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# Osteoporosis: Bones in Contention

Course #458  
10 Contact Hours

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## About the Authors

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## Purpose and Goals

The goal of the enclosed course is to provide a comprehensive overview of osteoporosis for nurses and other healthcare professionals. Emphasis is on an understanding of the life-long risk factors for the development of the disease, particularly those that reflect lifestyle choices. New treatment approaches are outlined based on medical research. Consideration is also given to the unique challenges presented by osteoporosis in men.

## Instructional Objectives

Upon completion of the course, the learner will be able to:

1. Identify the types of bone tissue and their anatomical distributions.
2. Outline the physiological and emotional sequence of events resulting in osteoporosis.
3. Define the role of the "skeletal storehouse" in maintenance of the body's calcium balance.
4. Name health conditions that can increase risk for development of osteoporosis.

5. List lifestyle factors contributing to the risk for osteoporosis.
6. Identify the prevalence and risk factors for development of osteoporosis in men.
7. Outline current methods and criteria for the diagnosis of osteoporosis.
8. List the primary pharmacological approaches to osteoporosis treatment.
9. Recognize recent medical research findings relevant to osteoporosis treatment and prevention.
10. Identify ways to minimize fractures in patients with osteoporosis.

## Introduction

Leaning heavily upon her cane, the elderly woman strained to look upward. It was difficult since her head jutted sharply forward from her body and her back was twisted and bent. An ungainly dowager's hump pulled her jacket tight across her drooping shoulders. Every day, thousands like her hobble about their homes, struggle to climb stairs, brave boarding a bus or shuffle along the streets, fearful that a stumble will lead to a fracturing fall.

Age and a long life have not brought them freedom to enjoy their remaining days; instead, they are burdened by the visible evidence of their frailty and robbed of their independence. They are victims of the most common bone disease in the world, osteoporosis.

According to the International Osteoporosis Foundation, osteoporosis affects as much as one in four women and over one in eight men over the age of 50. One of the least understood and most hotly debated chronic degenerative diseases in medical science today, its actual cause is not fully known, although numerous theories abound.

This serious public health problem, whose name literally means porous or brittle bones, afflicts over nine million Americans, another 48 million have low bone density. Millions more currently have the disease but don't know it, as the symptoms are most times undetectable until a bone breaks. Latest estimates from the NIH indicate 1.5 million fractures a year which, besides the personal toll on the patient, costs the nation \$14 billion annually.

Since 1900, the number of Americans over the age of 65 has quadrupled. With the development of antibiotics, vaccines, enhanced nutrition, improved sanitation

and food processing, many diseases have been eradicated. As a result, life expectancy has increased dramatically, now reaching the predicted age of 79 for women and 74 for men. Thirteen percent of the U.S. population is now 65 years of age or older; that's approximately 35 million senior citizens, and that proportion is expected to double to over 88.5 million by 2050. Seniors 75 years of age and over are the most rapidly growing group in our society.

In correlation with the increase in life expectancy, the rate of age-related illnesses has also increased. These illnesses include vascular diseases of the heart, brain, and limbs; certain types of cancer; Alzheimer's disease; and osteoporosis.

Recent information obtained from the National Institute for Arthritis and Musculoskeletal and Skin Diseases reported that osteoporosis is responsible for almost 300,000 hip fractures, 500,000 vertebral fractures, 250,000 wrist fractures and approximately 300,000 fractures at other sites.

Fractured hips are the leading cause of accidental death in people over 75 years of age and the second leading cause of death in those aged 45 to 74. Nearly two million Americans over the age of 65 will fracture their hips this year, and 15 to 30 percent of them will die within one year of their injury due to complications associated with their immobility.

To put these numbers into perspective, more women are likely to die from an osteoporosis-related fracture than from breast, cervix and uterine cancer combined. Of the survivors, more than 40 percent will be unable to get around or care for themselves without mechanical aids or someone's help.

In the United States, over 60,000 patients are admitted to nursing homes every year after suffering a hip fracture. This statistic makes hip fractures in women the second leading cause of admission to long-term care facilities. Moreover, only 40% of these patients fully gain their former level of mobility.

## History of Osteoporosis: Facts and Figures

Although British surgeon Sir Astley Cooper wrote in 1824 that the skeleton seemed to become more fragile with age, osteoporosis was not fully described in medical texts until the mid 1920's. After

the discovery of X-rays in 1895 by the German physicist, Wilhelm Roentgen, treatises on fractures in weakened bones began appearing in the German medical literature. In 1941, Boston endocrinologist Fuller Albright explored the increased bone breakdown after menopause, but his findings drew little attention since the American medical community, like the public at large, was focused on more immediate issues: fear of polio epidemics was rampant; pneumonia was a lethal killer; and syphilis and TB were medically uncontrollable crippling.

Even after the discovery of antibiotics, public attention was directed toward two other dramatic killers, cancer and heart disease. The incidence of osteoporosis was increasing right along with life expectancy, but an awareness of its seriousness didn't keep pace with the threat it posed to the health and well being of the elderly.

Post-menopausal osteoporosis was first described in the 1940s but financial backing for research projects was slow to come. In 1965, a landmark conference, sponsored by the World Health Organization (WHO) and the National Institutes of Health (NIH), formulated a worldwide epidemiological study of osteoporosis giving recognition to the seriousness of the problem. In 1984, the National Institutes of Health publicized this disease, citing it as a significant threat to health and emphasizing that bone loss could be reduced by estrogen therapy, calcium, good nutrition and exercise.

These findings were expanded in a 1987 conference sponsored by the NIH and the National Osteoporosis Foundation. Fifteen years ago, only about 15 percent of Americans knew about this disease. Today, over 90 percent do. In view of the latest research and information, osteoporosis should be viewed as a largely preventable disease.

## Bone Basics

Most people don't think about skeletons except at Halloween, or maybe worry about one tucked away in a closet, so it is easy to take our skeletal health for granted. The human skeleton is like the super-structure of a building with its framework supporting and protecting the organs of the body.

Bones are functional in design, and vary depending on location and intended use. Protective bones like the skull and pelvis

are thick, while arm and leg bones are long, hollowed out cylinders that combine lightness with strength. Engineers recognize the cylinder as the geometric form best able to withstand forces from above and below (compression), as well as twisting and bending forces (torsion). The leg bones can endure a tremendous amount of pressure from running and jumping, while resiliency is the primary characteristic of the 24 vertebrae.

Bone is living, growing tissue. It is composed of collagen, a leathery protein material that differs from the collagen found in other parts of the body. Chains of peptide molecules make up the bone collagen, and they are bound around each other in long strands in the base material of the bone. These strands are stacked together like a wall of bricks, with tiny openings within which calcium and phosphorus crystallize. Calcium ions are small, but when placed in a latticework of crystals, they form a huge surface area. This combination of collagen and calcium makes bone both flexible and strong, which in turn helps it to withstand stress.

Ninety-nine percent of the body's calcium is held in the bones, and it combines with phosphorus, hydrogen and oxygen to form crystals called hydroxyapatite, which resemble table salt crystals. This gives it hardness and structural integrity. Bone crystals also contain sodium, potassium, magnesium, strontium, carbon and chlorine. Without these minerals, collagen would be like a pile of spaghetti and not at all like bone. The remaining one percent is found in the blood.

Separately, neither collagen nor the crystals are durable; but together, they are amazingly strong. Throughout your lifetime, old bone is removed (resorption) and new bone is added to the skeleton (formation). During childhood and teenage years, new bone is added faster than old bone is removed. As a result, bones become larger, heavier, and denser. Bone formation outpaces resorption until peak bone mass (maximum bone density and strength) is reached around age 30. After that time, bone resorption slowly begins to exceed bone formation. Bones are heaviest and strongest around age 35, at which point they make up about 10 percent of the body weight.

