Understanding Osteoporosis and Clinical Strategies to Assess, Arrest, and Restore Bone Loss

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Abstract

Osteoporosis is the most common metabolic bone disease, affecting over 20-25 million, mostly older, Americans and is projected to cost $30-40 billion dollars annually by the turn of the century. Women have a four-times greater risk of developing osteoporosis than men, with post-menopausal women at greatest risk. Osteoporosis is an end-stage disease, caused by a chronic disruption of skeletal homeostasis. Significant contributors to this degenerative process include nutritional, endocrine, physical, lifestyle, genetic and environmental factors. The current medical treatment mainstay, hormone replacement therapy, slows bone loss and reduces fracture incidence, but must be taken for ten or more years to realize this benefit. Unfortunately, this approach subjects women to a long-term therapy that poses significant concern due to increased risk of breast cancer, uterine cancer and side effects. The only practical and economic approach is a prevention and treatment strategy which addresses an often complex etiology and includes educating patients in diet, exercise and lifestyle factors that enhance bone density.

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Introduction

Many women will initiate care with an alternative medical practitioner at midlife. For some, the approaching menopause is the harbinger of aging and they want to explore options for enhancing their health and longevity. Others question the safety and/or necessity of hormone replacement therapy (HRT). Still others have a pre-existing health problem that precludes them from HRT and are seeking guidance in alternative therapies. For all, osteoporosis and its complications are of great concern. This concern is well founded. Women have a four-times greater risk of developing osteoporosis than men, with post-menopausal women at greatest risk. Osteoporosis is an end-stage disease resulting from a long-term disruption of skeletal homeostasis secondary to nutritional, endocrine, physical, genetic and environmental factors. The current mainstream medical maxim of menopausal osteoporosis prevention and management is limited to calcium intake (Tums®) and anti-resorptive medications such as estrogens and bisphosphonates. While these prescriptions may be clinically useful, their safety is questioned, and they do not address the individual's unique and often complex etiology for osteoporosis. Osteoporosis is a preventable condition and a treatable condition if the obstacles to normal skeletal homeostasis are removed. Every female from childhood on should be assessed

for her individual risk factors and a rational prevention or treatment plan should be implemented.

Osteoporosis is the most common metabolic bone disease, affecting 20 to 25 million, mostly older, Americans, and it accounts for approximately 1.5 million fractures per year. One-third of American women over 65 will eventually have a spinal fracture. Of the 250,000 individuals who sustain hip fractures each year, 50% will need help with activities of daily living and 15 to 25% will need to enter a long-term care institution shortly after the fracture. Between 12 and 20% of hip fracture victims die shortly after fracture, usually from complications related to either the fracture or surgery.[1]

The health-care costs of osteoporosis are staggering. In 1988 osteoporosis and its consequences were estimated to cost between $7 and $10 billion.[2] With the increasing percentage of the population over age 65, it is estimated that by the turn of the century the number of hip fractures will climb to 350,000 per year and osteoporosis will cost $30 to $40 billion annually.[3] This increase is only partially due to an increased aging population, as data is emerging which indicates that there is an increase in age-specific fracture incidence.[4]

Osteoporosis is a state of decreased bone mass per unit volume of normally mineralized bone. Since bone mass is correlated with bone strength, the decrease in bone mass leads to bone fragility and consequently an increase in fracture risk. Elderly individuals are less likely to protect themselves from a fall because of decreased muscle strength, proprioception, and coordination, so less trauma is necessary to cause a fracture. In addition, medication- or age-related disturbances in cognition, vision and hearing increase the risk of fracture.

In osteoporosis, as in hypertension and hyperlipidemia, there is a long, quiet, symptomless period before clinical symptoms appear. The first symptoms to appear occur when the mechanical workload exceeds the strength of skeletal framework and a fracture occurs with minimal force. The most common sites for osteoporosis fractures are the distal radius (Colles' fracture), the proximal femur ("hip fracture") and the spine (vertebral compression fractures). Vertebral fractures cause loss of height, kyphosis, and back pain; and hip fractures are responsible for significant morbidity and mortality. However, osteoporosis typically affects the entire skeleton, so a fracture can occur at any skeletal site.

