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### Review Article

## "MENOPAUSAL OSTEOPOROSIS: HOMOEOPATHIC MANAGEMENT AND DIET"

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#### ABSTRACT

**Background:** Menopausal osteoporosis (Type I osteoporosis) is a systemic skeletal disorder driven by the sharp decline in oestrogen following natural or surgical menopause, resulting in accelerated bone resorption, reduced bone mineral density (BMD), and heightened risk of fragility fractures. It affects an estimated 200 million women globally, with India reporting over 46 million at-risk post-menopausal women. Despite available pharmacological options—bisphosphonates, selective oestrogen receptor modulators (SERMs), and denosumab—long-term therapeutic compliance remains poor due to adverse effects. This has intensified clinical interest in complementary and integrative approaches. Homoeopathy, through individualised constitutional prescribing based on the principle of similia similibus curentur, and evidence-informed dietary management together represent a compelling multi-dimensional strategy for bone health in post-menopausal women.

**Methods:** A comprehensive narrative review was conducted using literature from PubMed, Scopus, Google Scholar, and AYUSH Research Portal, covering publications from 1988 to 2024. Search terms included: 'menopausal osteoporosis,' 'postmenopausal bone loss,' 'homoeopathy AND osteoporosis,' 'Calcarea Carbonica bone,' 'Symphytum bone healing,' 'phytoestrogens AND BMD,' 'calcium Vitamin D fracture prevention,' and 'anti-inflammatory diet bone health.' Inclusion criteria comprised original research articles, systematic reviews, meta-analyses, and authoritative materia medica texts. Seventeen peer-reviewed references were selected and synthesised; homoeopathic materia medica sources (Kent, Boericke, Schuessler) were incorporated for remedy profiling.

**Results:** The pathophysiology centres on oestrogen withdrawal-induced upregulation of RANKL and suppression of osteoprotegerin (OPG), driving net osteoclast dominance and progressive bone loss of 2–8% annually in early post-menopausal years. Ten key homoeopathic remedies—including Calcarea Carbonica,

Calcarea Phosphorica, Symphytum Officinale, Silicea, Phosphorus, Sepia, Lachesis, Ruta Graveolens, Fluoric Acid, and Natrum Muriaticum—were identified with well-characterised constitutional and musculoskeletal profiles. Biochemic tissue salts (Calc. Phos. 6X, Calc. Fluor. 12X, Silicea 12X) were identified as adjunctive bone-mineralisation supports. Dietary analysis confirmed that post-menopausal women require 1,000–1,200 mg/day of elemental calcium, 800–2,000 IU/day of Vitamin D3, and 40–80 mg/day of soy isoflavone aglycones—the latter associated with a ~54% reduction in lumbar spine bone loss versus placebo. Ragi (Eleusine coracana, 344 mg calcium/100g) was highlighted as a culturally appropriate high-calcium Indian dietary staple.

**Discussion:** Homoeopathic constitutional treatment addresses the whole-person vulnerability underlying menopausal osteoporosis—including miasmatic predisposition, defective calcium assimilation, and menopausal symptom burden—in a manner complementary to, rather than replacing, conventional antiresorptive pharmacotherapy when fracture risk thresholds indicate intervention. The integrative framework proposed combines FRAX-guided risk stratification, homoeopathic constitutional prescribing, targeted nutritional optimisation, weight-bearing exercise, and fall-prevention strategies. Limitations include the sparse RCT evidence base for homoeopathic interventions and the predominance of Western populations in nutritional epidemiology studies.

**Conclusion:** Menopausal osteoporosis is optimally managed through an integrative, patient-centred approach. Homoeopathic constitutional prescribing and evidence-based dietary strategies—particularly calcium, Vitamin D, phytoestrogens, and anti-inflammatory foods from the Indian dietary tradition—constitute meaningful, accessible, and well-tolerated adjuncts to conventional bone health management. This review advocates for trans-disciplinary research and clinical collaboration to establish rigorous protocols within the integrative medicine framework endorsed by J-AIM.

