

# Natural Approach to Preventing Osteoporosis

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## **Introduction: Blood Pressure Regulation and Hypertension**



Osteoporosis, one of the most common conditions associated with aging, is characterized by excessive loss of calcified matrix and collagenous fibers of bone. Holes or pores are formed as bone tissue is lost, increasing the risk of fracture.

Osteoporosis may be primary or secondary. Primary osteoporosis as defined by western medicine, is of unknown origin, occurs with aging, accelerates with menopause, and has no direct or singular cause. There are two types of primary osteoporosis, Type I, which involves losses of trabecular bone, and Type II, which involves losses of cortical and trabecular bone. Secondary osteoporosis has a direct cause. It can be due to endocrine abnormalities, bone marrow disorders, connective tissue disorders, gastrointestinal disorders, renal disorders, and due to some prescription drugs. An effort should be made to rule out the above anytime excessive bone loss occurs.

Nutrition, lifestyle and genetics contribute to the pathogenesis of osteoporosis. Primary osteoporosis can be prevented through proper diet, supplementation, and lifestyle modifications. Many nutrients are required to build bone. The minerals boron, calcium, copper, magnesium, manganese, phosphorus, silica, and zinc; the vitamins A, B6, B12, C, D, folic acid, K; and essential fatty acids are all involved in the bone building process.

The Standard American Diet is deficient in many of the above nutrients, and thus conducive to poor bone health. This diet also contains many excesses that can be considered as bone robbing. The high phosphoric acid content of many American diets due to the consumption of excess soda and other carbonated beverages has been linked to a higher rate of bone fractures. The high content of sodium from consuming processed foods results in a 20% increase in urinary calcium. High sugar intake increases urinary excretion of calcium, magnesium, chromium, copper and zinc. Diets high in the wrong types of fat (trans-fatty acids, hydrogenated, high quantity of saturated) may result in an increased

incidence of osteoporotic fracture. To the contrary, essential fatty acids are important for bone health. In some cases, excess protein consumption may induce a condition called acidosis. The effects of which include increased leaching of calcium from bone.

### **Protein Controversy**

Research has pushed to and fro regarding protein intake and bone loss. Some studies show that a high intake of protein from animal sources (milk, eggs, meat) increases calcium loss in the urine, threatening bone health. While other studies indicate that protein intake from food sources (particularly meats) have a negligible affect on calcium status, and argue that the studies that find high protein intakes increase urinary calcium excretion are based on protein powders and not animal foods. Furthermore, diets very low in protein, such as vegan diets, or strict vegetarian diets, have been shown to promote bone loss over time.

Let's look to the past to learn more about the present association of calcium and protein. The question of high levels of protein in the diet and the issue of calcium excretion is of particular interest in light of Paleolithic diet research for two reasons. First, because estimates of the levels of protein--and specifically animal protein--in the human diet during at least the last 1.7 million years of human evolution (from the time of Homo erectus) are much higher than considered prudent in some sectors of the nutritional research community today. Secondly, at the very same time, the fossil evidence shows Paleolithic humans to have had high bone mass that would have been robust and fracture-resistant compared to that of modern Western humans: in exact opposition to some of current nutritional theory about the alleged role of protein in causing osteoporosis. Let's examine this apparent paradox.

Our hunter-gatherer ancestors certainly consumed a high intake of animal protein. However, they also had low intakes of sodium, higher magnesium, higher boron and higher vitamin K intakes than their modern counterparts. Estimates of calcium intake show that our hunter-gatherer ancestors consumed a diet rich in calcium from plant sources. Plant sources of calcium, such as collard greens and kale, can provide as much as a 55% absorption rate, compared to only 30% absorption rate from dairy. So, while our ancestors consumed large amounts of protein, yet had strong bones, we must note that their diet was better for bone health than our current standard American diet.

Although dietary calcium intake is most often the focus of nutritional recommendations for osteoporosis, what's important is the calcium balance, not just calcium intake. This is another case in which just looking at a single nutrient does not tell the whole story. Rather, you have to consider the entire diet. Whether planning an osteoporosis prevention program, or treating the condition, always work with a certified nutritionist to find your ideal intake of

all essential nutrients, including protein.