Bone tissue is arranged in two different ways. Some is densely packed into compact layers forming a tough outer shell

called the cortex. Eighty percent of the skeleton is this hard bone. The remaining 20 percent is woven into a meshwork of thick, tough plates called trabecular bone. This spongy, porous tissue fills the inner cavities of the bones, which also contain marrow and fat. Cortical bone receives added strength from tightly packed rods called Haversian systems. Their canal-like walls are hardened with minerals and are usually too narrow for blood cells to pass through, but fluids and ions are able to circulate freely. Due to this continuous movement and chemical activity, bone tissue is able to replace itself completely about every seven years. The overall structure remains fairly constant, but the tiny mineral crystals and molecules that compose it are replaced.

Bone serves as an effective reservoir of essential minerals for the rest of the body, supplying calcium to tissues and acting as a dam when the tissues are getting too much. If the concentration in the blood begins to fall, the bones release more calcium to make up for the deficit. If the concentration gets too high, the bones extract the excess. There is a state of dynamic equilibrium in which the calcium is always moving in one direction or another to maintain a constant level in nonskeletal tissue. It attracts other elements such as lead and mercury, which are toxic. The bones take up and bind fluoride, the same substance that most communities add to their drinking water to prevent dental caries.

By measuring strontium in excavated bones, anthropologists and archaeologists have learned facts about the eating habits of ancient man and animals, since meat eaters accumulate less of this element than vegetable eaters do. In modern day nuclear tests or accidents, large amounts of radioactive strontium reach the bone and remain there, destroying bone tissue over a long period of time.

While 99 percent of all calcium is in the bones, the remaining one percent is spread throughout the body and is crucial to the proper functioning of all organs. Calcium is necessary for the smooth performance of all body systems. Circulating in the bloodstream, it is available to energize countless electrical impulses and chemical reactions that operate the heart, brain and other organs. Muscle cells cannot contract without it, nerve cells cannot send impulses without it, and it's needed for blood to clot. Dangerously low levels of calcium

can cause life-threatening seizures, organ malfunctions and abnormalities in the heartbeat. It is so important that the body has a specific mechanism for keeping available calcium at an appropriate level at all times.

Since food and dietary supplements are the only outside sources of calcium, enough must be consumed and absorbed to maintain normal blood levels. Therefore, an insufficient calcium intake places the bony framework in jeopardy.

This deficiency is particularly dangerous in growing children, adolescents and people over 45 years of age, since these groups need to gain calcium while maintaining their bone tissue.

Seventy percent of the bone mass is accumulated in the three to four year growth spurt during the teenage years, and additional bone is built up until around age 35.

When the blood level of calcium is low, the intestine will absorb more and the kidneys will eliminate less. If this does not provide an adequate amount, the skeletal storehouse will be called upon to supply more of the needed mineral.

## Hormone Regulation

Three hormones play an important role in this complicated biological process: 1) parathyroid hormone (PTH), 2) calcitonin, a peptide hormone and 3) hormonal Vitamin D. One medical expert called the effects of these three hormones “an exquisite control system.” As the blood calcium level falls, PTH is excreted by the parathyroid gland to speed up bone breakdown. If the blood level becomes too high, the amount of PTH will drop.

Calcitonin is a hormone produced by specialized cells within the thyroid gland, and it acts to conserve calcium in the bones by blocking the effects of PTH. Vitamin D is produced in skin tissue during exposure to the sun’s ultraviolet rays; then this “sunshine” vitamin must be chemically converted in the liver and kidneys into an active form. The end substance stimulates calcium absorption in the intestine.

The PTH secreted by the parathyroid glands also helps protect against low blood calcium levels by increasing the activation of Vitamin D by the kidneys. If there is little exposure to sunlight, a dietary deficiency, or kidney or liver disease, the resulting deficiency in Vitamin D causes abnormally low levels of magnesium and

phosphorus, and insufficient calcium for bone growth and maintenance.

In menopause, the kidney also loses some of its ability to convert Vitamin D into its active form. The price paid for maintaining a constant level of calcium in the blood is a loss in bone quality and strength.

## Remodeling

The hormones that are involved in the maintenance of calcium blood levels are also part of a chemical process that controls bone growth and repair. There is a constant tearing down and rebuilding of the skeleton that occurs in millions of sites within the bones.

In the late 1800’s, a German orthopedic surgeon named Julius Wolff formulated a theory of bone transformation now known to the world as Wolff’s Law: Bone accommodates the forces applied to it by altering its amount and distribution of mass. It refers to the process by which bone elements arrange themselves and grow larger or smaller in an orderly manner as they constantly tear down and rebuild, shaping new growth in response to stress.

This renewal process, called remodeling, enables bones to repair themselves and to release vital calcium into the bloodstream.

Bone deposit and resorption occur at the outside and the inside surfaces of the bone, called the periosteum and the endosteum respectively. Osteoblasts and osteoclasts are the cells responsible for the remodeling of bone. Osteoblasts are the cells responsible for bone deposit, and osteoclasts are responsible for resorption of bone.

If bones undergoing the remodeling resorption phase were magnified, their surface would look like pot-holed pavement after a hard winter. The osteoclasts are busily tearing down, the surface, creating craters for the busy osteoblasts to patch.

Osteoclasts are related to macrophage cells, and their precursors originate in the bone marrow and circulate through the blood. The osteoclasts migrate to specific bone sites and create an acid environment in which the calcium is dissolved. Like PacMan®, they chew away at the bone surface as enzymes dissolve the bone matrix of collagen and proteins. One large osteoclast can remove an area twice its size in 24 hours.

Researchers are now exploring the means by which the osteoclasts are able to establish the acid environment that is essential for bone erosion. The calcium dissolved in the process passes into the bloodstream.

Reformation of new tissue begins when osteoblasts migrate to the sites where bone was resorbed by the osteoclasts, and they go into action to produce collagen and begin the repair work.

Osteoblasts form a closely packed sheet on the surface of the bone, from which cellular processes extend through the developing bone. After they lay down the basic bone structure, the growing bone matrix engulfs the osteoblasts; as the material calcifies, the cells are trapped and can no longer lay down new bone.

They then develop into osteocytes, stable bone cells that regulate the finishing stages of bone production. This remodeling process continues throughout adult life. However, the amount of bone surface being remodeled at any one time is small, probably only about five percent. This turnover occurs at specific sites throughout trabecular bone, while cortical bone remodeling occurs only along its border with the marrow and near the Haversian canals that penetrate it.

The entire process from the initiation of resorption to the completion of repair takes about 100 days. Normally, the balance between the two determines whether bone tissue accumulates or disappears. With advancing age, the constant remodeling jeopardizes the skeleton as bone reformation lags behind bone loss.

Bones become fragile as a result of reduction in bone mass and density, and reduction in the effectiveness of trabecular cross-bracing. If there is a reduction in bone connectivity, and impact from a mechanical load occurs, then the bone will fracture.

Loss of bone connectivity occurs with aging; the loss is greater in women than in men, and is greater still in individuals with osteoporosis. Once this bone fatigue occurs, the osteoclasts begin a process called apoptosis, or programmed cell death, and the bones no longer are able to repair damage.

Matrix quality is also a factor, and a reduction in the degree of cross linkage is associated with a loss of bone strength.

Low serum copper and low calcium intake appear to be associated with poor cross-linkage within the bones.

There are several current clinical approaches that are used to enhance bone regeneration. These strategies include autologous bone grafts, free fibula vascularised grafts, allograft implantations, as well as the utilization of growth factors. While these regenerative approaches have shown satisfactory results, recent studies have revealed some limitations in efficacy and cost-effectiveness. This necessitates a current need for the development of alternative treatments of bone regeneration in patients with osteoporosis. New methods of studying bone regeneration using finite modeling at microscopic levels are currently underway.