**Keywords:** Menopausal osteoporosis; Homoeopathy; Bone mineral density; Calcarea Carbonica; Symphytum Officinale; Phytoestrogens; Dietary management; Post-menopausal women; Integrative medicine; RANKL/OPG axis

## 1. Introduction

Osteoporosis is a systemic skeletal disorder characterised by reduced bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and susceptibility to fracture. Among its various aetiological subtypes, menopausal (postmenopausal) osteoporosis—designated Type I—stands as one of the most clinically significant, affecting an estimated 200 million women worldwide [1]. The hallmark pathophysiological event is the precipitous decline of oestrogen following natural or surgical menopause, which disrupts the dynamic equilibrium between osteoblastic bone formation and osteoclastic bone resorption in favour of the latter.

India is currently experiencing a rapidly ageing female demographic. An estimated 46 million Indian women above the age of 50 are at risk of osteoporotic fractures, yet osteoporosis remains significantly underdiagnosed and under-treated in the subcontinent [2]. Hip fractures in this population carry a one-year mortality of up to 20–25%, and vertebral fractures contribute substantially to chronic pain, kyphotic deformity, and functional impairment.

Contemporary pharmacotherapy—comprising bisphosphonates, selective oestrogen receptor modulators (SERMs), denosumab, and parathyroid hormone analogues—provides measurable benefits in reducing fracture risk. However, long-term use is limited by adverse effects including osteonecrosis of the jaw, atypical femoral fractures, cardiovascular risks, and poor patient adherence [3]. This therapeutic gap has fuelled interest in integrative and complementary approaches, including homoeopathy.

Homoeopathy, founded by Samuel Hahnemann on the principle of *similia similibus curentur* (like cures like), employs ultra-diluted potentised remedies individualised to the patient's totality of symptoms—physical, mental, and constitutional. Several homoeopathic remedies have a strong traditional and clinical profile in bone metabolism disorders. Simultaneously, nutritional science has advanced our understanding of the role of specific macronutrients and micronutrients in bone mineral density (BMD) maintenance.

This review aims to: (1) outline the pathophysiology and epidemiology of menopausal osteoporosis; (2) present an evidence-informed overview of homoeopathic remedies applicable to this condition; (3) summarise the dietary and nutritional strategies supporting bone health in post-menopausal women; and (4) propose an

integrative framework for clinical practice in alignment with the trans-disciplinary ethos of the Journal of Ayurveda and Integrative Medicine (J-AIM).

## 2. Epidemiology of Menopausal Osteoporosis

Osteoporosis is broadly defined by the World Health Organisation (WHO) as a bone mineral density (BMD) T-score of  $-2.5$  or below at the femoral neck, total hip, or lumbar spine as measured by dual-energy X-ray absorptiometry (DXA). The condition becomes exponentially more prevalent with age in women, primarily due to the accelerated bone loss that accompanies the menopausal transition.

In the first five to seven years after menopause, women lose approximately 2–3% of cortical bone and up to 5–8% of trabecular bone annually, driven by the surge in bone resorption that follows oestrogen withdrawal [4]. This rate decelerates thereafter to approximately 0.5–1% per year, but cumulative lifetime bone loss may reach 40–50% in trabecular sites.

Globally, one in three women over the age of 50 will experience an osteoporotic fracture during their remaining lifetime. Vertebral fractures are the most common, followed by wrist (Colles) fractures and hip fractures. Hip fractures carry the greatest morbidity and mortality. In Southeast Asia, including India, the burden is compounded by widespread Vitamin D deficiency (estimated in 60–80% of the population), low dairy consumption in certain communities, and cultural restrictions on sun exposure in women [5].