### **The Caffeine Connection**

America's love affair with caffeine can have profound negative consequences on skeletal health. Recent research in the American Journal of Clinical Nutrition examined the role of caffeine as a risk factor for bone loss. Women with high caffeine intakes had significantly higher rates of bone loss at the spine than did those with low intakes of caffeine. The researchers concluded that intakes of caffeine in amounts up to 300 mg (equivalent to 18 oz. of brewed coffee) accelerate bone loss at the spine in elderly postmenopausal women. Interestingly, women with a genetic variant of the vitamin D receptor gene appear to be at a greater risk for this deleterious effect of caffeine on bone.

According to the National Coffee Association and the US Department of Agriculture, a 6 oz. cup of brewed coffee contains 103 mg of caffeine. Coffee is not the only caffeine culprit. Soda, tea and medications contribute to the daily caffeine total.

### **Caffeine Content of Beverages, Foods, and Medications**

- **Dark chocolate, semi-sweet** - 1 oz. serving = 20 mg of caffeine.
- **Baker's chocolate** - 1 oz. serving = 26 mg of caffeine.
- **Soft Drinks (various brands)** - 12 oz. serving = 44.5 to 55 mg of caffeine.
- **Aqua Ban (diuretic)** - 1 standard dose = 200 mg of caffeine.
- **Anacin, Excedrin, Midol (analgesics)** - 1 standard dose = 33 to 65 mg of caffeine.
- **Tea, major US brands** - 6 oz. serving = 40 to 70 mg of caffeine (it seems that theanine, a component in tea, prevents the negative effects of caffeine – please read the article on theanine on this site).

### **Skeletal Health and Nutrient Deficiency**

Nutrient deficiencies must be addressed. One example of how a single nutrient lacking in the diet can result in weakening the skeletal system can be provided by magnesium. Magnesium stimulates the thyroid's production of the bone preserving hormone calcitonin, which is necessary for the conversion of vitamin D into its active form, and is required by an enzyme necessary for the formation of new calcium crystals. It is easy to understand why a deficiency of this one mineral can result in vitamin D resistance syndrome, hyperparathyroidism (in which excess parathyroid hormone is produced, causing the withdrawal of calcium from bone), and thus is a causative factor in osteoporosis. Hypochlorhydria (low stomach acid) may also lead to

deficiencies, since an acid medium is required for the absorption of certain minerals, especially calcium. Calcium citrate or citrate malate may circumvent low stomach acid production as a route to absorption. However, it is better to correct the underlying problem as it can lead to other chronic conditions.

### **Vitamin D**

Without vitamin D, the small intestine absorbs no more than 10 to 15% of dietary calcium. In a person with vitamin D insufficiency, the small intestine absorbs, on average, 30% of dietary calcium; during growth, lactation, and pregnancy, the efficiency increases to 80%. Vitamin D deficiency during bone development and growth causes the bone-deforming disease rickets. In adults bone growth stops and bone remodeling continues. Vitamin D deficiency in adults causes secondary hyperparathyroidism that can precipitate and exacerbate osteoporosis. How common is vitamin D deficiency? Surprisingly, it has made a resurgence in neonates and young children, in part because of the campaign to encourage all women to provide all of their infants' nutrition through breastfeeding. Because there is very little, if any, vitamin D in human milk, infants, especially infants of women of color, are at high risk of developing vitamin D deficiency and rickets if they are not given a vitamin D supplement. Breastfeeding provides excellent nutrition, but truth be told, a vitamin D supplement is imperative to prevent deficiency.

The elderly are at risk for vitamin D deficiency because of poor dietary vitamin D intake and decreased exposure to sunlight. Dr. Michael Holick observed that 30%, 42%, and 84% of free-living white, Hispanic, and African American elderly women respectively, were vitamin D deficient at the end of August in Boston.

It has always been assumed that young and middle-aged adults are not at risk of vitamin D deficiency because of their outdoor activities and dietary intake. However, it was recently recognized that 42% of African American women throughout the United States were vitamin D deficient at the end of winter. Hard-working young and middle-aged adults who seldom spend any time outdoors or always wear sun protection outdoors are also at high risk of vitamin D deficiency. Holick observed that 32% of healthy adults 18 to 29 years of age were vitamin D deficient at the end of the winter in Boston. Obesity is often associated with vitamin D deficiency. It is now recognized that, whether vitamin D is ingested in the diet or obtained from exposure to sunlight, it is efficiently deposited in the large body fat stores and is not bioavailable. This is probably the reason that obese persons are chronically vitamin D deficient.

