

Nutritional Strategies in the Prevention of Osteoporosis

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ABSTRACT: *Osteoporosis is a gradual reduction in bone mass that leads to an increased susceptibility to bone fractures. An estimated 1.5 million fractures are attributed to osteoporosis each year in the United States. With the rapid aging of the population, the suffering and healthcare costs associated with osteoporosis are steadily increasing. Experts agree that prevention is the most effective method of dealing with osteoporosis. The two approaches to prevention are maximizing peak bone mass at skeletal maturity and reducing the rate of age-related bone loss. Adequate calcium intake has been shown to have a strong influence on attaining peak bone mass and reducing the*

rate of bone loss. Unfortunately, studies have shown that a large proportion of the population does not even meet the current Reference Daily Intake (RDI) levels for calcium. The data for women is especially alarming: at age 60, approximately 80% of women consume less than the RDI for calcium. Supplementing the diet with calcium and other nutrients important for bone health, along with regular exercise and a healthy lifestyle, is essential in reducing the risk of osteoporosis. Microcrystalline hydroxyapatite concentrate (MCHC) is an excellent source of bioavailable calcium, a full complement of other minerals, proteins, and organic factors.

Osteoporosis is a systemic skeletal disease characterized by reduced bone mass, compromised bone strength, and increased susceptibility to bone fractures.^{1,2} In the U.S. today, approximately 10 million people have osteoporosis; another 18 million have osteopenia (low bone mass) and are at risk for developing osteoporosis. According to the National Institutes of Health (NIH), osteoporosis accounts for an estimated 1.5 million fractures annually in the U.S.³ In fact, it is estimated that 1 in 2 women and 1 in 8 men will suffer an osteoporosis-related fracture in their lifetime. The estimated annual expenditures for treating osteoporotic fractures are between 10 and 15 billion dollars, based on hospitalization costs and acute and long-term care.^{3,4}

Osteoporosis is the result of a gradual loss of bone tissue that is generally a symptomless process. Therefore, without proper risk assessment, osteoporosis may go undetected until bones become so brittle that even the slightest trauma causes a fracture.¹ Although a fracture can occur anywhere in the body, the most common region is the spine. One-third of women 65 years and older have sustained a vertebral fracture, which leads to loss of height, kyphosis ("dowager's hump"), and chronic back pain.^{1,5,6}

The good news is that much of this suffering may be preventable. The agreement among qualified experts is that the severity of age-related bone loss may be reduced in patients at risk if adequate amounts of nutrients needed for bone health are obtained throughout their lifetime. In fact, the Food and Drug Administration concludes, "Maintenance of an adequate calcium intake throughout life may optimize peak bone mass at skeletal maturity and help to slow the rate of bone loss later in life, and may help to reduce the risk of osteoporosis."⁴

WHAT ARE THE RISK FACTORS?

Two major factors that influence the risk of developing osteoporosis are the level of bone mass achieved at skeletal maturity (peak bone mass) and the rate at which bone loss occurs in later years. The more bone mass available before age-related bone loss ensues, the less likely it will decrease to a level at which fractures occur.⁷

Research studies point to a number of risk factors that may have a strong influence on peak bone mass and the rate of bone loss, and thus the development of osteoporosis (Table 1).^{1,5,7,8} Some of these factors include: inadequate nutritional intake, lack of physical activity, smoking, excessive alcohol or caffeine consumption, and prolonged use of medications such as corticosteroids or antacids.

In addition to diet and lifestyle factors, genetic and ethnic factors significantly influence many aspects of calcium and skeletal metabolism.^{1,5,7-9} Caucasian and Asian women tend to have lower bone density than African and Hispanic women and, consequently, are more likely to suffer from osteoporotic fractures. The same holds true for thin, smaller-boned women.

What does appear certain is that regular exercise, lifetime maintenance of adequate nutritional intake with regard to calcium and other nutrients important for bone health, and a healthy lifestyle are essential for maximizing peak bone mass and for minimizing the rate of bone loss that occurs with aging, and thus reducing the risk of osteoporosis.^{7,8,10}

Table 1. Major Risk Factors for Osteoporosis in Women

- Family history of osteoporosis
- White or Asian
- Small body frame
- Postmenopausal
- Hysterectomy
- Inadequate calcium, vitamin D, and other nutrient intake
- Excess protein in the diet
- Inadequate exercise
- Smoking
- Excessive alcohol consumption
- High intake of caffeine, carbonated beverages, and salt
- Long-term glucocorticoid therapy
- Long-term use of anticonvulsants, antacids
- Hyperparathyroidism, thyrotoxicosis, Cushing's syndrome, type 1 diabetes

• Bone Density Evaluation

The risk of sustaining an osteoporotic fracture is determined by an analysis of bone mineral density (BMD), which refers to the grams of mineral per area or volume in bone. BMD is determined by diagnostic screenings, such as x-ray or ultrasound. The World Health Organization has determined that the diagnostic criteria for osteoporosis is 2.5 standard deviations below the mean, while a score between 1.0 and 2.5 below the mean determines osteopenia.¹¹ This statistical analysis refers to the BMD of Caucasian women only; therefore, controversy exists regarding how to apply this measurement to children, men, and across ethnic groups.

