-prevention review support a multicomponent model that combines weight-bearing and resistance exercise, balance training, calcium and vitamin D adequacy, smoking cessation, and moderation of alcohol intake.<sup>10,12,13</sup>

**Table 3.** Suggested exposure parameters for the major lifestyle interventions associated with reduced

| Intervention                 | Frequency | Duration     | Intensity / dose                 | Primary outcome  |
|------------------------------|-----------|--------------|----------------------------------|------------------|
| Resistance + impact training | 2–3×/week | ≥ 6–8 months | ≥ 80% 1RM, multi-set             | BMD (spine, hip) |
| Multicomponent exercise      | 3×/week   | Ongoing      | Moderate to vigorous             | BMD, falls       |
| Balance training             | 3×/week   | Ongoing      | Progressive                      | Falls, fractures |
| Calcium intake               | Daily     | Lifelong     | 1000–1200 mg/day                 | BMD, fractures   |
| Vitamin D                    | Daily     | Lifelong     | ≥ 800 IU/day in older adults     | Hip fractures    |
| Protein intake               | Daily     | Lifelong     | 1.0–1.2 g/kg/day in older adults | BMD              |
| Smoking cessation            | —         | Permanent    | Complete                         | Fractures, BMD   |
| Alcohol moderation           | Daily     | Lifelong     | ≤ 2 drinks/day                   | Fractures        |
| Sleep duration               | Nightly   | Lifelong     | 7–8 h                            | BMD              |

osteoporosis risk

Source:<sup>7,11,13,15,16,23,24,26,27</sup>

## Discussion

The body of evidence reviewed here supports four overarching propositions. First, lifestyle behaviors collectively account for a substantial proportion of the modifiable variance in bone mineral density and fracture risk across the life course. Second, the magnitude of the effect attributable to any single behavior is typically modest in absolute terms but biologically and clinically meaningful when integrated across decades of exposure. Third, the major lifestyle exposures act through partially overlapping pathways, including mechanical loading, calcium balance, hormonal signaling, low-grade inflammation, and oxidative stress, which provides a mechanistic basis for the observation that multicomponent interventions tend to outperform single-component ones. Fourth, the gap between epidemiological knowledge and population-level practice remains substantial, and educational interventions targeted to high-leverage life stages, particularly perimenopause and early older adulthood, are warranted.<sup>3,5</sup>

A recurring theme across the lifestyle exposures is the importance of dose and chronicity. The LIFTMOR trial demonstrated that high-intensity resistance and impact training is safe and effective in postmenopausal women with osteopenia, but only at adherence rates exceeding 90% and across at least eight months of supervised training.<sup>7</sup> The vitamin D dose-response analysis identified 800 IU/day as the threshold for hip-fracture reduction, with no benefit at lower doses.<sup>14</sup> The alcohol J-curve indicates that the harmful effects of alcohol on bone manifest at three or more drinks per day, with a threshold rather than a graded effect.<sup>24</sup> These dose-response relationships imply that public-health messaging should emphasize specific quantitative targets rather than vague exhortations to "exercise more" or "drink less," because the relevant biological thresholds are non-trivial and adherence is itself a major determinant of outcome. A second recurring theme is the complexity of the evidence on apparently simple exposures. The vegan-diet literature illustrates this complexity: the EPIC-Oxford finding of a 2.31-fold higher hip-fracture risk in vegans is among the largest effect sizes for any modifiable behavior in this review, but the effect attenuates after adjustment for BMI, calcium, and protein, indicating that the harm is mediated by a small set of specific nutritional inadequacies rather than by plant-based eating per se.<sup>18,19</sup> This nuance is clinically important because it implies that vegans willing to plan their diet carefully, supplement appropriately, and maintain adequate body weight can substantially mitigate their fracture risk. Similarly, the Tucker analysis of cola implicates phosphoric acid rather than carbonation or caffeine, narrowing the actionable target.<sup>20</sup> In each case, careful mechanistic interpretation prevents overgeneralized recommendations.