Skeletal Dynamics

Bone is a composite tissue consisting of a 30% organic component and a 70% mineral component. The organic component, referred to as osteoid, is a matrix of collagen and non-collagenous proteins, and is produced by cells called osteoblasts. Type I collagen is the most abundant structural protein, accounting for 95% of the total osteoid volume. The mineral component of bone is 95% hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$) and small amounts of magnesium, sodium, potassium, fluoride and chloride. After the osteoid is laid down by the osteoblasts, it becomes mineralized within 25 to 30 days.[1]

Bone mass increases from birth until it reaches its maximum, known as peak bone mass, which occurs for cortical bone at age 30 to 35 and earlier for trabecular bone.[5] Peak bone mass is under genetic control, but in order for the adult skeleton to reach its full genetic potential the individual must have optimal intake of nutrients known to enhance bone density, avoid factors that deplete bone density, and mechanically load the skeleton.[6]

As part of normal physiologic skeletal maintenance and to allocate the matrix and mineral stores along the vectors of mechanical stress, bone is in a constant state of turnover called remodeling. Remodeling consists of bone resorption by osteoclasts and bone formation by osteoblasts. Ideally there is a balance between resorption and formation, producing a stable bone mass with no net change. Once peak bone mass is reached, the annual bone turnover rate is about 25% in trabecular bone and 3% in cortical bone.[1] Given the greater remodeling in trabecular bone it is not surprising that trabecular bone is greatly affected at menopause.

After age 40, age-related bone loss occurs due to a slow loss of cortical bone in both sexes. At menopause women lose cortical bone at a rate of 2 to 3% per year, which is superimposed upon the age-related loss. This accelerated rate of cortical bone loss reverts to age-related levels after eight to ten years.[7] While osteoporotic changes are commonly thought to begin with menopause, in many women half of the total vertebral (trabecular) bone loss occurs before serum estrogen levels decline. Therefore, menopause cannot be the only cause of vertebral osteoporosis.[8] Identifying and preventing this pre-menopausal trabecular bone loss is a key feature in an aggressive osteoporosis prevention program, since accelerated loss of trabecular bone is the major problem in post-menopausal bone loss. The denser the bone is as a woman enters menopause, the less clinically significant accelerated post-menopausal bone loss becomes. The cumulative lifetime losses of bone mass range from 20 to 30% in men to 40 to 50% in some women.[5]

Bone turnover is influenced by many factors, such as acid-base balance, physical activity, nutrient intake, endocrine and lesser-understood local bone factors. The principal regulators of bone remodeling are parathyroid hormone (PTH), vitamin D, and calcitonin, since they tap into bone in order to maintain optimal serum calcium levels. As serum calcium declines PTH is released, stimulating the renal hydroxylation of vitamin D to its active 1,25 $(OH)_2D$ form, which then enhances intestinal and renal absorption of calcium. PTH also activates osteoblasts that, via cytokines, activate osteoclasts, thus maintaining net bone formation through balanced osteoblastic and osteoclastic activation. Once optimal calcium levels are reached, calcitonin inhibits osteoclast activity and also stimulates the renal clearance of calcium. Several other hormones influence bone remodeling to a lesser extent, including estrogens, androgens, progesterone, glucocorticoids and thyroid hormone.