Additional known causes of bone fragility are a high level of bone remodeling and factors relating to bone geometry. The hip axis length and hip angle affect risk of fracture independent of bone mass, and some data suggest that differences in hip axis length may help to explain the differences in fracture prevalence between Japanese and Caucasian populations. According to a report published in *Osteoporosis International Journal*, a recent cohort study investigated the fracture risk in middle-aged and elderly Japanese women. The results of this study revealed a three-fold increase in the risk of fracture in Japanese women with prevalent vertebral deformities.

Treatments to protect or increase bone mass will reduce fracture; non-mass factors, which also make an important contribution to skeletal strength and reduced susceptibility to bone fragility, need further investigation. According to Robert Heaney, M.D., a professor of medicine at the Osteoporosis Research Center at Creighton University School of Medicine in Omaha, Nebraska. "Imagine if you started remodeling your house: first you put an extension on one side, but before you finished that, you decided to tear out the garage, and before finishing that, you decided to put a deck on," says Heaney. "You'd have a pretty fragile house. That's what's happening with accelerated bone remodeling."

Now that they understand the importance of bone remodeling, osteoporosis experts are trying to use that knowledge to help predict osteoporosis risk factors. They're developing tools known as bio-

markers, which are chemical measures of the rate of bone remodeling that can be found in secretions from blood or urine. There are already biomarkers for the rate of bone remodeling that work very well in large population studies, says Heaney, but they do not yet have markers that work well in the doctor's office, on an individual patient level. Once more accurate biomarkers are developed, these and advanced imaging techniques may enormously improve our understanding of who is at greatest risk from osteoporosis. "This allows us to focus on where the problem really lies: the excess remodeling that's making bone fragile," Heaney says.

## Aging and The Skeletal System

Age disrupts the dynamic equilibrium between the skeleton and the rest of the body. As the body slowly and silently wastes away, tissue is lost from within, progressively thinning the bones' walls and framework. With advancing age, both cortical and trabecular bone are lost, and the rates are highest in women around the time of menopause. Women lose 30% to 50% of trabecular bone mass, and 25% to 30% of cortical bone mass; losses in men are on the order of 15% to 45% and 5% to 15% for trabecular and cortical bone, respectively.

Bone mass in adults is calculated by the peak bone mass by age 18 to 25 years minus the amount of bone that is later lost. This peak bone mass is determined by factors such as nutrition, genetics, endocrine status and physical activity. With osteoporosis, the natural loss of bone that accompanies aging is accelerated to abnormal, critical levels. The bones become weaker, thinner and more brittle with a marked increase in susceptibility to fracture. The skeletal framework can no longer withstand sudden or even normal mechanical stresses, and fractures occur.

Often, the first warning signal is a fractured wrist. Until the age of 45, this type of fracture occurs equally between the sexes. But after 45, ten times as many women as men break their wrists. In 1882, German surgeon Paul Bruns was the first to observe in print that fractured wrists occurred more often in older women, claiming they tripped over their long skirts. We know better today.

Recent studies have helped to shine some light in regards to the pathophysiol-

ogy of the aging skeleton. According to these clinical studies, a variety of intrinsic factors, such as peak bone mass accrual in youth, genetics and biochemical cues play an important role in the later fragility of the human skeleton.

Everyone begins losing bone mineral at around 40 years of age, but women who haven't been pregnant are at the greatest risk of suffering the fractures that are a major symptom of osteoporosis. Cortical bone is lost continuously and evenly by both sexes, but after menopause, women lose this tissue twice as fast as men. One expert stated, "Osteoporosis is not a disease like tuberculosis that the patient either has or does not have." Mayo Clinic studies show that one-half of all elderly women eventually suffer fractures due to osteoporosis.

Trabecular bone is destroyed quickly and by the age of 80, osteoporotic women have lost more than half of their trabecular bone tissue. The vertebrae are mainly composed of trabecular bone, and they can become compressed like so many crushed shredded wheat biscuits.

The vertebrae of the upper spine often collapse more in the front and become wedge shaped, bending the upper spine forward and causing the back to hunch over. When an entire vertebra is damaged, the "crush" fracture may be painful and take several months to heal. Rest, analgesics and "tincture of time" are usually the best treatments.

Some women may experience several essentially asymptomatic compression fractures, and only become concerned when they develop a hunched back and round shoulders, and notice dropping hemlines which emphasize their loss of height. This can be a dramatic change, sometimes three to eight inches. The osteoporotic vertebrae, particularly those in the lower back which bear the most weight, eventually shrink and collapse, sometimes because of the simple act of standing or sneezing.

The ordinary physical efforts required in everyday life - picking up a lightweight bag of groceries, bending over, climbing stairs, or just turning over in bed - can cause a compression fracture.

The collapse of multiple vertebrae over a period of time leads to shrinkage of the entire spinal column. As the spine shortens, it bends forward, shortening the chest and causing the lower ribs to rest upon the bones of the pelvis. Breathing

becomes difficult and many people complain of shortness of breath, with lung infections becoming a danger. The abdomen, also compressed, bulges and digestive problems are common. Over 1/3 of the women with collapsed vertebrae develop a curvature of the lower spine which causes pain and stiffness, making a tortured ordeal of many ordinary movements.

Deforming though they are, these vertebral fractures rarely threaten life. The most common cause of fatal fractures among the elderly is a fall from a standing height or less. At least 1/3 of the nation's elderly fall each year, and those aged 65 and up make up 3/4 of all fatal falls recorded.

According to the Centers for Disease Control and Prevention, falls are the leading cause of death in the elderly. Most deadly slips and tumbles happen indoors and only a surprising eight percent are caused by missteps in snow or on ice. A fall from a standing height in a person with normal bone density will usually provoke nothing more than a surprised look and maybe a few bruises. But in an individual with osteoporotic bones, a spill is likely to result in a fractured hip or back.

The broken bone itself is not as dangerous as the long period of inactivity required for healing, with the ever-present potential for complications such as pneumonia, pulmonary embolism, decubiti, urinary infection, depression and malnutrition. Many fractures result in a death that could have been avoided by a reduction in bone loss.

## The Role of Estrogen and Other Hormones in Bone Biology

Preserving a balance between bone resorption and reformation is the key to maintaining bone mass, with osteoporosis being the consequence of an imbalance between the two. Estrogen has been shown to have a role in the bone remodeling

## Hormones that Regulate Calcium Levels in the Blood

### Parathyroid Hormone (PTH)

- Secreted by parathyroid glands in the neck.
- Activated by low levels of blood calcium.
- Stimulates bone resorption - increases release of calcium from bones.
- Acts on kidneys to:
  - 1) reduce calcium loss in urine.
  - 2) convert Vitamin D to an active chemical form that stimulates absorption of calcium in the intestine.

### Calcitonin

- Produced by thyroid gland.
- Activated by high levels of blood calcium.
- Blocks bone resorption and reduces the release of calcium from bones.

### Vitamin D

- Is not just a vitamin since it is produced in skin cells during exposure to ultraviolet rays from the sun.
- Must be converted to its active chemical form by the liver and kidneys before it works.
- Stimulates calcium absorption from the intestine.

can thus result in calcium deficiency, which in turn affects bone health.

The thyroid hormones, thyroxine (T4), and triiodothyronine (T3), also influence the formation of bone as they can speed up remodeling. An overactive thyroid gland can cause bone loss as can taking too much thyroid medication.

Steroids are hormones produced by the testes, ovaries and adrenal glands. Some sex hormones help maintain bone and prevent osteoporosis by exerting a protective effect on the osteoblast cells; others in this complex group contribute to the disease process, apparently by weakening these cells and hastening cell death, and many of their functions are in opposition to each other.

Cortisone and the related glucocorticoids have been shown to cause osteoporosis, by mechanisms such as reducing calcium absorption, increasing calcium excretion, and limiting production of the gonadal hormones.

Estrogen, as described above, and progesterone block the resorption of calcium in the bones and have an effect upon the adrenal glands' production of the bone destroying hormones mentioned above. Estrogen also triggers the release of the bone-conserving hormone calcitonin, which helps form liver hormones that block the cortisone-like steroids from resorbing bone.