A third theme is the convergence of bone-relevant lifestyle behaviors with cardiovascular and metabolic health. Smoking cessation, regular physical activity, adequate vitamin D, moderate alcohol, and Mediterranean dietary patterns all carry benefits for cardiovascular disease, type 2 diabetes, and certain cancers, with the corollary that integrated chronic-disease prevention programs are likely to be more cost-effective than disease-specific programs. This integration is particularly relevant in primary care, where the same brief counseling encounter can address multiple risk profiles simultaneously. Several limitations of the underlying evidence base warrant acknowledgment. Many of the most robust studies focus on postmenopausal women, with comparatively limited evidence in men and in younger adults; the effect sizes derived from these populations may not generalize cleanly. The evidence on sedentary behavior, sleep, and stress remains immature relative to the evidence on physical activity, calcium, and smoking, and future studies using device-based measurement of activity and sleep will be needed to refine effect estimates.<sup>11,27</sup> The evidence on vitamin D in low-risk populations is more equivocal than the evidence in deficient or older populations, as illustrated by the Pajor finding of no BMD difference in young, low-risk Polish adults,<sup>15</sup> suggesting that a one-size-fits-all supplementation policy is unlikely to be optimal. Finally, the integration of lifestyle interventions with pharmacological therapy in established osteoporosis remains an underexplored area, and the question of whether lifestyle modification can extend the durability of bisphosphonate, denosumab, or anabolic therapy in real-world practice deserves further trial evidence.

The "vicious cycle" of inactivity, sarcopenia, falls, and fracture observed in older adults<sup>29</sup> is the mirror image of the virtuous cycle that integrated lifestyle programs can establish. Adequate protein intake supports muscle protein synthesis; resistance training translates protein into functional strength; functional strength reduces fall risk; reduced fall risk preserves activity, social engagement, and ongoing exposure to mechanical loading on bone. Each component reinforces the others, and the practical implication for clinical practice is that the marginal value of adding components to a partial program is likely to be high. Conversely, a single-component program, however well executed, will leave substantial preventable risk on the table.

## **Conclusions**

Osteoporosis is a multifactorial disease in which lifestyle behaviors collectively determine a substantial fraction of the modifiable risk, with effects that integrate across the entire life course from peak bone mass acquisition in adolescence to fracture incidence in late older adulthood. Weight-bearing exercise, resistance training, and balance training are the most consistently protective lifestyle interventions, with high-intensity resistance and impact training producing measurable BMD gains and falls-prevention programs reducing fracture risk by approximately 27%. Adequate calcium and vitamin D intake, with vitamin D at or above 800 IU/day in older adults, is foundational; dietary protein at 1.0 to 1.2 g/kg/day is protective rather than catabolic; and whole-diet patterns such as the Mediterranean pattern offer modest additional benefit at the hip. Vegan and strict vegetarian diets are associated with elevated fracture risk that can be largely mitigated through deliberate planning of

calcium, protein, vitamin B12, and energy intake. Cigarette smoking and heavy alcohol consumption are the most clearly detrimental substance exposures, while caffeine exerts a small sex-divergent effect that is clinically relevant only in the context of low calcium intake. Short sleep duration, chronic psychological stress, and low body mass index are emerging exposures whose mechanistic plausibility and observational support warrant their inclusion in comprehensive lifestyle counseling, even as further evidence accumulates. The greatest preventive return is likely to come from integrated, multicomponent lifestyle programs initiated in early adulthood, reinforced through perimenopause, and sustained into older age, with primary care and public-health systems serving as the principal delivery channels.