Dr. Holick feels there are 3 reasons for the increase in vitamin D deficiency. First, it is believed that either exposure to sunlight or dietary intake of vitamin D is adequate, or, therefore, that Americans and Europeans are not at risk of

vitamin D deficiency. Second, physicians who perform routine blood work-ups often obtain a blood calcium value. If they find it to be normal, they assume that the patient is vitamin D sufficient, which is not correct. Third, the wise physician that wants to test for vitamin D, erroneously orders an analysis for the active form of vitamin D, 1,25-dihydroxyvitamin D know as (1,25(OH)<sub>2</sub>D), to determine the vitamin D status of a patient. Unfortunately, the test 1, 25(OH) 2D not only is not a measure of vitamin D status. The appropriate test is 25(OH) D. This blood test done through many labs, including Quest, is an accurate measure of vitamin D status.

The simplest way to obtain vitamin D is from moderate exposure to sunlight. Dr. Holick recommends exposure of hands, face and arms, or arms and legs to sunlight for a period equal to 25% of the time that it would take to cause a light pinkness to the skin (1 minimum erythemal dose). This is sufficient not only to satisfy the body's requirement, but also to make sufficient amounts of vitamin D to store in the body for use on rainy days and during times when sun exposure is inadequate to produce enough vitamin D in the skin. Granted this is a very controversial position to take. I have attended numerous lectures given by Dr. Holick, and lectured at the same medical conference for the American College for Advancement in Medicine in May 2004. Holick is certain that he worked out the details to guidelines for the amount of sun exposure needed by people of all skin types to achieve their vitamin D requirement without significantly increasing the risk of skin damage and skin cancer. I'm not currently aware of any dermatologists that endorse this belief, but I can tell you that it has made a big difference in patients that I see in clinical practice. However, if a patient is at risk for, or had skin cancer, I will only recommend oral vitamin D supplementation. Please consult with your physician prior to attempting any sun exposure to replenish vitamin D stores.

#### **Food Allergies/Sensitivities and Intolerances**

A food allergy, defined as an immune response to a normally innocuous substance in the diet, can prevent absorption of some important bone building nutrients. The reason is that food allergies can induce micro inflammation in the small intestine where many bone building nutrients are absorbed, thus reducing surface area of absorption, resulting in a secondary deficiency. Food allergies/sensitivities and intolerances play a major role in health and disease. One fact that you should remember is that approximately 65% of the human immune system is in the intestines. It is called the gut associated lymphoid tissue or GALT. Can you imagine having an immune response to a food (a food allergy)? How could that not affect how you feel, and absorb nutrients.

Lactose intolerance can induce diarrhea in sensitive individuals which can result in loss of important bone building nutrients, especially magnesium.

Gluten intolerance or celiac disease (two completely separate conditions)

definitely induces mineral deficiencies that will affect bone health. The only treatment known to date is a strict gluten free diet. Diagnosis of either condition is made by a medical doctor and the patient is routinely sent to a clinical nutritionist to eliminate the offending foods.

### **Anaerobes and Bone Health**

Bacterial overgrowth of the small intestine is a serious digestive disorder that can inhibit nutrient absorption and lead to many health problems. Although widespread, it is frequently unsuspected in cases of chronic bowel problems and carbohydrate intolerance because its symptoms often mimic other disorders. What does this have to do with bone health? According to a new study by gastroenterologists at the University of Pavia in Italy, bacterial overgrowth of the small bowel may significantly increase the risk of progressive bone thinning. Using hydrogen breath testing, researchers identified fourteen patients with bacterial overgrowth of the small intestine. Researchers also measured bone density in these patients and in healthy controls. Based on World Health Organization criteria, researchers found that 86% and 93% of patients with small intestine bacterial overgrowth had significant bone loss near the hip (proximal femur) and lower back (lumbar spine), respectively. In fact, these patients were more than twice as likely to have bone loss than healthy controls. Their bone loss also tended to be more severe (Am J Gastroenterol 2000; 95(12)). Although underlying mechanisms are still unclear, researchers postulated that bacterial overgrowth in the small bowel could trigger bone loss by promoting calcium malabsorption as well as the loss of key enzymes in the small intestine.