In addition, it prevents the action of PTH while interacting with the growth hormone, thyroid hormones and others. Testosterone, produced by the testicles and in lesser amounts by the adrenal glands, preserves bone and offers some protection to counter the loss of estrogen in women as well as in men.

process.

The cellular action of estrogen is mediated by estrogen receptors in both osteoclasts and osteoblasts. It acts through these receptors to regulate the process of apoptosis, or programmed cell death. Estrogen accelerates the death of osteoclasts, while prolonging the life of osteoblasts.

In postmenopausal women between the ages of 50 and 60, there is a decrease in estrogen levels; bone resorption increases and the formation process cannot keep up. Bone replacement with new tissue will be slowed and bone mass will be gradually decreased.

Estrogens also have striking positive effects on the intestinal absorption of calcium and the reabsorption of calcium from the renal tubule, thus promoting a positive calcium balance. Menopause and decreased estrogen levels are associated with intestinal resistance to Vitamin D. Reduced estrogen levels at menopause

## Risk Factors for Osteoporosis

There are still many unknown facts about osteoporosis, but certain risk factors now associated with it are helpful in identifying those who are most prone to developing the disease. In addition, since osteoporosis can be caused or exacerbated by certain diseases or drugs, appropriate steps can be taken to decrease the risk. Everyone has some risk of developing the disease - some more than others. Dr. Robert Heaney of Creighton University at Omaha, Nebraska, says, "Osteoporosis is a total life-style problem." You can't change your physical heredity, but you can change the way you live.

### Age

The longer you live, the greater your chances of developing osteoporosis. A recent study conducted at the University Clinical Center of Sarajevo focused on the aging process as one of the key factors for osteoporosis. During this investigation, 1000 female patients between the ages of 40 to 75 were examined for a one year period. The screening methods consisted of quantitative ultrasound (QUC) and densitometry (DXA). The results showed that signs of osteoporosis were present in 92% of the women. Moreover, menopause was the leading indicator, and was present in 890 participants. Other risk factors included genetics (22%), smoking (33%), and physical inactivity (31%).

The research is clear, osteoporosis is essentially a disease of aging, and all the physiological aspects of growing older contribute to its development. Sluggish, older intestines often don't absorb enough calcium to replenish the bones, and if an adequate amount isn't consumed to begin with, the problem is compounded. Inactive older people often remain inside and frequently do not get enough exercise to maintain bone strength and are not exposed to enough sunshine for the production of Vitamin D.

As the body ages, especially after menopause, there is a decline in the secretion of calcitonin, which discourages bone resorption. To compound the problem, there is an increased production of PTH (parathyroid hormone), which encourages the tearing down of bone tissue.

Some research indicates that the aging osteoblasts simply can't manufacture

enough new bone for the helpful osteocytes to keep up. As osteocytes die, there are fewer new ones to replace them, which has an adverse effect upon bone replacement. This form of osteoporosis is characterized by a thinning of the bones, which happens slowly over the years without any outward sign.

In many cases a bone fracture is the first clue that a woman has this condition. But by this time, osteoporosis has already caused irreversible damage. Loss of the spongy trabecular bone is paramount, and results in many crushing vertebral fractures, primarily in women between 55 and 75 years of age.

### Sex

Women with their smaller, thinner bones are far more susceptible to osteoporosis than men. Their peak bone mass is 30 percent less than that of men so there is simply less bone to lose. Women begin to give up bone earlier, and the reduction goes at a much more rapid pace than in men.

The calcitonin levels are lower in women so bone destruction is not as effectively controlled. In the early to mid 30's, bones stop growing and start weakening as they give up calcium to the blood.

Men lose bone at a constant rate, about 0.3 percent a year for the cortical bone; a slightly higher amount of trabecular bone is destroyed. Women lose about one percent of their trabecular bone and one percent of the cortical bone mass yearly, and that rate accelerates dramatically after menopause.

Three to seven years following menopause, the bone loss in women averages three percent. However, as much as eight percent can be lost from the lumbar vertebrae, which are composed mainly of trabecular bone. The formation of new bone can't keep up with the speed at which it's being torn down and there is a great deterioration of bone mass.

In approximately 20 years following menopause, the bones in a female skeleton may be reduced as much as 30 percent. All evidence points to the fact that over a woman's lifetime, estrogen protects bone mass while retarding the rate of loss. The longer and greater the exposure a woman has to estrogen, the lower her risk of osteoporosis. From puberty to menopause estrogen levels are high, and they rise during pregnancy or when oral contraceptives are used.

When production dramatically drops as with natural or surgical menopause, the sudden decline in estrogen level affects the complex relationship among other hormones and bone resorption and replacement.

Pre-menopausal, amenorrheic women are also at a greater risk for osteoporosis, because their hormone level is already low and menopause often begins early. The longer a woman lives after menopause, the greater the risk of osteoporosis.

### Race

Petite, fair skinned Caucasian and Asian women are at a much greater risk of developing the disease. In general, blacks have thicker bones and average ten percent more bone mass than others. Some researchers speculate that skin pigmentation and the higher ultraviolet levels in the tropics stimulate the production of Vitamin D in the skin, which influences bone tissue production.

Osteoporosis is much less prevalent among blacks worldwide, and in women of the Mediterranean regions. While African American women tend to have higher bone mineral density (BMD) than white women throughout life, they are still at significant risk of developing osteoporosis.

The misperception that osteoporosis is only a concern for white women can delay prevention and treatment in African American women who do not believe they are at risk for the disease.

Many scientific studies highlight the risk that African American women face with regard to developing osteoporosis and fracture. From this research, the medical community has learned the following facts:

- Osteoporosis is under recognized and under treated in African American women.
- As African American women age, their risk for hip fracture doubles approximately every 7 years.
- African American women are more likely than white women to die following a hip fracture.
- Diseases more prevalent in the African American population, such as sickle-cell anemia and lupus, can increase the risk of developing osteoporosis.
- African American women consume 50 percent less calcium than the Recommended Dietary Allowance. Adequate intake of calcium plays a

crucial role in building bone mass and preventing bone loss.

As many as 75 percent of all African Americans are lactose intolerant. Lactose intolerance can hinder optimal calcium intake. People with lactose intolerance often may avoid milk and other dairy products that are excellent sources of calcium because they have trouble digesting lactose, the primary sugar in milk.

A recent research symposium on musculoskeletal healthcare disparities studied the current literature concerning racial differences in osteoporosis related to the frequency of fractures.

The results of this review revealed that race and ethnicity influence the epidemiology of fractures. The results clearly indicate that Caucasian women experience the highest rate of fractures; however, African American women are more likely to die after a hip fracture, and typically have longer hospital stays related to fractures.

The research shows that African American women have higher bone mass density, which relates to the lower risk of fractures. However, the higher mortality rate in African American women is still not completely understood.

Current prevention efforts and screening should be targeted toward all women, especially those who have suffered multiple fractures.

### **Family History**

Take a good look at your mother and grandmother to see what's in store for you. Women with a family history of osteoporosis are much more likely to develop this disease. Potential bone mass is a genetically determined factor. A tendency toward early menopause may also be inherited, bringing additional years for the bones to go without estrogen protection. Bone mineral density (BMD) is a predictor of fracture, but association studies with candidate genes, including those for vitamin D, estrogen, and androgen receptors, have produced inconsistent and contradictory results to date. BMD is too crude a measure to detect variations in remodeling due to genetic differences.

Some promising research has indicated, however, that a particular polymorphism of the androgen receptor may serve as a molecular marker of risk for osteoporosis in men.

Tremendous progress has been made regarding genetics relevance to the pathogenesis of osteoporosis. Recent twin and family studies have revealed that between 50% to 85% of BMD variance is genetically determined. Other studies have shown that age-related bone loss can also be inheritable. Despite this progress, genetics have yet to become a key diagnostic tool. Future research regarding genome-wide sequencing may help in understanding the complete scope that genetics plays in osteoporosis.