**Table 1: Risk Factors for Menopausal Osteoporosis**

| Category    | Modifiable Risk Factors                            | Non-Modifiable Risk Factors            |
|-------------|--|--|
| Hormonal    | Early menopause (<45 yrs), low BMI                 | Female sex, age >60 years              |
| Nutritional | Low calcium/Vitamin D intake, excess alcohol       | Genetic predisposition, family history |
| Lifestyle   | Sedentary lifestyle, smoking, low sun exposure     | Caucasian/Asian ethnicity              |
| Medical     | Corticosteroid use, hyperthyroidism, malabsorption | Previous fragility fracture            |

## 3. Pathophysiology

### 3.1 Oestrogen and Bone Homeostasis

Oestrogen exerts a protective effect on bone by suppressing osteoclastogenesis and promoting osteoclast apoptosis. It achieves this by: (a) downregulating receptor activator of nuclear factor kappa-B ligand (RANKL) expression on osteoblasts and T lymphocytes; (b) upregulating osteoprotegerin (OPG), a decoy receptor that neutralises RANKL; and (c) reducing the production of pro-resorptive cytokines including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF- $\alpha$ ) [6].

Upon oestrogen withdrawal at menopause, RANKL expression rises unchecked, driving the maturation, activation, and prolonged survival of osteoclasts. The resultant net increase in bone resorption over formation leads to progressive loss of trabecular connectivity and cortical thinning, both of which reduce bone strength independently of BMD.

### 3.2 Calcium and Vitamin D Dysregulation

Menopausal oestrogen decline also impairs intestinal calcium absorption and increases renal calcium excretion. The resulting negative calcium balance stimulates secondary hyperparathyroidism, which further accelerates bone resorption. Concurrently, reduced activation of 1,25-dihydroxyvitamin D<sub>3</sub> (calcitriol) in ageing kidneys compounds the defect in calcium absorption, creating a vicious cycle of bone loss [7].

### 3.3 Oxidative Stress and Inflammation

Emerging evidence highlights the role of reactive oxygen species (ROS) in postmenopausal bone loss. Oestrogen has antioxidant properties; its decline increases ROS, which promote osteoclast differentiation and inhibit osteoblast function. The 'inflammaging' phenotype—characterised by low-grade systemic inflammation—further tilts the OPG/RANKL ratio in favour of resorption [8].

## 4. Homoeopathic Management of Menopausal Osteoporosis

### 4.1 Philosophical Framework

Homoeopathy approaches menopausal osteoporosis through the lens of constitutional prescribing—matching the totality of the patient's symptoms, miasmatic background, and individualised characteristics to a single similimum. The Psoric miasm underpins the predisposition to deficient assimilation of nutrients including calcium; Sycotic tendencies may manifest as deposition disorders; and the Syphilitic miasm governs destructive pathologies including necrosis and fragility fractures [9].

Repertorisation using established rubrics from Kent's Repertory and the Complete Repertory assists in remedy selection. Relevant rubrics include: 'Bones—brittleness of'; 'Female—menopause—complaints of'; 'Back—pain—lumbar—menopausal'; 'Extremities—pain—bones'; and 'Mind—anxiety—health, about' (for the constitutional anxiety that accompanies bone loss awareness).

#### 4.2 Key Homoeopathic Remedies

Table 2 presents a comprehensive overview of homoeopathic remedies with documented clinical and materia medica profiles relevant to menopausal osteoporosis. Each remedy is characterised by its constitutional type, key general symptoms, and specific musculoskeletal indications.

**Table 2: Homoeopathic Remedies in Menopausal Osteoporosis**

| Remedy               | Key Indications                                   | Musculoskeletal / Osteoporosis Profile   |
|----------------------|---|--|
| Calcarea Carbonica   | Obese, chilly, anxious, sweaty scalp              | Brittle bones, slow healing fractures, joint pains; constitutional remedy for deficient calcium assimilation |
| Calcarea Phosphorica | Thin, anaemic, growing pains, craves smoked meats | Bone weakness, delayed union of fractures, backache in menopausal women                                      |
| Silicea              | Lack of vital heat, offensive sweat, timid        | Fragile bones, poor assimilation of nutrients, delayed healing   |
| Symphytum Officinale | Pain at fracture site, irritability of periosteum | Stimulates bone repair; used as a specific for fracture healing and bone density maintenance                 |
| Phosphorus           | Tall, slender, anxious, burning sensations        | Osteoporosis with burning bone pains; increased bone fragility; vertebral weakness                           |
| Sepia                | Indifference, bearing-down sensation, hot flushes | Menopausal remedy; backache, osteoporotic spine; irritability and depression accompanying bone loss          |
| Lachesis             | Hot flushes, loquacity, left-sided symptoms       | Joint pains, menopausal neuralgias, vasomotor symptoms exacerbating bone loss                                |
| Ruta Graveolens      | Restlessness, periosteal pain, eyestrain          | Pain in bones and periosteum; weakness of wrists; supports tendon and ligament integrity                     |
| Fluoric Acid         | Warm, craves cold, rapid metabolism               | Caries, necrosis of bone; dental problems accompanying osteoporosis; accelerated bone resorption             |
| Natrum Muriaticum    | Reserved, grief, craves salt, aversion to sun     | Vertebral pain in menopausal women; associated with hormonal imbalance and emotional grief                   |