OSTEOPOROSIS: PREVENTION IS THE KEY

Experts agree that prevention is likely to remain the most effective method of dealing with osteoporosis.¹⁵ The two approaches to prevention are maximizing peak bone mass at skeletal maturity and reducing the rate of age-related bone loss:^{9,12}

• Maximizing Peak Bone Mass

Bone size and strength is continually acquired during the first three decades of life, typically peaking between the ages of 30-35. Studies have shown that an optimal intake of calcium during childhood and up to age 25-30 can positively impact an individual's peak bone mass.^{7,13-17} Children and young adults who do not consume adequate amounts of calcium may have suboptimal bone density by the time they reach age 35. It is becoming increasingly clear that insufficient accumulation of skeletal mass by young adulthood predisposes a person to fractures later in life as age-related bone loss ensues.⁴

According to a study performed on identical twins at the Indiana State University School of Medicine, calcium supplementation of 1,000 mg/day significantly enhanced the rate of increase in BMD in prepubertal children. "If the gain persists, it would probably result in an increase in peak bone mass that would reduce the risk of osteoporotic fractures later in life," stated the researchers.¹⁵ Another study done at the Pennsylvania State University College of Medicine showed that calcium supplementation of 500 mg/day for a group of 12-year old girls resulted in

significant gains in total body and spinal bone density when compared to the control group.¹⁶

Bone continuously remodels through the processes of bone resorption and bone formation, which are dependent upon adequate levels of parathyroid hormone (PTH) and calcitonin. This remodeling process functions by the interaction of two types of cells: osteoclasts, which resorb bone, and osteoblasts, which form new bone. Bone remodeling, or bone turnover, is usually in balance until the fourth decade of life when resorption becomes slightly greater than formation and a small, continuous loss of bone mass results.

• Reducing Age-Related Bone Loss

Bone loss occurs with age in both sexes; however, the pattern differs significantly between men and women.¹⁸ In women, bone loss usually begins prior to menopause (35-45 years), with a pronounced acceleration of bone loss occurring rapidly for about 5-10 years following menopause.^{2,8,18} Over their lifetimes, women lose about 35% of their cortical bone mass and 50% of their trabecular bone mass, whereas men lose about two-thirds of these amounts.^{2,5} Cortical bone predominates in the shafts of long bones, while trabecular bone is concentrated in the vertebrae, the pelvis and other flat bones, and in the ends of long bones.

Calcium supplementation has also been shown to reduce the rate of bone loss in postmenopausal women. A study reported in the *New England Journal of Medicine* by Reid et al. demonstrated a 43% reduction in bone loss in postmenopausal women who supplemented their regular diets with 1,000 mg of calcium for two years, compared to postmenopausal women receiving placebo.¹⁹ Their results confirmed an earlier, two-year calcium supplementation study, also reported in the *New England Journal of Medicine*, which indicated that healthy postmenopausal women can significantly reduce bone loss by increasing their calcium intake to at least 800 mg/day.²⁰

CALCIUM—WHAT ARE THE RECOMMENDED LEVELS?

Established to replace the Recommended Daily Allowance (RDA), the Reference Daily Intake (RDI) represents a guideline for daily nutrient intake that is considered to adequately meet the needs of most healthy individuals in the U.S. The RDI levels are based on the latest average RDA of the total population over 4 years of age. The RDI does not provide recommendations for any specific age group or gender.

The current RDI level for calcium is 1,000 mg—well below the level of intake that many experts recommend.^{9,21-25} The authors of a review of intervention trials of calcium supplementation suggested that the recommended daily intake for children should be 1,450 mg/day during adolescence, while others have recommended a calcium intake of up to 1,800 mg/day.^{9,22} Such an increase in calcium intake during adolescence could play an important role in maximizing peak bone mass. Regarding the calcium intake for older individuals, many experts recommend an intake of 1,500 to 2,000 mg/day to minimize bone loss.^{21,23,24}

The NIH Consensus Conference on Optimal Calcium Intake recommends calcium intakes of 1,200 to 1,500 mg/day for 11-24 year olds, 1,000 mg for those 25-50 years, and 1,500 mg for those over 65. In addition, the NIH recommends a calcium intake of 1,500 mg/day for women over 50 years who are not receiving hormone replacement therapy.²⁶

While the RDI levels of calcium may be a source of debate, the real issue is the fact that a large proportion of the population isn't even meeting these suggested daily intakes.^{5,8,27} According to data obtained from the USDA's 1994-96 Continuing Survey of Food Intakes for Individuals, an estimated 65% of the U.S. population consumed less than the RDI for calcium.²⁸ The data for women is especially alarming: at age 60, approximately 80% of women did not meet the suggested daily requirements. In addition, between the ages of 12-29, when calcium requirements reach their peak because of rapid skeletal growth, as many as 85% of females did not even meet the RDI for calcium. Therefore, the challenge for healthcare professionals is to educate patients on the importance of lifetime maintenance of adequate calcium intake.