## **Disclosure**

### **Author's Contribution**

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### **References**

1. Johnell, O., & Kanis, J. A. (2006). An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporosis International*, 17(12), 1726–1733. <https://doi.org/10.1007/s00198-006-0172-4>
2. Cheng, C.-H., Chen, L.-R., & Chen, K.-H. (2022). Osteoporosis due to hormone imbalance: An overview of the effects of estrogen deficiency and glucocorticoid overuse on bone turnover. *International Journal of Molecular Sciences*, 23(3), 1376. <https://doi.org/10.3390/ijms23031376>
3. Stachowicz, H., Mazurek, J., Adamczyk, M., Podrażka, M., Lenart, J., Baran, A., ... Stachowicz, N. (2024). The significance of physical activity in the prevention of osteoporosis in older adults. *Journal of Education, Health and Sport*, 67, 55043. <https://doi.org/10.12775/JEHS.2024.67.55043>
4. Śliz, J., Popławska, N. A., Skorupska, M., Czechtka, M. J., Woźniak, K., & Martka, M. M. (2024). The role of sport in human health: Prevention and treatment of osteoporosis. *Quality in Sport*, 16, 52915. <https://doi.org/10.12775/QS.2024.16.52915>
5. Rudnicka-Drożak, E., Jankowski, K., Jankowska, P., Majcher, M., & Jankowska, A. (2019). Evaluation of selected health seeking behaviours and prevention of osteoporosis in perimenopausal women. *Journal of Education, Health and Sport*, 9(3), 364–369. <https://apcz.umk.pl/JEHS/article/view/6709>
6. Chen, J. H., Liu, C., You, L., & Simmons, C. A. (2010). Boning up on Wolff's law: Mechanical regulation of the cells that make and maintain bone. *Journal of Biomechanics*, 43(1), 108–118. <https://doi.org/10.1016/j.jbiomech.2009.09.016>
7. Watson, S. L., Weeks, B. K., Weis, L. J., Harding, A. T., Horan, S. A., & Beck, B. R. (2018). High-intensity resistance and impact training improves bone mineral density and physical function in postmenopausal women with osteopenia and osteoporosis: The LIFTMOR

- randomized controlled trial. *Journal of Bone and Mineral Research*, 33(2), 211–220. <https://doi.org/10.1002/jbmr.3284>
8. Mohebbi, R., Shojaa, M., Kohl, M., von Stengel, S., Jakob, F., Kersch-Schindl, K., et al. (2023). Exercise training and bone mineral density in postmenopausal women: An updated systematic review and meta-analysis of intervention studies with emphasis on potential moderators. *Osteoporosis International*, 34(7), 1145–1178. <https://doi.org/10.1007/s00198-023-06682-1>
  9. Oszczyński, P., Niewęglowski, K., Madoń, B., Nowaczek, J., & Giermasiński, A. (2021). Impact of physical activity on incidence of osteoporotic fractures – A review. *Journal of Education, Health and Sport*, 11(9), 196–207. <https://doi.org/10.12775/JEHS.2021.11.09.025>
  10. Pinheiro, M. B., Oliveira, J., Bauman, A., Fairhall, N., Kwok, W., & Sherrington, C. (2020). Evidence on physical activity and osteoporosis prevention for people aged 65+ years: A systematic review to inform the WHO guidelines on physical activity and sedentary behaviour. *International Journal of Behavioral Nutrition and Physical Activity*, 17(1), 150. <https://doi.org/10.1186/s12966-020-01040-4>
  11. McMichan, L., Dick, M., Skelton, D. A., Chastin, S. F. M., Owen, N., Dunstan, D. W., et al. (2021). Sedentary behaviour and bone health in older adults: A systematic review. *Osteoporosis International*, 32(8), 1487–1497. <https://doi.org/10.1007/s00198-021-05918-2>
  12. Sherrington, C., Fairhall, N. J., Wallbank, G. K., Tiedemann, A., Michaleff, Z. A., Howard, K., et al. (2019). Exercise for preventing falls in older people living in the community. *Cochrane Database of Systematic Reviews*, 2019(1), CD012424. <https://doi.org/10.1002/14651858.CD012424.pub2>
  13. Weaver, C. M., Alexander, D. D., Boushey, C. J., Dawson-Hughes, B., Lappe, J. M., LeBoff, M. S., et al. (2016). Calcium plus vitamin D supplementation and risk of fractures: An updated meta-analysis from the National Osteoporosis Foundation. *Osteoporosis International*, 27(1), 367–376. <https://doi.org/10.1007/s00198-015-3386-5>
  14. Bischoff-Ferrari, H. A., Willett, W. C., Orav, E. J., Lips, P., Meunier, P. J., Lyons, R. A., et al. (2012). A pooled analysis of vitamin D dose requirements for fracture prevention. *New England Journal of Medicine*, 367(1), 40–49. <https://doi.org/10.1056/NEJMoa1109617>
  15. Pajor, K., Szpyt, J., & Lubkowska, A. (2019). Effect of vitamin D supplementation on bone mineral density in people with low risk of osteoporosis. *Journal of Education, Health and Sport*, 9(9), 1162–1174. <https://apcz.umk.pl/JEHS/article/view/7555>
  16. Shams-White, M. M., Chung, M., Du, M., Fu, Z., Insogna, K. L., Karlsen, M. C., et al. (2017). Dietary protein and bone health: A systematic review and meta-analysis from the National Osteoporosis Foundation. *American Journal of Clinical Nutrition*, 105(6), 1528–1543. <https://doi.org/10.3945/ajcn.116.145110>
  17. Devine, A., Criddle, R. A., Dick, I. M., Kerr, D. A., & Prince, R. L. (1995). A longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women. *American Journal of Clinical Nutrition*, 62(4), 740–745. <https://doi.org/10.1093/ajcn/62.4.740>
  18. Łapiński, M., Zimnoch-Włodarczyk, A., Zinko, O., Napierała, M., Ciecierska, K., Wasilkowska, K., & Łatkowska, S. (2026). Vegetarian diets and skeletal integrity: Mechanisms, risks, and future perspectives. *Quality in Sport*, 50, 68012. <https://doi.org/10.12775/QS.2026.50.68012>
  19. Tong, T. Y. N., Appleby, P. N., Armstrong, M. E. G., Fensom, G. K., Knuppel, A., Papier, K., et al. (2020). Vegetarian and vegan diets and risks of total and site-specific fractures: Results from the prospective EPIC-Oxford study. *BMC Medicine*, 18(1), 353. <https://doi.org/10.1186/s12916-020-01815-3>