Estrogen increases the formation of calcitonin, thus inhibiting osteoclastic activity as well as stimulating calcium absorption by the intestine and facilitating the synthesis of vitamin D. It is well documented that estrogen replacement therapy decreases bone resorption and stabilizes bone mass, but confers only a 2% increase in bone density at best, and must be taken for ten or more years to reduce fracture incidence.[9] Given that in many women trabecular bone loss appears before the decline in serum estrogen,[8] and that the decline in progesterone precedes that of estrogen and is of greater magnitude,[10] the question is "Is there a role for progesterone in maintaining normal skeletal dynamics?" Although the role of progesterone is less well understood than that of estrogen, progesterone is active in bone metabolism and appears to promote bone formation. Some authors have postulated that post-menopausal osteoporosis may be in part a disease of progesterone deficiency. One study showed higher progesterone levels in 17 post-menopausal "slow losers" of bone than in 16 "rapid losers" of bone from the same population.[11] In a study of 100 post-

menopausal women followed for three years, Lee reports a progressive rise in bone density and zero fractures in women using estrogen and a transdermal natural progesterone cream alone. The benefit of the progesterone was independent of supplemental estrogen. Women in both groups were placed on a high calcium diet with additional supplementation of calcium, beta carotene, vitamin C and vitamin D. They were also told to exercise moderately and avoid cigarettes.[10] The treatment of osteoporosis with progesterone appears encouraging and deserves significantly more research. Maintaining normal progesterone production in the pre- and peri-menopausal woman is an especially important component in any prevention program. The herb *Vitex agnus-castus* increases luteinizing hormone, which enhances the production of estrogen and progesterone from the ovary.[12] *Vitex* is not useful in the post-menopausal woman, however, since its target organ cannot be stimulated to produce estrogen.[13] In addition, zinc, vitamin A, vitamin C and licorice root enhance progesterone production.[14]

In therapeutic doses, glucocorticords inhibit osteoblast formation and sensitize existing osteoblasts to PTH and vitamin D, which in turn activates osteoclasts and increases bone resorption. Glucocorticoids depress collagen synthesis, resulting in defective osteoid formation, and also inhibit calcium absorption by the intestine.

Adequate levels of dehydroepiandrosterone (DHEA) may be essential for maintaining bone mass in both men and post-menopausal women. DHEA levels decline with age; however, this is not observed with other adrenal steroids.[15] Several studies have observed that lower serum DHEA-Sulfate (DHEA-S) levels have been positively correlated with lower bone density and can be used as an indicator of low bone mineral density.[16-18] This observation, however, has been refuted by other researchers, who found no significant association of DHEA-S levels with bone mineral density at any site both before and after adjustment for age, obesity, cigarette smoking, and use of anti-hypertensive medications.[19] The mechanisms by which DHEA might prevent osteoporosis are: (1) DHEA, like estrogen, may inhibit bone resorption; (2) DHEA may stimulate bone formation and calcium absorption; and (3) DHEA can be further converted into estrogen and testosterone and thereby provide protection against bone loss.[15] The exact nature of DHEA's role in either the pathogenesis or treatment of osteoporosis remains unclear and deserves more research.

Lastly, thyroid hormone activates osteoclastic activity and bone resorption. As resorption raises serum calcium levels there is a decrease in PTH and vitamin D, resulting in a decline in calcium absorption. Hyperthyroidism is associated with increased bone resorption and no concomitant bone formation, resulting in a decrease in bone mass. Post-menopausal women who have had a hyperthyroid episode treated with radioiodine have reduced bone density whether or not they receive thyroxine therapy and should be evaluated for their bone density status.[20] Long-term suppressive therapy for thyroid carcinoma with thyroxine is also associated with bone loss.[21] Long-term thyroxine therapy for hypothyroidism results in bone loss at thyroxine equivalent of 1.6 µg/kg, whereas doses lower than 1.6 µg/kg are not associated with lower bone density values. Estrogen appears to negate thyroid-hormone-associated bone loss in post-menopausal women.[22] Thyroxine-treated women with low TSH levels lose more bone mineral from the spine than those without known thyroid disease. Even a relatively modest degree of

over treatment results in decreased bone mass.[23] The best strategy for hypothyroid patients is to monitor serum TSH to find the lowest possible thyroxine dose that will maintain a euthyroid state and to assess bone density status before the onset of menopause.