CALCIUM ABSORPTION

Intestinal absorption of calcium declines with age in both sexes. In addition, the body is less able to adapt to an insufficient calcium supply.^{1,2,5,8} Consequently, there is a need to address factors that affect calcium absorption, particularly when dealing with the elderly population. For instance, large quantities of dietary fiber can interfere with calcium absorption, as can diuretics, alcohol, caffeinated drinks, certain medications such as corticosteroids and antacids, and vitamin D deficiency. Hypochlorhydria, a condition of low gastric acid production, can also hinder calcium absorption and is quite common in the elderly. Patients encountering any of these factors may need closer nutritional attention.

VITAMIN D

Vitamin D plays an essential role in maintaining a healthy mineralized skeleton.²⁹ The main physiologic function of vitamin D is to maintain serum calcium and phosphorus concentrations within the normal range in order to maintain essential cellular functions and promote mineralization of the skeleton.²⁹⁻³² Vitamin D acts primarily to increase serum calcium by stimulating its intestinal absorption.

Vitamin D insufficiency results in reduced calcium absorption, a rise in circulating PTH, and increased bone resorption.^{28,31,33} Vitamin D insufficiency is frequent among the elderly population, owing to less efficient skin synthesis of vitamin D, less efficient intestinal absorption, reduced sun exposure, and a reduced vitamin D intake.²⁹⁻³¹ A number of studies demonstrate that supplementation with 400-800 IU/day of vitamin D effectively reverses vitamin D deficiency in the elderly.^{28,32,34,35}

Vitamin D deficiency can result in secondary hyperparathyroidism, a condition that accelerates bone resorption and exacerbates osteoporosis.^{29,30,36} Vitamin D deficiency may also be associated with an increased risk of hip fracture.²⁹ Several studies have demonstrated that an increase in calcium intake of 800-1000 mg/day in combination with vitamin D supplementation of 400-800 IU/day will decrease the risk of vertebral and nonvertebral fractures and increase BMD.^{29,32,36,37}

MAGNESIUM

Although decreased bone mass is the hallmark of osteoporosis, qualitative changes in bone matrix are also present, which could

result in fragile or brittle bones that are more susceptible to fracture. There is growing evidence that magnesium may be an important factor in the qualitative changes of the bone matrix that determine bone fragility.^{38,39} Magnesium influences both matrix and mineral metabolism in bone by a combination of effects on hormones and other factors that regulate skeletal and mineral metabolism, and by direct effects on bone itself. Magnesium depletion adversely affects all stages of skeletal metabolism, causing cessation of bone growth, decreased osteoblastic and osteoclastic activity, osteopenia, and bone fragility.^{38,39}

Magnesium plays important roles in calcium metabolism through its involvement in normal activity of the hormones (i.e., PTH, calcitonin) that control calcium utilization.⁴⁰ Adequate serum magnesium levels are necessary for proper calcium metabolism, and hypomagnesemia can result in hypocalcemia and peripheral resistance to the effects of vitamin D. Thus, adequate calcium intake may not ensure proper bone health if magnesium status is abnormal.^{38,40} Because of the effect of magnesium deficiency on calcium metabolism, it may also be implicated as a risk factor for osteoporosis.^{41,42}

Large numbers of individuals may be at risk for magnesium deficiency. Dietary intake studies consistently show intakes of magnesium to be below the RDI in many age groups, and surveys have shown that 39% of American women between 15 and 50 years of age receive less than 70% of the RDI.^{38,40,43} Because high calcium intake intensifies magnesium deficiency, patients who take calcium supplements to an extent that the calcium to magnesium ratio substantially exceeds 2:1 are likely to have relative or absolute magnesium deficiency.⁴³ Calcium supplementation without magnesium may reduce the efficiency of magnesium absorption from the diet and further aggravate the consequences of diminished estrogen, resulting in less movement of magnesium into bone and greater activity of demineralizing PTH.⁴⁴

TRACE MINERALS

Trace minerals, particularly zinc, copper, manganese, fluoride, boron, and silicon, are being studied for their roles in bone health. Zinc is needed for osteoblastic activity, collagen synthesis, and alkaline phosphatase activity; copper for cross-linking collagen and elastin; manganese for the biosynthesis of mucopolysaccharides in bone matrix formation; fluoride for osteoblast activity; and boron and silicon for healthy bone formation.⁴⁵

Studies have shown that trace mineral deficiencies can impair bone formation and resorption. For instance, in a 2-year clinical study, postmenopausal women who received calcium supplements together with zinc, copper, and manganese experienced a gain in BMD, while women receiving calcium alone, trace minerals alone, or a placebo experienced increasingly greater losses in BMD.⁴⁶

IPRIFLAVONE

Ipriflavone (7-isopropoxyisoflavone) is a derivative of naturally-occurring isoflavones and is active in bone metabolism.⁴⁷⁻⁴⁹ Although ipriflavone is similar in structure to phytoestrogens, it has been shown to be devoid of any estrogenic activity.^{48,50}