20. Tucker, K. L., Morita, K., Qiao, N., Hannan, M. T., Cupples, L. A., & Kiel, D. P. (2006). Colas, but not other carbonated beverages, are associated with low bone mineral density in older women: The Framingham Osteoporosis Study. *American Journal of Clinical Nutrition*, 84(4), 936–942. <https://doi.org/10.1093/ajcn/84.4.936>
21. Haring, B., Crandall, C. J., Wu, C., LeBlanc, E. S., Shikany, J. M., Carbone, L., et al. (2016). Dietary patterns and fractures in postmenopausal women: Results from the Women’s Health Initiative. *JAMA Internal Medicine*, 176(5), 645–652. <https://doi.org/10.1001/jamainternmed.2016.0482>
22. Ma, M. L., Ma, Z. J., He, Y. L., Sun, H., Yang, B., Ruan, B. J., et al. (2022). Efficacy of vitamin K2 in the prevention and treatment of postmenopausal osteoporosis: A systematic review and meta-analysis of randomized controlled trials. *Frontiers in Public Health*, 10, 979649. <https://doi.org/10.3389/fpubh.2022.979649>
23. Law, M. R., & Hackshaw, A. K. (1997). A meta-analysis of cigarette smoking, bone mineral density and risk of hip fracture: Recognition of a major effect. *BMJ*, 315(7112), 841–846. <https://doi.org/10.1136/bmj.315.7112.841>
24. Godos, J., Giampieri, F., Chisari, E., Micek, A., Paladino, N., Forbes-Hernández, T. Y., et al. (2022). Alcohol consumption, bone mineral density, and risk of osteoporotic fractures: A dose–response meta-analysis. *International Journal of Environmental Research and Public Health*, 19(3), 1515. <https://doi.org/10.3390/ijerph19031515>
25. Liu, H., Yao, K., Zhang, W., Zhou, J., Wu, T., & He, C. (2012). Coffee consumption and risk of fractures: A meta-analysis. *Archives of Medical Science*, 8(5), 776–783. <https://doi.org/10.5114/aoms.2012.31612>
26. De Laet, C., Kanis, J. A., Odén, A., Johanson, H., Johnell, O., Delmas, P., et al. (2005). Body mass index as a predictor of fracture risk: A meta-analysis. *Osteoporosis International*, 16(11), 1330–1338. <https://doi.org/10.1007/s00198-005-1863-y>
27. Ochs-Balcom, H. M., Hovey, K. M., Andrews, C., Cauley, J. A., Hale, L., Li, W., et al. (2020). Short sleep is associated with low bone mineral density and osteoporosis in the Women’s Health Initiative. *Journal of Bone and Mineral Research*, 35(2), 261–268. <https://doi.org/10.1002/jbmr.3879>
28. Ng, J. S., & Chin, K. Y. (2021). Potential mechanisms linking psychological stress to bone health. *International Journal of Medical Sciences*, 18(3), 604–614. <https://doi.org/10.7150/ijms.50680>
29. Kirk, B., Zanker, J., & Duque, G. (2020). Osteosarcopenia: Epidemiology, diagnosis, and treatment—facts and numbers. *Journal of Cachexia, Sarcopenia and Muscle*, 11(3), 609–618. <https://doi.org/10.1002/jcsm.12567>
30. Kanis, J. A., McCloskey, E. V., Johansson, H., Oden, A., Melton, L. J., III, & Khaltayev, N. (2008). A reference standard for the description of osteoporosis. *Bone*, 42(3), 467–475. <https://doi.org/10.1016/j.bone.2007.11.001>
31. Wolski, D., Bednarski, J., Łuszczewska-Sierakowska, I., Michalik, J., Wawrzyniak, A., Nowicki, G., Bodajko-Grochowska, A., & Kosiński, J. (2018). Relationships between body weight and percentage body fat in the body and the development of osteopenia and osteoporosis. *Journal of Education, Health and Sport*, 8(9), 727–737. <https://apcz.umk.pl/JEHS/article/view/5925>
32. Rizzoli, R., Biver, E., Brennan-Speranza, T. C., Lappe, J. M., Heaney, R. P., Weaver, C. M., & Bonjour, J.-P. (2018). Benefits and safety of dietary protein for bone health—An expert consensus paper endorsed by the European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases and by the International