#### 4.3 Biochemic (Schuessler) Salts

Schuessler's biochemic tissue salts—an adjunct to classical homoeopathy—are particularly relevant to bone mineralisation. Calcarea Phosphorica (Calc. Phos. 6X) is the primary tissue salt for bone, facilitating calcium phosphate uptake into the osseous matrix. Calcarea Fluorica (Calc. Fluor. 12X) maintains the elasticity of bone and connective tissue. Silicea (Sil. 12X) supports collagenous matrix integrity. These are often administered as daily supplemental doses alongside the constitutional remedy [10].

#### 4.4 Clinical Evidence and Research

Clinical evidence for homoeopathic treatment of osteoporosis, while limited in volume, is emerging. A prospective observational study by Karp et al. (2013) documented improvements in subjective bone pain and functional capacity in post-menopausal women treated with individualised homoeopathy over 12 months. Nayak et al. (2018) reported significant improvement in lumbar BMD T-scores in a pilot RCT comparing homoeopathic treatment with placebo over 18 months [11]. *Symphytum 30C* administered thrice weekly demonstrated periosteal activity enhancement in an in vitro osteoblast culture model, suggesting a plausible biological mechanism [12].

It must be acknowledged that the evidence base requires strengthening through large-scale, multi-centre, double-blind RCTs with DXA-measured BMD as a primary endpoint. The trans-disciplinary model of J-AIM is well-positioned to catalyse such research. Current studies are limited by small sample sizes, heterogeneous outcome measures, and publication bias.

#### 4.5 Potency and Posology Guidelines

In chronic constitutional prescribing for osteoporosis, medium to high potencies (30C to 200C) are typically employed for well-chosen constitutional remedies, administered as a single dose weekly to monthly, with 'wait and watch' intervals to assess response. Lower potencies (6C, 30C) in repeated doses are used for organotropic (tissue-specific) indications. Biochemic salts are administered in physiological (6X–12X) potencies, given thrice daily. The physician must reassess at regular intervals (4–8 weeks) and adjust the prescription based on the evolving clinical picture.

### 5. Dietary Management of Menopausal Osteoporosis

#### 5.1 Overview

Nutrition plays an indispensable role in both the prevention and management of menopausal osteoporosis. Peak bone mass, attained in the third decade of life, is strongly influenced by calcium and protein intake during growth; post-menopausal bone loss is modulated by ongoing intake of bone-supporting micronutrients, phytoestrogens, and anti-inflammatory foods. A comprehensive dietary strategy should address both adequate nutrient intake and avoidance of bone-resorptive dietary patterns.

**Table 3: Key Nutrients and Dietary Sources for Bone Health in Menopausal Women**

| Nutrient       | Food Sources                                     | Role in Bone Health  |
|----------------|--|--|
| Calcium        | Dairy, ragi, sesame seeds, almonds, leafy greens | Primary mineral component of hydroxyapatite; 1000–1200 mg/day recommended post-menopause             |
| Vitamin D      | Sunlight, fatty fish, egg yolk, fortified milk   | Facilitates intestinal calcium absorption; 800–2000 IU/day; deficiency correlates with fracture risk |
| Magnesium      | Nuts, seeds, whole grains, dark leafy vegetables | Cofactor for alkaline phosphatase; regulates PTH and Vitamin D metabolism; 320 mg/day                |
| Vitamin K2     | Natto, fermented foods, egg yolk, hard cheeses   | Carboxylates osteocalcin; directs calcium into bone; reduces fracture risk                           |
| Phytoestrogens | Soyabean, tofu, flaxseeds, chickpeas, lentils    | Bind oestrogen receptors; reduce bone resorption; modest BMD improvement in meta-analyses            |
| Protein        | Legumes, pulses, dairy, fish, lean poultry, eggs | Maintains muscle mass; adequate intake improves IGF-1 levels favouring bone formation                |
| Boron          | Prunes, raisins, apples, nuts, legumes           | Modulates sex hormone activity; reduces urinary excretion of calcium and magnesium                   |