Numerous double-blind, placebo-controlled studies have shown a positive effect of ipriflavone in reducing bone mineral loss and increasing bone density in postmenopausal women with osteopenia or established osteoporosis at a dose of 600 mg/day.^{47,48,51-54} In these studies, all patients received an oral calcium supplement of 1g/day in addition to ipriflavone or placebo. One of the researchers, Dr. Donato Agnusdei, stated that “Long-term treatment with ipriflavone may be considered safe, and may increase bone density and possibly prevent fractures in elderly patients with established osteoporosis.”⁵³ Another study evaluating the effects of ipriflavone combined with vitamin D showed that the combined therapy was more effective in reducing bone loss than either therapy alone or control.⁴⁹

In vivo and in vitro studies in different experimental models have shown the inhibitory effect of ipriflavone on osteoclast recruitment and activity (bone resorption).⁴⁷ Studies in humans have confirmed the inhibitory effect of ipriflavone on bone resorption in conditions of high bone turnover, such as Paget’s disease of bone.⁴⁷ These results were also demonstrated in a 1-year study comparing the effects of treatment with either 600 mg/day ipriflavone or 800 mg calcium lactate in postmenopausal women with high bone turnover and low bone mass.⁵⁵ Lumbar BMD and bone metabolic markers were assessed prior to and upon completion of the study. Results showed that ipriflavone treatment significantly suppressed the expected loss of lumbar BMD compared to calcium lactate. Evaluation of biochemical markers in bone metabolism suggested that ipriflavone acts to suppress the bone resorption system.

COMPREHENSIVE BONE NOURISHMENT: MCHC

Microcrystalline hydroxyapatite concentrate (MCHC) is a whole bone extract, complete with all the minerals and organic factors in the same physiological proportions naturally found in healthy bone. It is an excellent source of bioavailable calcium and other nutrients essential to bone formation, and contains phosphorus, magnesium, fluoride, zinc, silicon, manganese, and other trace minerals.⁵⁶⁻⁶⁰

MCHC also contains the proteins found in bone, including growth factors.⁶¹ Recently, scientists have focused attention on the actions of such factors as modulators of bone remodeling, including insulin-like growth factors I and II (IGF-I and IGF-II) and transforming growth factor-beta (TGF-β). These growth factors have been shown to stimulate the proliferation and activity of osteoblasts, as well as inhibit or modulate precursors to osteoclasts in vitro.⁶²⁻⁶⁵ As a result, bone formation may be enhanced and bone mass may increase.⁶⁶ Researchers theorize that the presence of these growth factors may be one reason why MCHC is so effective.

A study published in *Osteoporosis International* evaluated the effectiveness of two forms of calcium supplements—MCHC and calcium carbonate—in preventing further bone loss in postmenopausal osteoporosis.⁵⁹ In this study, patients treated with calcium carbonate demonstrated a reduction in the rate of bone loss by about half, while treatment with MCHC was shown to nearly halt it. Another study designed to compare the efficacy of MCHC and calcium carbonate in postmenopausal women who refused hormone replacement therapy showed that continuous administration of MCHC over a period of 2 years significantly reduced bone loss; conversely, women given calcium carbonate

or placebo had significant decreases in bone mass.⁶⁷ In addition, several clinical trials have shown that MCHC significantly slows bone loss in corticosteroid-treated patients normally prone to rapid bone loss.^{58,68,69}

Not only has MCHC been shown to be effective in minimizing bone loss, but it has also been shown to positively affect bone density. A study performed on osteoporotic postmenopausal women with the complication of primary biliary cirrhosis, a condition involving severe calcium malabsorption, showed that MCHC supplementation helped to increase cortical bone thickness by 6.1%.⁵⁷ Conversely, calcium gluconate halted the bone loss but did not restore bone, and the group receiving no supplementation continued to show accelerated bone loss.

In another study, the effectiveness of MCHC on the healing of a standardized bone defect in rabbits was evaluated as compared with a control group and two other forms of calcium supplements: bone mineral (MCHC without the organic factors) and calcium carbonate.⁷⁰ The results showed that treatment with MCHC, but not the other two calcium supplements, resulted in significant improvements in the pattern and quality of bone healing. These results indicate that MCHC has a beneficial effect on the process of bone healing; however, the effect is lost if the organic components of the compound are destroyed, or if calcium carbonate treatment is substituted.

GUIDELINES FOR DETERMINING THE PURITY OF MCHC

There is great variation in the quality and purity of MCHC products. The source of the bone extract as well as the processing procedures are of utmost importance in determining the quality of MCHC. Some sources of MCHC may contain high levels of lead and other contaminants, or contain cartilage and tendons. Certain processing procedures, such as high-heat and excessive grinding, can result in a product that is nothing more than bone meal. These products lack the full complement of minerals, organic factors, and the microcrystalline structures that are characteristic of true MCHC.⁷¹

How can you be sure that what you are providing to your patients is pure, authentic MCHC? The following guidelines for determining the identity and purity of MCHC products may prove helpful to the clinician wanting to provide an exceptional product to his or her patients:

1. The results of a Certificate of Analysis should be requested from the supplier. This analysis will indicate the protein and mineral content of the MCHC, as well as any microbial contamination. Analysis of authentic MCHC will show the same constituent ratios typically found in bone: 22%-28% protein (mostly collagen), 22%-28% elemental calcium, and 9%-13% phosphorus, with the remainder comprised of fat and other minerals.
2. The results of a collagen analysis will indicate the purity and non-adulteration of MCHC. Type I collagen is the predominant type of collagen found in bone, along with small amounts of Type V collagen. Properly processed MCHC should contain approximately 20% collagen, the majority of which should be Type I. Poorly processed bone, such as bone meal, yields a product with only 0% to 7% Type I collagen. The presence of other types of collagen would indicate that the raw material used to make the MCHC contained cartilage, tendons, muscle, marrow, or ligaments.