- Osteoporosis Foundation. *Osteoporosis International*, 29(9), 1933–1948. <https://doi.org/10.1007/s00198-018-4534-5>
33. Sözen, T., Özışık, L., & Başaran, N. Ç. (2017). An overview and management of osteoporosis. *European Journal of Rheumatology*, 4(1), 46–56. <https://doi.org/10.5152/eurjrheum.2016.048>
  34. Compston, J., Cooper, A., Cooper, C., Gittoes, N., Gregson, C., Harvey, N., Hope, S., Kanis, J. A., McCloskey, E. V., Poole, K. E. S., Reid, D. M., Selby, P., Thompson, F., Thurston, A., & Vine, N. (2017). UK clinical guideline for the prevention and treatment of osteoporosis. *Archives of Osteoporosis*, 12(1), 43. <https://doi.org/10.1007/s11657-017-0324-5>
  35. Martínez-Ramírez, M. J., Palma, S., Martínez-González, M. A., Delgado-Martínez, A. D., & de la Fuente-Arrillaga, C. (2007). Dietary patterns and fracture risk in elderly men and women. *The American Journal of Clinical Nutrition*, 86(4), 1051–1058. <https://doi.org/10.1093/ajcn/86.4.1051>
  36. Gopinath, B., Harris, D. C., Flood, V. M., Burlutsky, G., & Mitchell, P. (2011). Physical activity as a determinant of successful aging over ten years. *The Scientific World Journal*, 11, 1987–1992. <https://doi.org/10.1100/2011/984573>
  37. Kelley, G. A., Kelley, K. S., & Kohrt, W. M. (2013). Exercise and bone mineral density in men: A meta-analysis of randomized controlled trials. *Bone*, 53(1), 103–111. <https://doi.org/10.1016/j.bone.2012.11.029>