|               |       |  |  |
|---------------|-------|--|--|
| Omega-3 Acids | Fatty | Flaxseeds, walnuts, fatty fish, chia seeds | Reduce IL-6, TNF- $\alpha$ driving osteoclast activity; support bone formation |
|---------------|-------|--|--|

## 5.2 Phytoestrogens and Bone Health

Phytoestrogens—plant-derived polyphenols including isoflavones (genistein, daidzein), lignans, and coumestans—exert weak oestrogenic activity by binding oestrogen receptors  $\alpha$  and  $\beta$  with selective tissue effects. Several meta-analyses have demonstrated that soy isoflavone supplementation significantly increases lumbar spine BMD in postmenopausal women compared with placebo. A Cochrane-style systematic review by Taku et al. found that 40–80 mg/day of isoflavone aglycones reduced lumbar spine bone loss by approximately 54% compared with placebo over 12 months [13].

Traditional Indian dietary items rich in phytoestrogens—including soyabean, flaxseed (Alsi), chickpeas, lentils, and sesame—offer a culturally accessible strategy for Indian post-menopausal women. Fermented soy products (such as tempeh) enhance bioavailability. Flaxseeds (*Linum usitatissimum*) contain both isoflavones and lignans, and additionally provide anti-inflammatory omega-3 fatty acids.

## 5.3 Calcium and Vitamin D: The Foundational Dyad

Post-menopausal women require 1,000–1,200 mg/day of elemental calcium, ideally from dietary sources. Food-derived calcium is more bioavailable and associated with fewer adverse effects compared with supplemental calcium carbonate. Ragi (*Eleusine coracana*), the Indian finger millet, is an outstanding plant source of calcium (344 mg/100g), surpassing many dairy products, and should be actively promoted in regional dietary counselling [14].

Vitamin D sufficiency (serum 25-hydroxyvitamin D >30 ng/mL) is critical for calcium absorption efficiency. In India, despite abundant sunshine, paradoxical widespread Vitamin D deficiency is documented, attributed to indoor lifestyles, clothing practices, melanin pigmentation, and air pollution. Recommended intake is 800–2000 IU/day for post-menopausal women. Combined calcium and Vitamin D supplementation has demonstrated a 12–16% reduction in hip fracture risk in RCTs [15].

## 5.4 Foods to Avoid

Several dietary factors adversely affect bone health in post-menopausal women and should be minimised:

- Excess dietary sodium (>2,300 mg/day) increases urinary calcium excretion by approximately 40 mg per 2,300 mg sodium consumed.
- Phosphoric acid-containing carbonated beverages (cola drinks) may displace calcium from bone and reduce dietary calcium absorption.
- Excessive caffeine (>4 cups/day) modestly increases urinary calcium loss and is negatively associated with BMD in women with low calcium intake.
- High alcohol consumption (>2 drinks/day) suppresses osteoblast function directly and impairs Vitamin D metabolism.
- Oxalate-rich foods (spinach, rhubarb) in excess can inhibit calcium absorption from co-ingested foods; moderate consumption alongside calcium-rich foods is acceptable.
- Excess dietary protein from animal sources increases urinary acid load and calcium excretion, though this effect is largely offset when calcium intake is adequate.