The causes of osteoporosis are diverse and each must be ruled out in any person presenting with bone loss. (see Table 1) The most frequent forms of osteoporosis encountered in general practice are considered Type I, post-menopausal osteoporosis, and Type II, age-related osteoporosis. In both types an uncoupling between bone resorption and bone formation occurs.

Type I osteoporosis generally involves greater trabecular bone loss compared with cortical bone loss and therefore has a tremendous impact on the vertebral bodies. Recall that before menopause women may have lost close to 50% of their vertebral trabecular bone. Although the adrenal glands and adipose tissue are supposed to continue supplying estrogens, many women arrive at menopause in a state of adrenal fatigue and do not have enough of these estrogens available to them to protect their skeleton. With the decline in estrogen, the osteoclastic "brake" via calcitonin is lost and bone resorption accelerates, while bone formation remains static; this results in a net decline in bone mass. As mentioned above, a decline in estrogen results in a decrease both in calcium absorption and vitamin D formation and further accelerates resorption via PTH. If we are to make any impact on preserving the bone density in the menopausal woman we must: (1) arrest and restore the vertebral bone loss in the pre-menopausal woman; (2) preserve adrenal function and/or consider some form of

TABLE 1. Common Risk Factors For The Development of Osteoporosis.[1,3,5,7]

Nutritional
- Lactose intolerance
- High animal protein intake
- High caffeine intake
- Excessive soft drink intake
- High sodium intake
- High sugar intake
- Low nutrient intake- Calcium
  Magnesium, Trace
  Minerals, Vitamin D

Medical Problems
- Anorexia Nervosa
- Hyperthyroidism
- Cushings Syndrome
- Hyperparathyroidism
- Type 1 Diabetes
- Rheumatoid Arthritis
- Prolactinoma
- Ankylosing Spondylitis
- Connective Tissue Disease
- Malabsorbtion
- Primary Biliary Cirrhosis
- Oophorectomy
- Low Adrenal Reserve
- Early menopause
- Amenorrhea
- Liver Disease
- Kidney Disease
- Homocysteinemia
- Bone Marrow Tumors
- Osteogenesis Imperfecta

Genetic
- White or Asiatic ethnicity
- Female sex
- Small body frame
- Positive family history

Lifestyle
- Smoking
- Excessive alcohol intake
- Inactivity
- Null Parity
- Lack of sunlight exposure

Medications
- Steroids
- Thyroid Replacement Drugs
- Heparin
- Lithium
- Chemotherapy
- GnRH Agonist or Antagonist Therapy
- * Extended Tetracycline Use
- * Diuretics Producing Calcuria
- * Aluminum Containing Antacids

* Not yet associated with decreased bone mass although identified as either toxic to bone in animals or inducing calcuria and/or calcium metabolism in human beings.

HRT for those at greatest risk of fracture; (3) insure adequate intake and absorption of nutrients that enhance bone health; (4) address lifestyle factors that are deleterious to bone density; and (5) encourage regular physical activity. In Type II osteoporosis, trabecular vs. cortical bone loss is essentially equal and has the greatest impact on the femur and other long bones. With age, intake and intestinal absorption of calcium and other nutrients decline, and the hydroxylation of vitamin D is impaired. The net effect is a secondary hyperparathyroidism and accelerated bone resorption as the body attempts to stimulate vitamin D synthesis and calcium absorption in order to maintain serum calcium levels that cannot be met through diet. To slow or reverse osteoporosis in the elderly, we must: (1) insure adequate nutrient intake and absorption; (2) encourage sunlight exposure to activate vitamin D; (3) encourage regular weight-bearing exercise, and, as before; (4) address lifestyle factors that are deleterious to bone density.

