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Can a healthy life prevent us from post-menopausal osteoporosis? Myths and truths

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ABSTRACT

Postmenopausal osteoporosis is related to unmodifiable risk factors (genetics, race, gender and age). However, other controllable factors such as activity, body mass, hormone and calcium levels and dietary habits can reduce fracture risk. For example, significant increases in bone mass density have been linked to physical activity in children, supplements of Vitamin C taken for more than 10 years, or high consumption of fish, olive oil or dried plums. This review establishes the scientific basis behind these non-pharmaceutical practices and summarizes a number of basic studies and clinical trials involving them as well as discusses their potential for the osteoporosis treatment. A lack of clinical trials in postmenopausal women to establish effectivity and doses of alternative therapies is noted. Recommendations are based on both, epidemiologic studies and data obtained from pre-clinical trials in ovariectomized rats. Collagen, Lycopene, Hesperidin and Green tea pointed to changes in bone biomarker levels in epidemiology studies. Isoflavones demonstrated to prevent bone loss in ovariectomized rats. Some daily foods showed positive effects in BMD and bone mineral content but also that food will never be as effective as drugs. However, these alternatives lack serious adverse effects and then, could be mainstays of osteoporosis chronic treatment.

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Abbreviation: ADI, acceptable daily intake; BMC, bone mineral content; BMD, bone mass density; BMI, bone mass index; BSLAP, bone specific alkaline phosphatase; FAO, food and agriculture organization of the United Nations; HST, hormone substitutive therapy; IL, interleukin; JECFA, joint FAO/WHO expert committee on food additives; NTX, N-terminal telopetide crosslinks of type I collagen; PMW, post-menopausal women; PMOW, post-menopausal osteoporotic women; PTH, parathyroid hormone; RR, relative risk; TNF-α, tumor necrosis factor; TRAP, tartrate-resistant acid phosphatase-5b; WHO, World Health Organization.

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1. Introduction

Despite the incidence of osteoporotic fractures stabilizing during the last years, hip and vertebral fractures are still associated with a mortality risk, mainly amongst the elderly [1]. In fact, according to estimates, approximately 40% of women and 13% of men older than 50 years will have an osteoporotic related fracture in their lifetime. With these percentages, osteoporosis is the leading cause of morbidity and mortality in the elderly [2].

Epidemiological studies showed that if we achieve a 10% increase in the peak bone mass (the maximum bone mass accrual achieved at the end of growth) then we are able to reduce post-menopausal osteoporotic fracture risk by 50% [3–5]

Low Bone Mass Density (BMD) means higher risk of vertebral fractures, 1 T-Score standard deviation reduction reflects in a 2 per cent increase in risk fracture [6].

However, some post-menopausal, osteoporotic fracture risk factors cannot be modified such as,

- Age, each decade the risk of fractures due to post-menopausal osteoporosis is increased in 1,4–1,8 [7] (Table 1)
- Race, White and Oriental race have a higher risk of female osteoporotic fractures than in Black or Polynesian [8]
- Family history, it has been demonstrated that women who had a mother with femur fractures has more probability to sustain a fracture related to osteoporosis. In fact, between 60% and 80% of the variance in the peak bone mass can be explained by genetic factors [3]
- Estrogens, despite being able to intercede in the estrogen serum levels taking drugs to equilibrate hormone levels during the menopause, it is demonstrated that early menopause and late menarquia increase fracture risk factors, and we cannot modify both factors in time [7]

Nevertheless, there are a large number of risk factors in fracture development associated to post-menopausal osteoporosis that can be affected by our habits. Some of these are tobacco use and alcohol intake [7], weight [9], sedentary lifestyle [10], vitamin D and calcium deficiency and a hyper-protein diet [11]. In addition, some studies showed benefits in the daily consumption of vegetables [12,13], olive oil [14], orange juice [15], fruits [16], collagen [17], soy [18] and tea [19].

Despite this, there is an important lack of clinical trials that demonstrate the efficacy in humans. Data obtained from pre-clinical trials and from epidemiology studies have been interpreted to recommend ways for reducing fracture risk in PMW.

In this review, pre-clinical studies, clinical trials and epidemiological studies performed with the most popular alternative therapies used for the treatment of post-menopausal osteoporosis in women have been analyzed, trying to find out if these have efficacy or not in the prevention or treatment of the illness.

Unfortunately, post-menopausal osteoporosis (PMO) illness development is closely related to genetics, race, gender and age. All of these are factors that cannot be modified, but some studies demonstrated that activity, body mass, hormone and calcium levels and dietary habits are modifiable factors that can reduce fracture risk factors.

The objective of this review is to find out the results regarding fracture risk reductions in post-menopausal women (PMW) when dietary and/or activity were modified, and we tried to conclude whether these changes really have a significant impact or not.

2. Material and methods

We reviewed information already published in Scientific Publications such as the British Journal of Clinical Pharmacology, Nutrients, the National Institutes of Health, the Journal of Nutrition, The Ontario Health Technology Assessment Series, Preventive Medicine and Nutrition, Metabolism and Cardiovascular Disease, both from Elsevier, The Spanish Public Health Journal, etc.

This information has been searched using PubMed with keywords such as Post-menopausal Osteoporosis; Bone mass density; Clinical Trials; Dietary; Activity.

3. Results

3.1. Physical activity

When a subject is immobilized, the stimulus for bone acquisition is insufficient and resorption gained to bone formation. This happens because osteocytes, as bone pressure receptors do not detect gravity properly. The same problem affects thin or lean people. For this reason, it is very important to practice regular exercise, better hitting the ground. In fact, three controlled trials done in pre- and peri-pubertal American children showed that jumping several minutes, three times a week stimulated bone formation. McKey demonstrated that trochanteric BMD increased in the interventional group vs the control group when jumps were tracked 3 times a week for 8 months in 144 children from 6 to 10 years old [20]. Mackelrie measured BMD in 177 children with ages between 8 and 11 years old, who jumped 10 min 3 times a week for 7 months. In these, femoral neck and spine BMD increased in the interventional group vs the control group in early puberty, but no effects were observed in pre-pubertal girls [20,21]. Fuchs evaluated BMD in 89 children (5–9 years old) who jumped 100 times 3 times a week for 7 months. Results showed that spine BMD increased more in the interventional group vs the control group [22]. Another 6-year longitudinal study [23] demonstrated that the most active teenagers have more bone mineral content (BMC) in the spine, femoral neck and whole body compared to those who are inactive (Table 2).

However, after menopause, there is no evidence that physical activity prevents bone loss or increases BMD. Possible reasons are (i)

Table 1
Age-related osteoporosis in Spanish women [7].

Women-Years old	Lumbar Spine Osteoporosis (%)	Femoral neck Osteoporosis (%)
45–49	4.31	0.0
50–59	9.09	1.3
60–69	24.29	5.71
70–79	40.0	24.24