### **Lifestyle Factors**

In addition to the risk factors listed above, there are a variety of lifestyle characteristics that have also been shown to increase the risk for development of osteoporosis and subsequent bone fracture.

These include nutritional status, smoking, excessive alcohol use, exercise and general activity level. Because modification of these factors is one of the most important strategies for osteoporosis treatment and prevention, they will be covered in depth in those sections of this course.

### **Alcohol, Tobacco and Caffeine**

There is universal agreement that alcohol is a strong contributing factor to osteoporosis, but it's not known "how much is too much." A recent study of men who drank socially, one to two drinks per day, showed that they had a two-fold increase in risk of osteoporosis. It is known that alcoholism is the most common bone destroying disease in men under 60.

Even newer research has shown that tobacco and alcohol consumption can have a negative clinical impact on bone mineral density (BMD) in men.

Alcohol decreases the absorption of calcium in the intestine and has a negative effect upon the liver, which must help convert Vitamin D to its active form. Alcoholism is a notable cause of magnesium depletion that can result in concurrent hypocalcemia (low blood calcium levels). The exact mechanism is not yet defined, but this mineral deficiency will cause the blocking of calcium from the bones and also affect the secretion of PTH, which plays a role in bone building.

Cigarette smoking carries an increased risk of osteoporosis. Studies have shown that it results in lower estrogen levels, with menopause usually occurring about

two years earlier in smokers. Smokers lose bone at a greater rate during the first years after menopause, and smoking is associated with a 40% to 50% increase in hip fracture. Nicotine constricts the blood vessels and this may impair bone nourishment as well as causing other diseases that affect bone health.

Add about five cups of coffee a day to those cigarettes and the risk of brittle bones is increased even more. Nutritionists tell us that caffeine encourages waste of calcium, with less being absorbed by the bones.

### **Exercise**

Defining the role of physical activity in both the development and maintenance of bone health throughout the life cycle has taken on increased importance. As a key modifiable factor, exercise is important in both the attainment of peak bone mass and in the reduction of bone loss in later life. In fact, in the absence of weight bearing exercise, bone loss will occur at any age, at both axial (e.g., vertebrae) and appendicular (e.g., wrist and hip) skeletal sites.

In a recent study examining bone loss in Russian cosmonauts, loss at the weight-bearing tibial site occurred within the first month in space and worsened with mission duration. In those cosmonauts that spent six months in space, losses ranged up to 23%.

Exercise regimens for post-menopausal women have shown a clear benefit on total bone mass, total bone density, and lean muscle area. Participants also showed significant improvements in psychological well being as well as functional fitness.

It appears that, while normal levels of activity are sufficient to maintain bone health in pre-menopausal women, women who are estrogen depleted require greater amounts of mechanical force to preserve existing bone. It is encouraging to note, however, that low-strain physical exercise and rehabilitation programs can be beneficial to even very old women (aged 85 or older.) These exercise programs have the added benefit of improving overall muscle strength, which leads in turn to an improvement in balance and posture in the elderly population, thus lessening the risk of falls and fractures.

Regular exercise that puts mechanical stress on the bones increases their strength and retards loss in later years. The best

exercises are those that require the spine and long bones to bear weight. Doctors at the Mayo Clinic studied a group of 68 postmenopausal women and proved a correlation between the strength of the extensor muscles of the back and the bone health of the vertebrae to which they were attached.

To quote Dr. Mersheed Sinaki, "It seems that the muscles that are directly attached to the bones play the biggest role." She is now trying to determine whether strengthening back muscles can stop or reverse the loss of calcium from the vertebrae.

Dr. Sinake points out that specific exercises are the most beneficial and recommends the extension exercises of arching the back and then straightening it. Women in this study who did the exercises showed 1/3 the spinal fractures of those doing no exercise or only bending ones. NIH has recommended three to four hours weekly of weight bearing exercise such as walking or jogging to increase bone density in those under 30 and slow bone loss in those who are older.

According to the National Osteoporosis Foundation, studies have shown that while resistance training can strengthen bones, other regular exercise programs can help to reduce the likelihood of falls. These programs include routines, such as tai chi that help focus on balance.

One word of caution, however: There is increasing concern for the bone health of women who engage in high-intensity physical training, for whom amenorrhea is a common condition. Many competitive women's sports also require extremely low body weights, and this combination can be very detrimental to bone health. Also, if bones are already weakened, any exercise program should be started with care and medical advice.

## Fitness Vibration

One of the first studies of osteoporosis and whole-body vibration was published by the Journal of Bone and Mineral Research. During this study, Belgian scientists divided 70 postmenopausal women into three groups: Some did vibration; some lifted weights; and some weren't asked to do anything at all. Six months later, the only women who saw an increase in hipbone density (nearly 1 percent) were those using whole-body vibration.

Called a Power Plate (there are several other brands, too), the machine alternately vibrates side to side, up and down, and

back and forth, sending a vibration through the user's body from toes to tear ducts. An additional perk: Women in both the vibration and weight-lifting groups lost fat and upped muscle strength.

So why aren't doctors telling every postmenopausal woman to get shaking? "I think this study should help advance the field," says Thomas A. Einhorn, spokesperson for the National Osteoporosis Foundation and chief of orthopedic surgery at Boston Medical Center. "But I don't think whole-body vibration is ready for prime time. There are still too many questions." Such as, whether standing on a vibrating platform is really safe for someone with fragile muscles and bones. The fact is, too much vibration can be dangerous even for people with healthy spines. People in jittery jobs, like truck drivers and heavy-machinery operators, are very prone to back pain and circulatory disorders.

A more recent study published in the Journal of Men's Health investigated the effects of vibration exercise on the human skeleton. During the study, 24 volunteers, ages 46 to 56, were randomly assigned whole body vibration and resistance exercise four times weekly for 16 weeks, while the control group did not participate in any training.

At the beginning and conclusion of the study, hip bone density was measured using DEXA. The results showed a significant increase in hip BMD as well as improved leg strength. Researchers are still looking for clues regarding the potential for harm to the joints from whole-body vibration.

## Health Problems Contributing to Osteoporosis

One out of three men, and one out of five women, who sought medical care for osteoporosis (which was first manifested as a fracture), were discovered to have an underlying illness that contributed to the development of the disease. Diagnosis and treatment of the underlying ailment is essential for appropriate care. Medical conditions that may contribute to the development of osteoporosis include:

**Hyperthyroidism** (excessive amounts of thyroid hormone): The disease may be due to an overactive thyroid gland or may be the result of taking too high a dosage

or too lengthy a regimen of the thyroid hormone in tablet form. The longer it continues, the more severe the bone damage. This condition escalates bone loss and after several years, can cause osteoporosis.

**Hyperparathyroidism:** Hyperactivity of the parathyroid gland causes such a degree of excess to be secreted that it keeps a persistently high level of blood calcium. There is often wasting of bone tissue, as well as abdominal pain, mental dysfunction, and development of kidney stones. Sometimes called the "Hungry Bone Syndrome," this disorder is more common than realized, and mild forms are often discovered in routine lab tests. Since it may not cause symptoms, the silent bone loss in postmenopausal women can be severe. The only treatment is surgical removal of the parathyroid glands.

**Cushing's Syndrome:** This is a disorder in which an overactive pituitary gland stimulates the adrenal glands to produce too much cortisone. One of the effects is osteoporosis. A 'buffalo hump' develops between the shoulder blades due to a fat pad, and often the spine is bent forward by deformities caused by compressed vertebrae.

**Rheumatoid Arthritis:** Rheumatoid arthritis is often characterized by a bone calcium content that is 10 percent lower than normal. However, there are thick calcium deposits along the edges of bone and around the joints. Some believe the decrease in bone calcium results from the decreased mobility associated with the disease; however, a similar decrease is not seen in patients with osteoarthritis, which can also cause significant reductions in mobility.