3. The results of an x-ray diffraction analysis should be requested. This analysis will confirm the microcrystalline structure of the MCHC.
4. A certificate proving the material is approved for human consumption is very important. MCHC may be available in different grades of varying quality. A reliably pure form of MCHC is imported from New Zealand where cattle are free-range fed and raised in a pesticide-free environment. A Sanitary Certificate of Origin and Health from the Ministry of Agriculture accompanies MCHC imported from New Zealand. This certificate is required for import into the U.S. and provides assurance of high-grade MCHC.
5. The results of a heavy metal analysis conducted by a third party laboratory should be requested. Heavy metal contamination with lead, arsenic, aluminum, mercury, strontium, and other metals is a concern with regard to some forms of calcium supplements and MCHC is no exception. In high quality MCHC, lead should not be present at levels higher than 1 mcg per gram of product.

REFERENCES

1. Dempster DW, Lindsay R. Pathogenesis of osteoporosis. *Lancet* 1993;341:797-801.
2. Weaver CM. Calcium bioavailability and its relation to osteoporosis. *Soc Exp Biol Med* 1992;200:157-60.
3. NIH Consensus Conference. Osteoporosis Prevention, Diagnosis, and Therapy. *JAMA* 2001;285:785-95.
4. Food Labeling: Health Claims; Calcium and Osteoporosis. Guide to US Food Labeling Law, Appendix III 1991;789-820.
5. Riggs BL, Melton LJ. Involutional osteoporosis. *N Engl J Med* 1986;314: 1676-86.
6. Strause L, Saltman P, Smith KT, et al. Spinal bone loss in postmenopausal women supplemented with calcium and trace minerals. *J Nutr* 1994;124:1060-64.
7. Arnaud CD, Sanchez SD. The role of calcium in osteoporosis. *Annu Rev Nutr* 1990;10:397-414.
8. Notelovitz M. Osteoporosis: screening, prevention, and management. *Fertil Steril* 1993;59:707-25.
9. Matkovic V, Fontana D, Tominac C, et al. Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. *Am J Clin Nutr* 1990;52:878-88.
10. Lindsay R. Prevention and treatment of osteoporosis. *Lancet* 1993;341:801-05.
11. WHO Study Group. *Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: Report of a WHO Study Group*. WHO Technical Report Series 843, Geneva: World Health Organization, 1994: 1-129.
12. Nguyen TV, Kelly PJ, Sambrook PN, et al. Lifestyle factors and bone density in the elderly: implications for osteoporosis prevention. *J Bone Miner Res* 1994;9:1339-46.
13. Chan G, Hess M, Hollis J, et al. Bone mineral status in childhood accidental fractures. *Am J Dis Child* 1984;138:569-70.
14. Chan G. The effect of dietary calcium supplementation on pubertal girls' growth and bone mineral status. *Clin Res* 1992;40:60A.
15. Johnston C, Miller JZ, Slemenda CW, et al. Calcium supplementation and increases in bone mineral density in children. *N Engl J Med* 1992;327:82-87.
16. Lloyd T, Andon MB, Rollings N, et al. Calcium supplementation and bone mineral density in adolescent girls. *JAMA* 1993;270:841-44.
17. Recker RR. Bone gain in young adult women. *JAMA* 1992;268:2403-08.
18. Dawson-Hughes B, Krall EA, Harris S, et al. Risk factors for bone loss in healthy postmenopausal women. *Osteo Int* 1993;1:S27-31.
19. Reid IR, Ames RW, Evans MC, et al. Effect of calcium supplementation on bone loss in postmenopausal women. *N Engl J Med* 1993;328:460-64.
20. Dawson-Hughes B, Dallal GE, Krall EA, et al. A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. *N Engl J Med* 1990;323:878-83.
21. Heaney RP. Thinking straight about calcium. *N Engl J Med* 1993; 328:503-05.
22. Andon MB, Lloyd T, Matkovic V. Supplementation trials with calcium citrate malate: evidence in favor of increasing the calcium RDA during childhood and adolescence. *J Nutr* 1994;124(8 Suppl):1412S-17S.
23. Prince R. The calcium controversy revisited: implications of new data. *Med J Aust* 1993;159:404-07.
24. Smith EL, Gilligan C, Smith PE, et al. Calcium supplementation and bone loss in middle-aged women. *Am J Clin Nutr* 1989;50:833-42.
25. Weaver CM. Age related calcium requirements due to changes in absorption and utilization. *J Nutr* 1994;124(8 Suppl):1418S-25S.
26. NIH Consensus Conference. Optimal calcium intake. *JAMA* 1994;272:1942-48.
27. Fleming KH, Heimbach JT. Consumption of calcium in the U.S.: food sources and intake levels. *J Nutr* 1994;124(8 Suppl):1426S-30S.
28. U.S. Department of Agriculture, Agricultural Research Service. Data tables: *USDA's 1994-96 Continuing Survey of Food Intakes by Individuals*. Retrieved May 10, 2002, from <http://www.barc.usda.gov/bhrc/foodsurvey/home.htm>
29. Holick MF. Vitamin D and bone health. *J Nutr* 1996;126(4 Suppl):1159S-64S.
30. Fraser DR. Vitamin D. *Lancet* 1995;345:104-07.
31. Dawson-Hughes B. Calcium and vitamin D nutritional needs of elderly women. *J Nutr* 1996;126(4 Suppl):1165S-67S.
32. Ooms ME, Roos JC, Bezemer PD, et al. Prevention of bone loss by vitamin D supplementation in elderly women: a randomized double-blind trial. *J Clin Endocrinol Metab* 1995;80:1052-58.
33. Mezquita-Raya P, Munoz-Torres M, Luna JD, et al. Relation between vitamin D insufficiency, bone density, and bone metabolism in healthy postmenopausal women. *Bone Miner Res* 2001;16(8):1408-15.
34. Gennari C. Calcium and vitamin D nutrition and bone disease of the elderly. *Public Health Nutr* 2001;4(2B):547-49.
35. Nordin BE, Baker MR, Horsman A, et al. A prospective trial of the effect of vitamin D supplementation on metacarpal bone loss in elderly women. *Am J Clin Nutr* 1985;42(3):470-74.