### **Diet and Supplementation**

Since each individual has a unique biochemistry, a proper bone building diet must incorporate healthy foods that contain all essential nutrients, specifically tailored and adjusted for food sensitivities, allergies, carbohydrate sensitivity, level of activity, and current state of health. One's diet should be based on fish, poultry, lean meats, eggs, low fat dairy (if not allergic), or dairy substitutes formulated to match the nutritional profile of dairy (soy milk, rice milk, etc.), legumes (especially those containing isoflavones), whole grains, seeds, nuts, cold pressed oils (such as flax), and vegetables (especially dark leafy greens).

Supplementation should be considered to ensure that all essential nutrients are acquired in optimal amounts. Totals from food should be factored in prior to choosing a dosage. Doses should be adjusted for each individual by a certified nutritionist.

### **Adult recommendations:**

**Boron** - 5mg

**Calcium**- 1200mg to 1500mg

**Copper-** 2mg

**Magnesium-** 400mg to 800mg

**Manganese-** 2mg to 5mg

**Zinc-** 15mg

**B6-** 50mg

**B12-** 50mcg to 5000mcg

**C-** 1000mg to 2000mg

**D-** 1000IU to 4000IU (extreme vitamin D deficiencies require a prescription dose).

**Folic acid-** 800mcg to 1mg

**Ipriflavone** – 600mg

**K** - 45mg

**Strontium citrate** – 680mg (taken away from any other mineral, especially calcium, preferably before bed).

#### **Ipriflavone: the good, the bad, and the ugly**

Isoflavones are compounds found in some plants that are analogous in structure to the estrogens found in animals and humans. Ipriflavone is a synthetic isoflavone synthesized from daidzein, an isoflavone of soy. Many researchers are increasingly touting the benefits of ipriflavone for prevention and treatment of osteoporosis, and other metabolic bone diseases including Paget's disease, and hyperparathyroidism. But what are the facts?

The isoflavone ipriflavone has been found to stimulate the activity of bone building osteoblasts in several studies, and inhibit the effects of osteoclasts, the cells responsible for bone resorption.

Preliminary studies have also found ipriflavone effective in preventing bone loss associated with chronic steroid use, immobility, oophorectomy, and renal osteodystrophy. While ipriflavone appears to enhance estrogen's effect, it does not possess intrinsic estrogenic activity, making it an attractive adjunct or alternative to conventional hormone replacement therapy.

In one study, researchers who previously found that 0.625mg per day of conjugated equine estrogen (CEE) did not prevent bone loss the first year after oophorectomy, discovered that adding 600mg of ipriflavone to the same treatment resulted in inhibitory action of CEE and ipriflavone on the turnover of bone metabolism, and stimulatory action of ipriflavone on bone formation. They concluded that concomitant use of ipriflavone with CEE at an early stage after oophorectomy inhibited bone loss, and was considered to be effective in

maintaining bone mass after oophorectomy

In a study to assess the effects of ipriflavone administration in the prevention of the rapid bone loss that follows oophorectomy in women, patients received either 500mg of elemental calcium or 600mg of ipriflavone in addition to the same daily calcium supplement for 12 months. At the conclusion of the study the data indicated that ipriflavone can restrain the bone remodeling processes, and radial bone density showed no significant modification during the 12-month study period. The researchers concluded that these results demonstrate that ipriflavone administration may prevent the rapid bone loss that follows oophorectomy. In addition, they suggested that ipriflavone might represent an attractive alternative for the prevention of osteoporosis in postmenopausal women with contraindications to estrogen replacement therapy.

In a randomized controlled trial designed to see whether oral administration of ipriflavone could prevent bone loss occurring shortly after menopause, fifty-six women with low vertebral bone density, and with a postmenopausal age less than five years, were randomly allocated to receive either ipriflavone, 200 mg three times daily, or a placebo. All subjects also received 1,000 mg of elemental calcium daily. At the conclusion of the study, vertebral bone density declined after two years in women taking only calcium, but it did not change in those receiving ipriflavone. The researchers concluded that ipriflavone prevents the rapid bone loss following early menopause by reducing bone turnover rate.