Assessment and Diagnosis

Since osteoporosis is clinically silent until a fracture occurs and since it affects a large percentage of the population, prevention and early intervention are essential before a fracture occurs. In addition to assessment of a patient's potential for osteoporosis from the known risk factors (see Table 2), there are both imaging and biochemical markers that can enhance clinical decision making.

Dual Energy X-ray Absorptiometry (DEXA) quantifies bone density at the relevant fracture sites of the lumbar spine, proximal femur, and forearm with excellent accuracy.[24] It is a quick, non-invasive test that exposes the patient to an extremely low level of radiation. It can be used to document current skeletal density and risk of fracture and is useful in monitoring the efficacy of therapy. The cost is about $275. Reimbursement for this study is variable, and many insurers require additional paperwork regarding the patient's risk factors before approving reimbursement. DEXA studies are particularly useful in post-menopausal women considering HRT, patients with x-rays demonstrating osteopenia, a history of amenorrhea, hyperparathyroidism, thyroid hormone therapy, luteinizing hormone antagonist therapy, glucocorticoid excess or therapy, long periods of immobilization, alcoholism, cigarette use, and a strong family history of osteoporosis. Bone density measurements provide the necessary data so that the physician and woman can make a rational decision regarding her need for treatment and her treatment options.

The amount and rate of bone loss vary from individual to individual and from skeletal site to skeletal site. Recently, two urine tests have been developed to assess bone resorption rates, thus distinguishing "rapid" losers of bone from "slow" losers of bone. The tests assay for collagen markers, either pyridinium cross links or N-telopeptides, which are released during bone resorption. Although these tests do not answer why a person may be losing bone, they can be useful in determining rapid resorption rates prior to actual changes in bone density and can help to monitor therapy.

Diet and Bone Health

Diet has a significant effect on bone health. Diets low in calcium and vitamin D and high in protein, phosphates, sodium, or sugar have all been demonstrated to accelerate bone loss.[25] Of particular importance is an alkaline ash diet. Chronic ingestion of an acid ash diet creates a measurable reduction in bone density, as the bone is resorbed to liberate calcium and other minerals to buffer the acid

state. As the bone is resorbed there is no compensatory stimulation of bone formation, thus resulting in a net loss of bone.[26] The recently published book *Better Bones, Better Body* by Susan E. Brown, Ph.D., has an excellent discussion on the alkaline ash diet.[3]

Excessive phosphates found in soft drinks and high protein diets can induce a "nutritional hyperparathyroidism" if dietary intake of calcium is not adequate. The optimal calcium to phosphorous ratio is estimated to be 1:1, and the standard American diet provides a 1:2 to 1:4 ratio. Ratios less than 1:2 enhance bone resorption regardless of calcium intake.[27]

In the seminal article, "Paleolithic Nutrition," Eaton and Konner demonstrated that the diet of the hunter-gatherer populations of 40,000 years ago contributed to their massive bone density. This diet was about 35% meat and 65% vegetables and fruit. There was little or no cereal grain consumption and no dairy foods at all. It was naturally low in sodium (690 mg) and high in calcium (1580 mg). While modern agribusiness and food processing have radically altered our dietary patterns over the last 100 years, we have not evolved genetically, and it may be wise to adopt our ancestors' dietary patterns as we try to prevent degenerative diseases such as osteoporosis.[28] High dietary intakes of either sodium or sugar, which are quite common in the standard American diet, will cause increased urinary calcium excretion and should be reduced.[3]

The goal of dietary intervention is to counsel patients to eat a nutrient-dense diet so that supplementation to the diet is as minimal as possible. As my noted nutrition professor, Steve Austin, N.D. taught me, "Food is more powerful than pills!" Teaching patients the concepts of a low acid-ash whole foods diet, with particular emphasis on increasing their intake of non-starchy vegetables, introducing them to sea vegetables, and reducing their intake of refined carbohydrates and grains will reduce their need for supplements as well as improve their overall health.