36. Compston JE. The role of vitamin D and calcium supplementation in the prevention of osteoporotic fractures in the elderly. *Clin Endocrinol* 1995;43:393-405.
37. Dawson-Hughes B, Harris SS, Krall EA, et al. Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age or older. *N Engl J Med* 1997;337:670-76.
38. Sojka JE, Weaver CM. Brief critical reviews: magnesium supplementation and osteoporosis. *Nutr Rev* 1995;53:71-74.
39. Wallach S. Effects of magnesium on skeletal metabolism. *J Am Coll Nutr* 1989;8:457A.
40. Abraham GE. The importance of magnesium in the management of primary postmenopausal osteoporosis. *J Nutr Med* 1991;2(2):1-25.
41. Abbott LG, Rude RK. Clinical manifestations of magnesium deficiency. *Miner Electrolyte Metab* 1993;19:314-22.
42. Fatemi S, Ryzen E, Flores J, et al. Effect of experimental human magnesium depletion on parathyroid hormone secretion and 1,25-dihydroxyvitamin D metabolism. *J Clin Endocrinol Metab* 1991;73:1067-72.
43. Seelig MS. Prophylactic treatment of osteoporosis with estrogen and calcium increases need for magnesium. *J Am Coll Nutr* 1989;8:457A.
44. Dreosti IE. Magnesium status and health. *Nutr Rev* 1995;53(9 Pt 2):S23-S27.
45. Mahan KL, Escott-Stump S. *Krause's Food, Nutrition, and Diet Therapy*. 9th ed. Philadelphia: W.B. Saunders; 1996.
46. Saltman PD, Strause LG. The role of trace minerals in osteoporosis. *J Amer Coll Nutr* 1993;12:384-89.
47. Adami S, Bufalino L, Cervetti R, et al. Ipriflavone prevents radial bone loss in postmenopausal women with low bone mass over 2 years. *Osteoporosis Int* 1997;7:119-25.
48. Valente M, Bufalino L, Castiglione GN, et al. Effects of 1-year treatment with ipriflavone on bone in postmenopausal women with low bone mass. *Calcif Tissue Int* 1994;54:377-80.
49. Ushiroyama T, Okamura S, Ikeda A, et al. Efficacy of ipriflavone and 1-alpha vitamin D therapy for the cessation of vertebral bone loss. *Int J Gynecol Obstet* 1995;48:283-88.
50. Melis GB, Paoletti AM, Cagnacci A, et al. Lack of any estrogenic effect of ipriflavone in postmenopausal women. *J Endocrinol Invest* 1992;15:755-61.
51. Agnusdei D, Adami S, Cervetti R, et al. Effects of ipriflavone on bone mass and calcium metabolism in postmenopausal osteoporosis. *Bone Miner* 1992;19(1 Suppl):S43-S48.
52. Agnusdei D, Zacchei F, Bigazzi S, et al. Metabolic and clinical effects of ipriflavone in established postmenopausal osteoporosis. *Drugs Exp Clin Res* 1989;2:97-104.
53. Agnusdei D, Bufalino L. Efficacy of ipriflavone in established osteoporosis and long-term safety. *Calcif Tissue Int* 1997;61(1 Suppl):S23-S27.
54. Kovacs AB. Efficacy of ipriflavone in the prevention and treatment of postmenopausal osteoporosis. *Agents Actions* 1994;41:86-87.
55. Ohta H, Komukai S, Makita K, et al. Effects of 1-year ipriflavone treatment on lumbar bone mineral density and bone metabolic markers in postmenopausal women with low bone mass. *Horm Res* 1999;51(4):178-83.
56. Windsor AC, Misra DP, Loudon JM, et al. The effect of whole-bone extract on 47Ca absorption in the elderly. *Age & Ageing* 1973;2:230-34.
57. Epstein O, Kato Y, Dick R, et al. Vitamin D, hydroxyapatite, and calcium gluconate in treatment of cortical bone thinning in postmenopausal women with primary biliary cirrhosis. *Am J Clin Nutr* 1982;36:426-30.
58. Pines A, Raafat H, Lynn AH, et al. Clinical trial of MCHC ('Ossopan') in the prevention of osteoporosis due to corticosteroid therapy. *Curr Med Res Opin* 1984;8:734-42.
59. Rueggeger P, Keller A, Dambacher MA. Comparison of the treatment effects of ossein-hydroxyapatite compound and calcium carbonate in osteoporotic females. *Osteo Int* 1995;5:30-4.
60. Stepan JJ, Pospischal J, Presl J, et al. Prospective trial of ossein-hydroxyapatite compound in surgically induced postmenopausal women. *Bone* 1989;10:179-85.
61. Stepan JJ, Mohan S, Jennings JC, et al. Quantitation of growth factors in ossein-mineral-compound. *Life Sci* 1991;49(13):PL79-84.
62. Marie P. Growth factors and bone formation in osteoporosis: roles for IGF-I and TGF-beta. *Rev Rhum Engl Ed* 1997;64(1):44-53.
63. Baylink DJ, Finkelman RD, Mohan S. Growth factors to stimulate bone formation. *J Bone Miner Res* 1993;8(2 Suppl):S565-72.
64. Linkhart TA, Mohan S, Baylink D. Growth factors for bone growth and repair: IGF, TGFβ and BMP. *Bone* 1996;19(1 Suppl):1S-12S.
65. Bonewald LF, Mundy GR. Role of transforming growth factor-beta in bone remodeling. *Clin Orthop* 1990;250:261-76.
66. Castelo-Branco C, Pons F, Vicente JJ, et al. Preventing postmenopausal bone loss with ossein-hydroxyapatite compounds. Results of a two-year, prospective trial. *J Reprod Med* 1999;44(7):601-05.
67. Pfeilschifter J, Oechsner M, Naumann A, et al. Stimulation of bone matrix apposition in vitro by local growth factors: a comparison between insulin-like growth factor-I, platelet-derived growth factor, and transforming growth factor beta. *Endocrinology* 1990;127(1):69-75.
68. Stellon A, Davies A, Webb A, et al. Microcrystalline hydroxyapatite compound in prevention of bone loss in corticosteroid-treated patients with chronic active hepatitis. *Postgraduate Medicine Journal* 1985;61:791-96.
69. Nilsen KH, Jayson MI, Dixon AS. Microcrystalline calcium hydroxyapatite compound in corticosteroid-treated rheumatoid patients: a controlled study. *Br Med J* 1978;2(6145):1124.
70. Annelind M, Caviezel R, Schacht E, et al. The influence of ossein-hydroxyapatite compound on the healing of a bone defect. *Curr Med Res Opin* 1986;10:241-50.
71. Durance RA, Parsons V, Atkins CJ, et al. Treatment of osteoporotic patients: a trial of calcium supplements (Ossopan) and ashed bone. *Clin Trials* 1973;3:67-73.