Cortical bone in most osteoarthritic patients is tough and hard, while that of an osteoporotic patient is often honeycomb. Those who suffer from rheumatoid arthritis have severe pain, decreased physical activity, and poor appetite and nutrition. Treatment with cortisone aggravates calcium loss and bone weakness. Anti-inflammatory medications often must be taken with antacids, which, in turn, block calcium absorption.

**Chronic Obstructive Pulmonary Disease (COPD):** Over the last few years studies have revealed evidence that inhaled corticosteroids can increase the risk of bone fractures. Studies show that people with emphysema or COPD who took high doses of inhaled corticosteroids

to treat their disease have more fractures. In the United States, corticosteroids are prescribed to over one million patients a year. They are also one of the most common causes of secondary osteoporosis. Research suggests that there can be a 10% loss of BMD annually; however, the rate typically declines after the first year.

For decades, it's been known that steroids mimic the naturally produced hormone, cortisol, which helps regulate blood sugar and metabolism. The hormone accelerates bone loss by preventing calcium absorption in the gut and increasing calcium loss in the urine. The drugs can even damage cells that help build bone. Whether taking high doses of inhaled steroids—the equivalent of 700 micrograms per day—were at increase risk for fracture. Bottom line practitioners should be closely monitoring a patient's bone density, with a baseline BEFORE therapy begins.

**Cancer:** Cancerous plasma cells produce a chemical osteoclast activating factor, OAF, which is a powerful stimulator of the production of bone-dissolving osteoclasts. Patients with multiple myeloma often have severe osteoporosis, and several other types of carcinoma show a particular affinity for bones. Chemotherapy for the treatment of breast cancer has been shown to decrease estrogen production by the ovaries, resulting in greater than expected bone loss in treated women.

**Hypertension:** A study of risk factors for bone loss and related fractures among 3500 elderly women suggested that high blood pressure that is not adequately controlled may increase the risk of osteoporosis. The study, reported in *Lancet*, suggests that high blood pressure may be associated with abnormal calcium metabolism and bone loss. Recent research reported in the *Journal of Human Hypertension* also showed the inverse relationship between sodium intake and hypertension regarding their effect on osteoporosis. This study shows that higher amounts of dietary sodium, increases urinary sodium and calcium extraction. Researchers advise that this plays a direct role on increased bone loss and osteoporosis.

## Emotional Issues

**Myth:** Osteoporosis Doesn't Cause Any Emotional Problems.

**Reality:** The emotional toll of osteoporosis is very real. Having a fracture, or

even seeing yourself as more “fragile” than you once thought you were, can lead to a negative body image, poor self-esteem, and a sense of limitations in activity and mobility.

Kyphosis, the “dowager’s hump” that results from vertebral fractures, has been associated with significant depression in people with osteoporosis. After one fracture, many people with osteoporosis are so fearful that they may fall and injure themselves again that they don't pursue the activities they enjoy.

**Depression:** In a review of published research, NIMH-funded scientists reported a strong association between depression and osteoporosis. The literature suggests that depression may be a significant risk factor for osteoporosis. Low bone mineral density (BMD), a major risk factor for fracture, is more common in depressed people than in the general population. Although its causes are unclear, major depression is associated with hormonal abnormalities that can lead to changes in tissue, such as bone. Research suggests that higher cortisol levels, often found in depressed patients, may contribute to bone loss and changes in body composition.

In one study, evidence revealed that bone density at the lumbar spine was 15% lower in 80 men and women older than 40 with major depression compared to 57 men and women who were not depressed. Factors such as smoking, a history of excessive or inadequate exercise, or estrogen treatment did not affect the study, implying that depression per se had an effect on bone mass.

The association between depression, BMD, falls, and risk of fracture was also examined in a study of 7,414 elderly women. Depression prevalence was 6%. Depressed women were more likely to fall (70% versus 59%) and had more vertebral (11% versus 5%) and non-vertebral (28% versus 21%) fractures compared with controls. This research underlines depression as a risk factor for osteoporotic fractures, and its identification would improve diagnosis and treatment. When one or more other risk factors is present, such as low BMD, family history, previous fracture, thinness, or smoking, a clinical evaluation for osteoporosis is recommended.

Another factor to consider regarding depressed patients is the effect of antidepressants on fractures and bone mass. One large cohort study investigated elderly

women, who were treated with serotonin reuptake inhibitors (SSRIs). The results of this study determined that these women experienced greater bone loss at the hip. In another study, tricyclic antidepressants (TCAs) with sedating effects were also associated with fractures. However, in this same study, TCAs without sedating effects showed no increase of fractures.

One way to cope with depression and other psychological problems related to osteoporosis is to find support from other people going through the same thing.

The National Osteoporosis Foundation sponsors local support groups called Building Strength Together. Anyone can start one in their community. You can find one, join one, or start one on their web site at <http://www.nof.org/connect/community-groups/support-groups>. Another important coping tool for depression is exercise.

Regular exercise is proven to boost self-esteem and relieve anxiety, stress, and depression. Exercise also helps maintain bone health. If you've had a fracture, it's important to talk to your doctor about what kinds of exercise are safe for you. With your doctor's advice, you should be able to pursue activities that will keep your body strong and help prevent depression at the same time.

**Other Diseases:** Patients with pancreatitis, severe liver disease, and emphysema often suffer from osteoporosis; since 1948, diabetes has also been recognized as a risk factor. Some researchers believe there is a common genetic risk factor for both diabetes and osteoporosis. A gastrectomy often results in poor absorption of calcium, but supplemental Vitamin D and calcium usually resolve the problem. Those with an untreated GI malabsorption syndrome or pancreatitis may develop magnesium deficiency, which results in lower blood levels of calcium and leaching of calcium from the bones.

## Be Alert to Medications

Many medications contribute to osteoporosis when they are prescribed for other conditions. In the U.S., the population over 65 years of age takes 25 percent of all medications, some of which might have begun early in life to deplete the bones of calcium. The following medications have an adverse effect upon bone health:

**Glucocorticoids:** Long-term glucocorticoid use is the most frequent cause of drug-induced osteoporosis, and the third leading cause of all osteoporosis in adults. Approximately 90% of long-term users may lose significant amounts of bone, resulting in an increased risk for fracture.

Although the mechanism of this effect has not been finally determined, it is believed that glucocorticoids accelerate bone loss in several ways. Decreased bone formation has been demonstrated, with an increase in osteoblast and osteocyte apoptosis.

Glucocorticoid use has also been associated with decreased absorption of calcium in the intestine, and lowered activity of the sex hormones. Finally, long-term uses may also contribute to muscle atrophy and progressive loss of muscle strength. All of these factors affect bone formation and may thus over time increase fracture risk. Risk appears to be greatest for fractures of trabecular bone.

Oral forms of these steroids include: Aristocort®, Celestone®, Deltasone®, Decadron®, Medrol®, Prednisone®, Hydrocortisone®, Cortef® and Cortisone Acetate®.

A marked decrease in bone mineral density is typically observed within a few weeks of initiation of treatment in adults, but bone loss will continue even after many years of use. Particular concerns have been raised by the increase in both long-term and -intermittent use of glucocorticoids for the treatment of a variety of childhood inflammatory diseases. Current research focuses on the relative effects of inhaled versus oral glucocorticoid use, and the possible benefits of prophylaxis for prevention of fractures.

**Antacids:** Those that contain aluminum interfere with calcium metabolism; these include: ALternaGEL®, Aludrox®, Amphojel®, Basaljel®, Camalox®, Delcid®, Di-gel®, Gaviscon®, Gelusil®, Kolan-tyl®, Maalox®, Mylanta®, Riopan®, Rolaid®, Silain Gel® and Simeco®.

The following antacids and medications for ulcer symptoms do not contain aluminum and are thus safer for long term use:

Alka Seltzer®, Alkets®, BiSodol®, Citrocarbonate®, Lo-Sal®, Mylicon®, Titalac®, Tums®, Tagamet® and Zantac®.