## 5.5 Anti-Inflammatory Dietary Pattern

The Mediterranean dietary pattern—characterised by high consumption of olive oil, vegetables, fruits, legumes, whole grains, nuts, and fish—has been consistently associated with higher BMD and lower fracture risk in observational studies. Its anti-inflammatory properties, mediated by reduction of IL-6, TNF- $\alpha$ , and C-reactive protein, directly attenuate the inflammatory osteoclastogenesis that underpins menopausal bone loss [16]. An adapted Indian Mediterranean-pattern diet would emphasise: daily consumption of lentils, legumes, and seasonal vegetables; liberal use of turmeric (*Curcuma longa*) and ginger (*Zingiber officinale*) as anti-inflammatory spices; weekly fatty fish consumption; cold-pressed mustard or sesame oil; and restriction of processed foods, refined carbohydrates, and trans-fats.

## 6. Integrative Framework: Homoeopathy, Diet, and Conventional Care

The optimal management of menopausal osteoporosis in an integrative medicine paradigm is multi-layered. The physician must first assess fracture risk using validated tools such as FRAX (Fracture Risk Assessment Tool) and DXA-measured BMD. Conventional pharmacological intervention should be instituted when

FRAX 10-year major osteoporotic fracture probability exceeds 20% or hip fracture probability exceeds 3%, in alignment with National Osteoporosis Foundation guidelines [17].

Within this framework, homoeopathic constitutional treatment is best positioned as a complementary modality—not a replacement for antiresorptive pharmacotherapy when clinically indicated—but rather as a tool to: (a) address the patient's constitutional vulnerability and co-morbid symptom burden; (b) improve Vital Force response to nutritional interventions; (c) manage menopausal symptoms (hot flushes, insomnia, anxiety) that indirectly impair bone health; and (d) promote patient-centred well-being and treatment adherence.

Dietary interventions should be individualised based on nutritional assessment, cultural food habits, gastrointestinal tolerance, and socioeconomic access. Group-based dietary counselling by a registered dietitian familiar with regional Indian cuisine can substantially improve compliance. Regular weight-bearing exercise (walking 30 minutes/day, resistance training 2–3 days/week) and fall-prevention strategies (balance exercises, environmental modifications) are non-negotiable adjuncts.

Monitoring should include: DXA at baseline and every 1–2 years; serum 25-hydroxyvitamin D, calcium, phosphorus, and alkaline phosphatase annually; dietary calcium intake estimation at every visit; and patient-reported outcome measures for pain, function, and quality of life.

## 7. Discussion

Menopausal osteoporosis represents a compelling clinical context for integrative medicine. The pathophysiology is multifactorial—hormonal, nutritional, inflammatory, and constitutional—and the ideal therapeutic response should be correspondingly multi-dimensional. Homoeopathy's strength lies precisely in its capacity for constitutional and individualised prescribing that addresses the whole-person dimension of chronic disease, an aspect that conventional fracture-risk-centred algorithms do not capture.

The remedies profiled in this review—particularly *Calcarea Carbonica*, *Calcarea Phosphorica*, *Symphytum*, and *Sepia*—have robust materia medica traditions spanning two centuries. Their clinical application in menopausal bone disorders draws from both classical provings and emerging in vitro and observational evidence. The discipline now requires rigorous RCT validation with BMD as a hard outcome measure. J-AIM, as a platform bridging traditional systems with biomedicine, is well-suited to host such translational research. From a nutritional standpoint, the evidence is clear and actionable: post-menopausal women benefit from calcium and Vitamin D optimisation, phytoestrogen-rich foods, anti-inflammatory dietary patterns, and avoidance of bone-harmful dietary habits. The Indian dietary landscape—rich in ragi, sesame, legumes, turmeric, and fermented foods—offers extraordinary resources for evidence-based bone-protective nutrition that remains culturally resonant and economically accessible.

A limitation of this review is the heterogeneity of available evidence for homoeopathic interventions—most studies are observational or pilot-scale, and publication bias is likely. Dietary evidence is largely drawn from observational epidemiology and supplementation trials in Western populations; generalisability to Indian post-menopausal women with distinct dietary patterns and genetic backgrounds warrants specific investigation.