The aim of a study at Cattedra di Medicina Interna, University of Rome, was to evaluate the effects of ipriflavone treatment on bone remodeling in primary hyperparathyroidism. Upon completion of the study, the researchers concluded that ipriflavone is indicated in the treatment of metabolic bone diseases characterized by a high bone turnover, since the data suggest that ipriflavone affects bone remodeling by inhibiting bone resorption without affecting bone formation.

In two double-blind studies, 149 osteoporotic women with vertebral fractures, age 65-79, received either 600mg of ipriflavone or a placebo. Both groups received 1000mg of elemental calcium. Urinary hydroxyproline was significantly decreased in ipriflavone treated patients, suggesting a reduction in bone turnover rate. A reduction of incident vertebral fractures was observed in ipriflavone treated women compared with control subjects. The data indicated 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.

However, not all studies are favorable. According to a study in *The Journal of the American Medical Association* (March 21, 2001;285:1482-1488), ipriflavone appears ineffective, based on the results of a 3-year study in Denmark. Researchers studied a group of 474 postmenopausal women. The

women received either 600 mg of ipriflavone daily or a placebo daily for 3 years. In addition, all women took 500 mg of calcium daily. Researchers measured the women's bone density at three different sites (spine, hip and forearm) every 3 months. No difference in bone density was seen between the two groups.

Researchers even found some women who took the supplement experienced a drop in their white blood cell counts, a condition that can impair the immune system. About 13% of women on ipriflavone developed lymphocytopenia, a drop in white blood cells that, in most of these cases, resolved after the women stopped taking ipriflavone. A drop in white blood cells can suppress immune function.

According to the researchers, although earlier studies have suggested ipriflavone does fight bone thinning, these findings suggest that compared with other osteoporosis treatments, ipriflavone offers little benefit. Discuss this issue with your doctor prior to supplementing with ipriflavone.

### **Hormones and Bone Health**

I already discussed the role of the parathyroid in bone health. There are other major endocrine glands that are major factors in skeletal health. The pituitary gland secretes growth hormone stimulates cell division and protein development in bone and cartilage. This is why children with a low production of growth hormone develop growth abnormalities including short stature. If the adrenal glands are stressed, excess cortisol is produced which is a catabolic hormone (tissue breakdown hormone), and may result in poor bone health. The female sex hormones estrogen and progesterone enhance bone health. You can see in the above section on ipriflavone, that low estrogen can equal bone loss. However, researchers are also looking at the importance of progesterone in bone health. Many women might fit the mold of estrogen dominance set by Dr. Lee. It is thought that progesterone promotes bone formation and increases bone regeneration, possibly by enhancing the function of bone-generating osteoblast cells. The male hormone testosterone has been shown to enhance bone health. Men with low levels of this vital hormone are more prone to getting osteoporosis.

### **Strontium for Bone Health**

Other than while studying the periodic table of elements, my first knowledge of strontium occurred while reading Dr. Wright's Guide to Healing with Nutrition, a wonderful book written in 1971 as a treatise for medical students that ended up being a popular press book. Dr. Jonathan Wright discussed how radio active strontium 90 could be found in human bone, and could prevent the absorption of calcium in bone, and lead to cancer. Of course this was of concern in the 1950's when above ground nuclear testing was in vogue. As a young student in nutrition 10 years ago, I actually pondered while reading that

information “I wonder what non-radioactive strontium would do to bone?”

Strontium has been safely used as a medicinal substance for more than a hundred years. It was first listed in Squire’s Companion to the British Pharmacopoeia in 1884. Subsequently, strontium was used therapeutically in the United States and Europe. As late as 1955, strontium compounds were still listed in the Dispensatory of the United States of America.