Nutrients and Bone Health

Calcium: The benefits of calcium supplementation on osteoporosis have received considerable attention, and unfortunately these supplements are considered to be a panacea by many. It must always be remembered that calcium is an integral part of any comprehensive approach to osteoporosis, but calcium alone is not the answer to osteoporosis.

Calcium appears to confer benefit in several areas: (1) optimal calcium nutriture as a child and young adult helps in achieving peak bone mass;[6] (2) calcium supplementation significantly decreases bone loss in the lumbar spine (trabecular bone) in pre-menopausal women and in early peri-menopausal women;[29] (3) calcium supplementation reduces cortical bone loss during the first five years of menopause;[30] (4) calcium supplementation produces a sustained reduction in the rate of total body bone loss in women at least three years after menopause.[31] Calcium supplementation alone does not appear to slow the rapid loss of trabecular bone during the first few years of menopause.[32] However, if peak bone mass is achieved and the pre-menopause loss of trabecular bone is arrested, then this acceleration of bone loss should become clinically less significant.

Since a large percentage of Americans fail to meet recommended guidelines for optimal calcium intake, supplementation is often necessary.[33] There are many forms of calcium available for supplementation, but the benefit of supplementation is closely related to its absorbability. When compared with an equal dose of elemental calcium from calcium

carbonate, 500 mg elemental calcium from calcium citrate-malate was more effective at increasing bone density in post-menopausal women.[32] Microcrystalline hydroxyapatite concentrate (MCHC), a whole bone preparation from raw calf long bones that has bone minerals in their natural ratios as well as residues of the osteoid matrix, proteins, and glycosaminoglycans,[34] has demonstrated enhanced absorption and increased bone density when compared with calcium gluconate in post-menopausal women with primary biliary cirrhosis.[35] In addition MCHC has been shown to help shorten fracture union time and to facilitate union in non-union fractures.[36] This might be potentially useful in treating fractures in osteoporotic individuals and warrants further investigation.

Magnesium: Magnesium depletion impairs mineral homeostasis by reducing skeletal and renal sensitivity to PTH and by reducing the activation of vitamin D.[37] Sixteen of nineteen osteoporotic women demonstrated low trabecular bone magnesium and abnormal crystalline structure of the bone.[38] This abnormal mineralization is probably another reason why osteoporotic bone is more subject to fracture than normally mineralized bone. Magnesium supplementation (250-750 mg/day) appears to significantly increase bone density and reduce the incidence of fracture.[39]

The appropriate intake ratio of calcium to magnesium is controversial, with some authors supporting the current 2:1 ratio and others recommending a 1:1 ratio.[40] Further studies are needed to elucidate the best form and ratio for osteoporosis prevention and treatment.

Vitamin D: Vitamin D is one of the most important hormone nutrients affecting bone, with its role in regulating intestinal and renal absorption of calcium and, in concert with parathyroid hormone, increasing bone resorption. Vitamin D deficiency has been found in 25% of elderly patients presenting with hip fracture.[5] This is due to decreased absorption, impaired hepatic and renal conversion to 1,25(OH)2D and/or deficient exposure to sunlight. Even a low normal level of serum 25-hydroxy vitamin D is associated with osteopenia in the proximal femur in men and women between 50 and 80 years of age.[41] The optimal dose appears to be between 400 and 800 IU per day.[42] Encouraging all patients to enjoy regular outdoor activity will also enhance their vitamin D status.