**Diuretics:** These medications increase calcium loss in the urine, and the thiazides are the most commonly prescribed:

Diuril®, Dyazide®, Corzide®, Diamox®, HydroDIURIL®, Aldoril®, Aldoclor®, Hydropres®, Aldactazide®, Apresazide®, Aldoclor®, Hydropres®, Naturetin®. Lasix® has the same effect.

**Tetracyclines:** These drugs bind to calcium and impair its absorption. The medications should be taken two to three hours apart from calcium supplements or calcium rich food. Individuals who take these medications often have high levels of calcium in their urine, but long lasting harm has not been proven.

**Anticonvulsants:** Dilantin, phenobarbital, Primidone® and Phensuximide® all may interfere with Vitamin D metabolism in the liver and decrease calcium absorption. Anyone on these medications may need calcium and Vitamin D supplements.

**Proton Pump Inhibitors (PPIs):** These drugs are used to treat gastroesophageal reflux disease, and can increase bone loss at higher doses. Examples include Prilosec®, Prevacid®, and Nexium®.

**Miscellaneous Medications:** Some of these drugs adversely affect bone health by encouraging the withdrawal of calcium from the bones or by interfering with Vitamin D production:

- antituberculars including Isoniazid®, Seromycin®, Capastat®
- anticancer agents such as Dactinomycin (Cosmegen)
- Questran® to lower cholesterol levels
- Benemid® and ColBENEMID® for gout
- Doriden® and lithium
- Accutane® mobilizes calcium and supplements should be taken with long-term use.

**Long-acting benzodiazepines and sedatives** have also been implicated in the development of osteoporosis. A recent international literature review investigated the potential impact of benzodiazepines on the rate of hip fractures in osteoporosis patients in five different studies. The data analysis showed that the groups that were at the highest risk of hip fracture were those who were most exposed to benzodiazepine use. It would be wise for any patient to consult the prescribing physician as to the effect of his or her medications upon calcium nutrition, because many helpful and frequently prescribed medications can have harmful side effects on bone health.

## Diagnosis of Osteoporosis

In osteoporosis, two factors are important: the amount of tissue inside the bone itself and the quality and health of the tissue. Ordinary X-rays can show fractures typical of osteoporosis, but they are not helpful in determining the amount of bone mass.

A person would have to lose as much as 40 percent of his bone calcium before it would become obvious on an X-ray. Therefore, techniques have been developed to measure the bone mass in the arms, legs and spine. They are helpful both in determining the degree of damage and in monitoring treatment response.

### Dual Energy X-ray Absorptiometry - DEXA

At the present time, the clinical diagnosis of osteoporosis is made by measurement of bone mineral density (BMD) with a technique considered the “gold standard” called the DEXA scan (dual energy X-ray absorptiometry). A very tiny beam of particles emitted by a radioactive isotope of iodine is projected through the bones of the wrist, hip or spine. These are the most common locations for bone fractures.

An instrument called an absorptiometer measures the number of particles that can be projected through the bone. The technology uses miniscule doses of radiation, approximately one-fifth that received from a standard chest x-ray for example, and provides fast, accurate, painless and completely reproducible results.

BMD as measured by this technique is expressed as an absolute value and may be designated as either the number of standard deviations from the mean of age matched controls (Z score), or the number of standard deviations from the young normal mean (T score). The World Health Organization currently defines osteoporosis as a BMD of 2.5 or greater standard deviations below the mean value for young adults. Efforts are currently underway to develop a more flexible set of diagnostic criteria, which would also include other measures of risk such as bone turnover markers or evidence of a previous fracture.

### Other Bone Densitometry Tools

Although DEXA is the most widely used method for measuring BMD, there

is additional technology available for osteoporosis patients:

- **Quantitative Ultrasound Densitometry (QUS)** – This measures the speed of sound and broadband ultrasound attenuation at the tibia, patella, heel and other skeletal sites.  
An example is a device, called the Sahara Clinical Bone Sonometer, which transmits sound waves through the heel of the foot to measure bone density. The sonometer is a portable, inexpensive device that may make screening more accessible. Although it is accurate enough for screening, it's not currently as sensitive a diagnostic tool as DEXA.
- **Quantitative Computed Tomography (QCT)** – This technology measures volumetric trabecular and cortical bone density at the hip, spine and bone structure. QCT is used to predict vertebral fractures in postmenopausal women. Peripheral QCT (pQCT) measures bone structure at tibia or forearm, and is used to predict hip fractures. The drawback of these CT-based scans is that they are associated with more radiation exposure than DEXA.
- **Peripheral Dual Energy X-ray Absorptiometry (pDEXA)** – This test measures the BMD of the finger, forearm or heel to predict vertebral fracture risk in postmenopausal women.

## Laboratory Testing

During the clinical evaluation of patients of patients with osteoporosis, laboratory studies may include the following:

- Complete blood count
- Thyroid-stimulating hormone (TSH)
- Chemistry levels - renal function, calcium, magnesium and phosphorus
- Parathyroid hormone (PTH)
- Liver function
- 24 hour urinary calcium
- Urinary free cortisol level

## Biochemical Markers

The use of biochemical markers of bone turnover in the management of osteoporosis raises considerable interest. A battery of biochemical markers are now available that allow for assessment of the rate of bone formation and bone resorption of the skeleton.

These markers appear to be useful for the individual monitoring of osteoporotic

patients treated with antiresorptive drugs, especially for those agents that induce only a small increase in BMD. Such markers can also be useful in selected cases to improve the assessment of the individual risk of fractures when BMD measurement by itself does not provide a clear answer.

Prospective studies have shown that urinary or serum levels of markers above the premenopausal range are associated with a 2-fold increase in the risk of fragility fractures, independent of the level of BMD. The combined use of BMD and bone markers is likely to improve the assessment of the risk of fractures in those cases.

A history of a low-trauma fracture, or a fracture sustained after the age of 50, is one of the strongest and most consistent risk factors for subsequent fracture; for hip and vertebral fractures, for example, the increased risk is independent of BMD. A history of fracture should be included in clinical guidelines to identify patients in whom a BMD test should be performed and for whom treatments are most likely to be beneficial. Without proper follow up, fractures represent a missed opportunity for appropriate intervention.

## Fracture Risk Algorithm (FRAX®)

This osteoporosis assessment tool was created to assess the 10-year probability of a hip fracture as well as the 10-year likelihood of a vertebral, forearm or proximal humerus fracture. The FRAX® algorithm calculates certain clinical risk factors along with femoral neck BMD. The tool can be found at [www.shef.ac.uk/FRAX](http://www.shef.ac.uk/FRAX). According to the National Osteoporosis Foundation, FRAX® is aimed toward postmenopausal women and men over 50; however, it has been validated for use in women and men between the ages of 40 to 90. Moreover, FRAX® is intended to calculate BMD from the femoral neck or total hip area, and is not suited to measure BMD in other areas.

## Treatment and Prevention Strategies

The best treatment for osteoporosis is prevention. Once a fracture has occurred as a result of osteoporosis, it is effectively too late to implement the most effective treatment strategies. While some improvement in bone mass and quality has been

demonstrated with aggressive treatment, the focus must be primarily on stopping or slowing any further bone loss. As noted above, alteration of lifestyle and patient education with regard to healthier choices are key to both prevention and successful treatment.

## Hormone Replacement Therapy

Hormone replacement therapy (HRT) has been used for many years in the treatment of osteoporosis but, despite its popularity, its efficacy in prevention of fractures is based more on opinion and anecdote than on clear scientific evidence. Ultimately, the decision regarding use of HRT is one that must be made by each woman and her healthcare provider, based on her own circumstances and assessment of the relative risks and benefits to her overall health. The FDA recommends that HRT be used in the lowest effective doses for the shortest period of time due to risks of breast cancer, stroke and myocardial infarction.