The processes of bone resorption and formation are tightly governed by a variety of systemic and local regulatory agents. In addition, minerals and trace elements affect bone formation and resorption through direct or indirect effects on bone cells or bone mineral. Some trace elements closely chemically related to calcium, such as strontium have pharmacological effects on bone when present at levels higher than those required for normal cell physiology. The human body contains approximately 320 to 400 mg of strontium in bone, and connective tissue. If we look at clinical studies, indeed, strontium was found to exert several effects on bone cells. In addition to its antiresorptive activity, strontium was found to have anabolic activity in bone, and thus may have significant beneficial effects on bone balance in normal and osteopenic animals. Accordingly, strontium has been thought to have potential interest in the treatment of osteoporosis.

In a three-year, randomized, double-blind, placebo controlled study using 680 milligrams of strontium daily, women suffering from osteoporosis experienced a 41 percent reduction in risk of a vertebral fracture, compared with placebo. And, overall vertebrae density in the strontium group increased by 11.4 percent but there was a 1.3 percent decrease in the placebo group.

In a second study, 353 women who had suffered at least one vertebral fracture due to osteoporosis took varying levels of a prescription for of strontium referred to as strontium ranelate or a placebo. The women who took 680 milligrams of strontium daily had an increase in lumbar bone mineral density of approximately 3 percent per year, significantly greater than placebo. By the second year of the study, there was a significant decrease in additional fractures in the strontium group as compared with the placebo group.

These studies, the benefits of strontium ranelate (reducing fracture risk by as much as 50%) and the history of strontium in medical practice were discussed as an Editorial in the New England Journal of Medicine, January 29 th, 2004.

Further, scientists are looking into the benefits of strontium for osteoarthritis because researchers hypothesized that strontium might also improve cartilage metabolism, and for dental carries since 10% of subjects that had no dental carries in a 10 year study sponsored by the US Navy, resided in a small town that had unusually high levels of strontium in the municipal water supply.

Strontium is available as strontium carbonate, strontium chloride, strontium sulfate, strontium gluconate and strontium citrate. In clinical research strontium gluconate was absorbed better than strontium carbonate. It is my clinical opinion that strontium citrate is absorbed better than the other forms of this metal.

Remember that strontium is very closely related to calcium. They both utilize the same carrier protein for transport. Calcium will win this tug of war effortlessly. The take home message is to take strontium 4 hours away from calcium (preferably other minerals as well) before bed. Currently, I dose strontium at 681mg in one dose prior to bed (each strontium citrate capsule contains 227mg of pure strontium citrate = 3 capsules) on an empty stomach (defined as 2 hours after a meal).

### **Live and Learn**

Lifestyle factors negatively and positively affect bone health. Smoking is toxic to the liver, depletes the body of vitamin C, and decreases blood levels of estrogen. Alcohol inhibits the absorption and increases the excretion of calcium, magnesium, C, zinc, copper, and inhibits B6 functioning. Inactivity is associated with poor bone health. However, regular strength training sessions, three to four times per week, will help increase bone formation. Tension applied to bone by the actions of the muscles during weight lifting stimulates bone regeneration.

### **Shake, Rattle and Roll**

The Beach Boy's weren't the only one's to sing the praises of "good vibrations". Researchers reported that having adult ewes stand on a platform with high-frequency vibration for 20 minutes each day for 5 days a week, for over 1 year, increased femoral trabecular bone density by 32%. Bone trabeculae were also shown to have closer spacing, which is consistent with stronger bone. However, there were no changes in cortical bone. This study follows a shorter study involving rats in which similar high frequency, low amplitude vibrations completely blocked the adverse effects on hind limb bone density induced by tail suspension. Could these animal studies be relevant to osteoporosis studies in human beings human beings?

At the recent meeting of the American Society for Bone and Mineral Research, Ward and Colleagues reported results of a small randomized, placebo controlled study among 20 children with cerebral palsy who used a similar vibration platform for 10 minutes per day, 5 days per week for 6 months. The researchers observed a significant increase in tibial, but not lumbar spine density in the treated group. The effects of a non-invasive 'good vibrations' approach, if shown to be generally applicable and comparably effective in human beings, would be of considerable potential benefit.

Osteoporosis doesn't have to occur. By eliminating dietary components and lifestyle factors that are detrimental to the skeletal system, and exchanging those with behaviors such as acquiring an optimal intake of all bone-building nutrients from diet and supplements, eradicating deleterious bacterial overgrowth, and partaking in a regular strength-training program, one can enjoy having a strong healthy skeletal system throughout their life.

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