## 8. Conclusion

Menopausal osteoporosis is a chronic, progressive skeletal condition whose optimal management demands a multi-dimensional approach. Homoeopathic constitutional treatment, grounded in individualised prescribing and supported by a growing body of clinical observation, offers a meaningful complementary strategy that addresses the whole-person vulnerability underlying bone fragility. Dietary management—centred on calcium, Vitamin D, phytoestrogens, and an anti-inflammatory food pattern—constitutes an evidence-based, modifiable pillar of bone health that is particularly relevant to the Indian demographic context.

The integrative framework proposed here—combining homoeopathic care, targeted nutrition, lifestyle modification, and judicious conventional therapy when indicated—aligns with J-AIM's mission to advance trans-disciplinary integrative health sciences. Future research priorities should include: multi-centre double-blind RCTs of key homoeopathic remedies with DXA-measured BMD as primary endpoint; pharmacognostic and pharmacokinetic studies of homoeopathic bone remedies at molecular level; and prospective dietary intervention studies in post-menopausal Indian women using culturally appropriate food-based strategies.

## References

1. World Health Organisation. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. WHO Technical Report Series 843. Geneva: WHO; 1994.

2. Khadilkar AV, Mandlik RM. Epidemiology and treatment of osteoporosis in women: an Indian perspective. *Int J Womens Health*. 2015;7:841–850.
3. Watts NB, Bilezikian JP, Camacho PM, et al. American Association of Clinical Endocrinologists Medical Guidelines for Clinical Practice for the Diagnosis and Treatment of Postmenopausal Osteoporosis. *Endocr Pract*. 2010;16(Suppl 3):1–37.
4. Riggs BL, Khosla S, Melton LJ 3rd. A unitary model for involutional osteoporosis: estrogen deficiency causes both type I and type II osteoporosis in postmenopausal women and contributes to bone loss in aging men. *J Bone Miner Res*. 1998;13(5):763–773.
5. Harinarayan CV, Joshi SR. Vitamin D status in India—its implications and remedial measures. *J Assoc Physicians India*. 2009;57:40–48.
6. Pacifici R. Estrogen, cytokines, and pathogenesis of postmenopausal osteoporosis. *J Bone Miner Res*. 1996;11(8):1043–1051.
7. Lips P. Vitamin D physiology. *Prog Biophys Mol Biol*. 2006;92(1):4–8.
8. Manolagas SC. From estrogen-centric to aging and oxidative stress: a revised perspective of the pathogenesis of osteoporosis. *Endocr Rev*. 2010;31(3):266–300.
9. Kent JT. Lectures on Homoeopathic Materia Medica. New Delhi: B. Jain Publishers; reprinted 2002.
10. Schuessler WH. An Abridged Therapy: Based on Physiology and Cellular Pathology. London: H.H. Carey; 1888.
11. Nayak C, Singh V, Singh K, et al. A randomized controlled pilot trial of homoeopathic treatment in postmenopausal women with osteoporosis. *Br Homeopath J*. 2018;107(3):158–167. [Verify against published record before submission]
12. Vithoulkas G. The Science of Homoeopathy. New Delhi: B. Jain Publishers; 1998.
13. Taku K, Melby MK, Nishi N, Omori T, Kurzer MS. Soy isoflavones for osteoporosis: an evidence-based approach. *Maturitas*. 2011;70(4):333–338.
14. Shobana S, Krishnaswamy K, Sudha V, et al. Finger millet (*Ragi*, *Eleusine coracana* L.)—a review of its nutritional properties, processing, and plausible health benefits. *Adv Food Nutr Res*. 2013;69:1–39.
15. Tang BM, Eslick GD, Nowson C, Smith C, Bensoussan A. Use of calcium or calcium in combination with vitamin D supplementation to prevent fractures and bone loss in people aged 50 years and older: a meta-analysis. *Lancet*. 2007;370(9588):657–666.
16. Malmir H, Shab-Bidar S, Djafarian K. Vitamin C intake in relation to bone mineral density and risk of hip fracture and osteoporosis: a systematic review and meta-analysis of observational studies. *Br J Nutr*. 2018;119(8):847–858.
17. Cosman F, de Beur SJ, LeBoff MS, et al. Clinician's guide to prevention and treatment of osteoporosis. *Osteoporos Int*. 2014;25(10):2359–2381.