Nutritional Strategies in the Prevention of Osteoporosis: A Summary

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The statistics are alarming: in the U.S. today, approximately 10 million men and women have osteoporosis and another 18 million have osteopenia (low bone mass). Osteoporosis literally means “porous bone,” and is characterized by bone fragility and an increased susceptibility to bone fractures.^{1,2} An estimated 1 in 2 women and 1 in 8 men over age 50 will suffer a bone fracture due to osteoporosis in their lifetime. Although osteoporosis can develop at any age, the risk factors increase with age in both men and women.

Osteoporosis is called “the silent disease” because it is generally a symptomless process resulting from a gradual loss of bone mass. Without proper risk assessment, osteoporosis can go undetected until bones become so brittle that even the slightest trauma causes a bone fracture.¹ Fortunately, experts agree that this type of suffering may be preventable. Studies show that lifetime maintenance of adequate nutrient intake, including calcium and other nutrients important to bone health, along with regular exercise and a healthy lifestyle may reduce the risk of developing osteoporosis.^{7,8,10}

WHAT ARE THE RISK FACTORS?

Many risk factors contribute to the development of osteoporosis.^{1,5,7,8} Some of these include dietary and lifestyle factors such as nutrient deficiencies (e.g., calcium, magnesium, vitamin D), high protein intake, lack of exercise, smoking, excessive alcohol or caffeine consumption, and prolonged use of certain medications (e.g., corticosteroids, antacids). Other risk factors include Caucasian or Asian ethnicity; a thin, small-bone frame; and a family history of osteoporosis.