Vitamin K: Vitamin K is important to the maintenance of healthy bone. The carboxylation of osteocalcin, one of the most abundant non-collagenous proteins in the osteoid, is vitamin K dependent. Osteocalcin facilitates calcium binding to the hydroxyapatite matrix.[43] Vitamin K deficiency has been observed in osteoporotic women.[44] Administration of vitamin K (1 mg/day for 14 days) has been shown to reduce urinary calcium and increase osteocalcin binding to hydroxyapatite in postmenopausal women.[43] Because 90% of the vitamin K in the human liver is made by the intestinal flora,[43] impaired vitamin K status should always be considered in anyone with frequent or long-term antibiotic treatment. Vitamin K is fat soluble and can be malabsorbed in those with chronic gastrointestinal problems or with malabsorption.[27] While coagulation function tests are often used as indicators of vitamin K nutriture, these are not adequate to evaluate osteocalcin integrity. Carboxylation of bone proteins may be more susceptible to reduced vitamin K levels than coagulation proteins.[43] Vitamin K is found in green leafy vegetables, legumes, canola oil and soybean oil.

Boron: Numerous animal and human studies suggest that boron interacts with other nutrients and plays a regulatory role in bone

metabolism.[45,46] In post-menopausal women, boron supplementation at 3 mg per day reduced the urinary excretion of calcium and magnesium and elevated the concentrations of serum 17-ß estradiol and serum testosterone. These results were most marked when dietary magnesium was low.[47]

Trace Minerals: Several trace minerals, especially copper, zinc, and manganese, are essential in bone metabolism as co-factors of specific enzymes in osteoid development and osteoid mineralization.[48] In a study of 59 post-menopausal women, the effects of calcium supplementation (as calcium citrate-malate, 1000 mg elemental Ca/day) with and without the addition of zinc (15.0 mg/day), manganese (5.0 mg/day) and copper (2.5 mg/day) on the lumbar spine was evaluated over two years. Bone loss was arrested the most in the-calcium-plus-trace-minerals group.[49]

The Homocysteine Connection: Elevated levels of homocysteine in post-menopausal women may accelerate osteoporosis by interfering with collagen cross-linking, causing a defective osteoid that cannot be effectively mineralized.[27] Vitamins B6, B12, and folic acid, and the trimethylated amino acid betaine, are necessary co-factors in the intermediate metabolism of homocysteine, and their supplementation has been shown to reduce elevated homocysteine levels.[50]

Lifestyle and Bone Health: Alcohol intake and cigarette smoking are associated with osteoporosis. Alcohol inhibits absorption and increases excretion of calcium, magnesium, ascorbic acid, zinc and copper. Cigarette smoking depletes the body of ascorbic acid and exposes it to a number of toxins, such as cadmium and lead, which directly damage bone and interfere with calcium absorption.[3] Caffeine in average daily doses of two to three cups accelerates bone loss from the spine and total body in women with calcium intakes below 800 mg/day.[51] Modification of these habits is essential to any bone-building program.

Exercise and Bone Health: Exercise has a central role in the prevention and treatment of osteoporosis. No agent, hormonal or mineral, can cause a skeleton to be heavier or sturdier than required by the uses to which the owner puts it.[6] Peak bone mass is achieved both by adequate bone-building nutrients and by mechanically loading the skeleton. In order for exercise to be effective it must be continued throughout life. Simply put, inactivity leads to bone loss. In one study, women who participated in vigorous exercise two or more times per week, or whose total physical activity amounted to four hours per week had significantly higher bone density than those who exercised less than two times per week or did less than four hours of physical activity per week. When other lifestyle factors were taken into account, such as smoking and drinking, a significant difference in bone density was found between physically-active and sedentary women, but not between the smokers and non-smokers or drinkers and non-drinkers.[52] It also appears that in post-menopausal women exercise plus calcium or exercise plus estrogen is more effective than exercise alone.[53]

Exercise builds bone mass and muscle mass, and enhances proprioception, coordination and flexibility. Exercise reduces the need for medications such as anti-hypertensives, anti-depressants, and hypnotics that can alter balance and coordination and result in a fall. In addition to reduced femoral neck density, other independent risk factors for hip fracture include slower gait, difficulty in doing a tandem walk, small calf circumference, and reduced visual acuity.[54] Exercise will have a positive impact on the above risk factors, with the exception of reduced visual acuity. In a study of 200 individuals 70 years of age or

older, doing Tai Chi and computerized balance training interventions for 15 weeks, there was a 47.5% reduction of risk of multiple falls! Range of motion in the lower extremities showed improvement, and lower blood pressure was observed before and after a 12-minute walk following Tai Chi participation.[55] With improved coordination, muscle strength and flexibility, a slip does not become a fall and a fall does not become a fracture.