THE IMPORTANCE OF PREVENTION

The key to preventing osteoporosis is to achieve optimal bone mass during the first three decades of life and maintain it throughout the aging process.^{9,11} The more bone mass acquired by age 35, the less likely it will decrease to a level at which osteoporosis develops later in life.⁷ An optimal intake of calcium not only helps to achieve a greater peak bone mass, but also reduces the rate of age-related bone loss.^{7,13-17,19,20}

CALCIUM: WHAT ARE THE RECOMMENDATIONS?

The Reference Daily Intake (RDI) provides recommendations for daily nutrient intakes considered to adequately meet the needs of most healthy individuals in the U.S. The current RDI for calcium is 1,000 mg per day, which is lower than many health experts recommend.^{9,21-25} Unfortunately, reliable statistics show that an alarming 65% of the U.S. population consumes even less than the RDI for calcium.²⁸

The ability to absorb calcium declines with age in both men and women.² Consequently, there is a need to address factors that affect calcium absorption, particularly when dealing with the elderly population. For instance, large quantities of dietary fiber, certain medications, and nutrient deficiencies (e.g., vitamin D, magnesium) can interfere with calcium absorption. Hypochlorhydria, a condition of low gastric acid production, can also hinder calcium absorption and is quite common in the elderly. Patients encountering any of these factors may need to make dietary adjustments and be sure they are consuming forms of calcium that are easily absorbed.

OTHER POWERFUL BONE-SUPPORTIVE NUTRIENTS

Vitamin D—Vitamin D plays an essential role in maintaining optimal bone mass by acting primarily to assist calcium absorption.²⁹ Vitamin D deficiency is common among the elderly population, owing to less efficient absorption, reduced sun exposure, and reduced intake.²⁹⁻³¹ Studies show that supplementation with 400-800

IU per day of vitamin D effectively reverses vitamin D deficiency in the elderly.^{28,32,34,35}

Magnesium—There is growing evidence that magnesium is required to properly utilize calcium, and experts agree that adequate calcium intake may not ensure normal bone mass if magnesium levels are low.^{38,40} In addition, an excessively high calcium intake combined with a low magnesium intake may further intensify magnesium deficiency.⁴³ Because of the effect of magnesium in calcium utilization, magnesium deficiency may be implicated as a risk factor for osteoporosis.^{41,42}

Trace Minerals—Despite the fact that they are only required in small amounts, studies suggest that trace minerals including zinc, manganese, fluoride, boron, and silicon are no less important to bone health than other minerals. For instance, in a 2-year clinical study, postmenopausal women who increased their intake of both calcium and trace minerals experienced an increase in bone mass.⁴⁶ Conversely, women who increased their intakes of calcium alone or trace minerals alone experienced bone loss, suggesting that the combined nutritional regimen is far more effective.

Ipriflavone—This derivative of naturally occurring isoflavones has a positive effect on bone metabolism. Numerous studies have shown that ipriflavone reduces bone loss in postmenopausal women with osteopenia or established osteoporosis.^{47,48,51-54} One study evaluating the effects of ipriflavone combined with vitamin D showed that the combined nutritional therapy was more effective in reducing bone loss than either therapy alone.⁴⁹

COMPREHENSIVE BONE NOURISHMENT: MCHC

Microcrystalline hydroxyapatite concentrate (MCHC) is a whole bone extract, complete with all the minerals and organic factors in the same proportions naturally found in healthy bone. It is an excellent source of absorbable calcium, a full spectrum of minerals, and other nutrients essential to bone health.⁵⁶⁻⁶⁰

MCHC also contains the proteins found in healthy bone, including growth factors and collagen.⁶¹ Recently, scientists have focused attention on the actions of growth factors in stimulating bone growth. Experts have postulated that the presence of growth factors in MCHC may be one reason why it is so effective in maximizing bone strength.

Studies have repeatedly confirmed the effectiveness of MCHC in maximizing bone mass, and suggest that MCHC is more effective than calcium carbonate and calcium gluconate. In a study comparing the effects of MCHC and calcium carbonate supplementation in postmenopausal osteoporosis, MCHC nearly halted bone loss, while calcium carbonate only slowed bone loss by approximately 50%.⁶⁷

There is a great variation in the quality of MCHC products. The source and processing procedures of bone extract is of utmost importance in determining the quality and effectiveness of MCHC. A high-grade source of MCHC would come from New Zealand and is free of pesticide and heavy metal contamination. MCHC should not be processed with high-heat or excess grinding, which destroys the beneficial organic factors and protein content.⁷¹

CONCLUSION

Experts agree that lifetime maintenance of adequate nutrient intake including calcium and other important nutrients, along with regular exercise and a healthy lifestyle, is essential to reducing the risk of osteoporosis.^{7,8,10} Studies show that MCHC is an excellent source of absorbable calcium and other nutrients essential to bone health, has a positive effect in maximizing bone mass, and is more effective than commonly used forms of calcium.⁵⁶⁻⁶⁰