Exercise does not appear to slow the bone loss associated with menopausal involution. However, exercise does have significant effect upon attaining peak bone density and maintaining bone mass, and it will have a positive effect whenever it is started.

Although any and all activities will have a positive effect on bone if done often and long enough, perhaps the best program to maintain bone density is a full yoga program. It is weight bearing, and in the various yoga postures the muscles pull on the bone, thus stimulating additional bone remodeling. If the patient has had a vertebral compression fracture or is at immediate risk of fracture, it is important that they work with a yoga teacher experienced in adapting yoga postures for those with musculoskeletal injuries. For some, gentle movements in a pool or bathtub filled with water can provide a welcomed buoyancy as they increase mobility and gain muscle strength. Osteoporotic patients may also be referred to physical therapists in order to learn optimal body mechanics and reduce the risk of fracture.

Additional Pharmaceutical Interventions

Sometimes a comprehensive natural approach to increasing bone mass is not enough or is not implemented soon enough. In these cases, pharmaceutical intervention besides HRT can help stabilize bone mass. Even when a pharmaceutical agent is used it will be much more effective when prescribed with a comprehensive bone-building program.

Bisphosphonates: The bisphosphonates editronate (Didronel®) and alendronate (Fosamax®) are used to inhibit bone resorption. The exact mechanism is not fully understood, but may be related to the inhibition of hydroxyapatite dissolution or an inhibition of osteoclastic activity. It is not yet certain that therapy with editronate prevents fractures. Alendronate is a highly-selective inhibitor of bone resorption and appears to reduce the number of new vertebral fractures and increase total bone mineral density. Treatment of osteoporosis unresponsive to other measures appears promising. Long term safety has not been established, although extension studies are ongoing. If alendronate is used it must be taken on an empty stomach and away from calcium and other multi-valent cations.[56]

Calcitonin: Calcitonin acts as a physiologic antagonist to parathyroid hormone and, administered intranasally, is used to decrease the rate of bone resorption in osteoporosis and Paget's disease. Response to calcitonin varies, but patients with high bone turnover appear to have the best improvement in bone mass.[5]

Fluoride: Fluoride ions are incorporated into the crystal lattice of hydroxyapatite, resulting in a mineral phase of greater crystallinity. Sodium fluoride stimulates osteoblastic proliferation, which results in increased bone formation.[5] Unfortunately, in spite of an increase in bone mass, an increased risk of hip fracture and other non-vertebral fractures has been observed with fluoride supplementation.[57]

Conclusion

The health of the skeleton is a reflection of the total health of the body. Osteoporosis prevention and treatment is an opportunity not only to address skeletal

concerns but also to make a tremendously positive impact on a woman's total health and well-being. The current medical mainstay of osteoporosis prevention and treatment, estrogen, slows bone resorption but fails to address both primary prevention and the chronic systemic dysfunctions that lead to osteoporosis. Estrogen reduces fracture incidence by 50%, but needs to be taken for ten or more years to produce this reduction, while subjecting the user to significant side effects and an increased risk for breast cancer. Women should be screened for osteoporosis and estrogen should be used only for those at greatest risk for osteoporotic complications. The use of progesterone in maintaining bone density appears quite promising and deserves more research. The only practical and economical approach to osteoporosis is an aggressive preventative and restorative program that includes educating patients in diet, exercise and lifestyle factors that enhance bone density, and prescribing additional nutrients when needed. This will increase bone density in most women while sparing them a potentially-hazardous lifelong therapy.

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