## Nutrition and Nutritional Supplements

Adequate nutrition influences all aspects of bone health throughout the life cycle, from the development of strong bones from childhood into early adulthood, to the maintenance of bone mass in adults, to the reduction of bone loss and fracture in the elderly. When it comes to your bones, you are what you eat and drink.

**Calcium:** Most Americans consume well below the US Recommended Dietary Allowance (RDA) of 1000 to 1500 mg of calcium per day, often because they avoid calcium-rich dairy products that they feel are too fattening.

One expert estimates that teenaged girls probably consume less than one-half of the RDA for their age group. Adult women often continue the foolish practice of shunning dairy foods that started in their teens.

During this period, they should be building bone mass to its peak levels but instead, they enter menopause far below their genetic potential, making their age and sex-related bone loss even more devastating. Bulimic and anorexic women compound the problem with their self-destructive habits, and their poor nutritional state and resulting amenorrhea take an early toll on their skeletons.

Little attention was paid to the calcium problem until recently. Two thirds of young American women aged 18 to 35, and 75% of the women over age 35, are consuming less than 800 mg of calcium per day.

<b>Daily Calcium Recommended:</b>	
<b>Women</b>	
• 14 - 18 y/o, Pregnant & lactating	1,300 mg
• 19 - 50 y/o, Pregnant & lactating	1,000 mg
• 51 + y/o	1,200 mg
<b>Men</b>	
• 50 - 70 y/o	1,000 mg
• 71 + y/o	1,200 mg

*Figure 1*

Calcium deficiency is common - more common than iron deficiency anemia. This can only lead to tomorrow's common disease - osteoporosis. See *Figure 1* for The National Institute Medicine current recommendation on calcium intake.

Recent studies have shown that those who have had a high lifelong daily intake of 1000 mg or more of calcium have a lower incidence of fractures than those who have taken less have.

According to the national Osteoporosis Foundation, calcium intakes over 1,500 mg per day have limited potential and may cause adverse health conditions. Four 8 oz glasses of milk will provide 1000 mg of calcium but, for many, supplements may be necessary. The average American diet without milk only provides 250 to 300 mg a day.

The effectiveness of calcium supplements in reducing peri-/post-menopausal bone loss remains controversial, perhaps because many of the research studies addressing this question failed to take into account the effects of estrogen withdrawal occurring at the same time.

It appears that supplementation is most effective in reducing bone loss in women who are more than five years post-menopause, and in those who have long-term calcium intakes of less than 400 mg per day. Calcium supplementation appears to improve bone mass density, but the effect on reduction of fractures is currently less clear.

## Phosphorus – Calcium

### Balance:

The optimal ratio for building bones is one part calcium to one part phosphorus. Many people consume an excess of phosphorus because it occurs naturally in foods and is added to many processed foods, breads and drinks. Estimates of the individual daily consumption of phosphorus from these sources vary from 400 to 1800 mg. Laboratory animals when fed a diet high in phosphorus but low in calcium developed secondary hyperparathyroidism, which is associated with a loss of calcium and demineralization of bone. Phosphorus supplements are usually not recommended as there is an adequate amount in the diet.

### Calcium Supplements:

Since the dangers of osteoporosis were first publicized in the 1980s, the sales of calcium supplements have skyrocketed despite the lack of solid scientific support. So many drug companies have jumped on the bandwagon that it's impossible to open a women's magazine without confronting many full-page ads for calcium supplements.

Packagers of calcium-enriched foods have joined the pharmaceutical companies, playing upon the fears of the public with advertisements of shrunken, little old ladies with bent spines. The sales of supplements have surged from \$18 million to over \$240 million annually; one distributor reported that sales of calcium have increased 40 percent, and are now his top selling product.

Consumers are confronted by a bewildering array of pills, tablets, liquids and powders loading the shelves of supermarkets, pharmacies and health food stores. Trying to pick the best brand and decide which dose is right could confound Einstein! For many, there is no right brand, only the right amount. For some, however, the type of calcium in the supplement does matter.

A key characteristic of calcium is its solubility. Certain forms dissolve readily in water - something the body has plenty of. But insoluble calcium supplements require hydrochloric acid to dissolve before they can be absorbed.

Elderly people frequently do not secrete a normal amount of acid. One gastroenterologist estimated that this is occurring in about 10 percent of the people over age

60 with the rate climbing to 30 percent among those 70 and over. People who have been treated for ulcers often have lower levels of gastric acid. Taking calcium supplements with food helps eliminate this problem, but experts advise anyone with impaired gastric acid secretion to use only soluble forms of calcium.

Label readers know that calcium carbonate is the most common calcium supplement. They might recognize it in the familiar tablet of TUMS® or CHOOZ®. Yes, TUMS® spells relief - not only for acid indigestion but also for brittle bones!

The only important difference between these antacids and the calcium carbonate supplement is the amount of calcium per tablet.

**Calcium carbonate** is usually the most inexpensive, concentrated source of calcium, containing 40 percent by weight. Sixteen TUMS® contain 800 mg calcium carbonate, of which 320 mg is pure calcium.

It is available in a wide range of dosages, 140-600 mg per tablet, powder, gum coated, flavored or unflavored. Ironically, the one drawback is acid rebound: its neutralization of stomach acid later results in increased secretion. Taking calcium carbonate with meals eliminates this problem.

Calcium carbonate has few other side effects and it enjoys the largest share of the market. The supplement is insoluble and requires hydrochloric acid to dissolve so it should be taken with food.

**Calcium citrate** may be somewhat more easily absorbed than the carbonate, however, available preparations tend to be more expensive, and they contain relatively less calcium per dose.

There are three forms of **calcium phosphate: mono, di and triphosphate**. Only the first offers any degree of solubility and it is not usually in the supplements.

Calcium phosphate is 23 to 39 percent calcium and rivals the carbonate compound as a source of the concentrated mineral.

There are fewer complaints of gastric distress, and it is well suited as a calcium-fortifying food additive. Because Americans consume many phosphorus-containing colas and their diet is meat heavy, however, the average woman can easily consume four times as much phosphorus as calcium.

**Chelates** are one of the most expensive forms of calcium. They are basic supple-

ments binding calcium to an amino acid, the building block of protein, rather than to a carbonate or phosphate. Manufacturers make unsubstantiated claims that this form is the most readily absorbed, but solubility varies as does the calcium content.

**Calcium chloride** is available in granulated form and is not recommended because of the gastric irritation it causes. Its main use is in pickling foods and as a salt replacement for those who must avoid the most common salt substitute, potassium chloride.

**Calcium lactate and calcium gluconate** are good choices for those who have an acid secretion problem, and they do not cause GI irritation or constipation. Their only drawback is a low calcium concentration: 13 percent in lactate and only 9 percent in the gluconate form.

To receive an adequate amount of calcium, a great number of tablets would have to be taken, since most contain less than 100 mg each. Powders of calcium gluconate, lactate, phosphate, or carbonate can be obtained in one-pound sizes. However, getting an adequate and accurate dose can be a problem. The cost is generally low, and the powder can be ordered by mail or from a health food store.

A liquid, similar to calcium gluconate, called Neo-Calglucon, is available at pharmacies without a prescription. About two tablespoons contains 600 mg of calcium. This is an expensive way to get your daily calcium. The most important information on the label is the amount of pure calcium.

Many companies market calcium bound with another chemical, and not all are pure calcium. Some zealots become so enthusiastic that they are likely to consume far more than is beneficial.

The NIH has stated that too much calcium can cause kidney stones in susceptible people who absorb calcium to abnormally high levels. This idiopathic hypercalciuria occurs when the kidneys try to excrete the calcium through the urine.

Recent randomized studies have reported the potential risks associated with calcium supplementation. One randomized trial tested 1471 postmenopausal women, who were randomized to calcium or a placebo. The results indicated that myocardial infarction was more prevalent in the calcium group than in the placebo group.

**Vitamin D:** During the recent National Institutes of Health Conference, extensive documentation was presented, which revealed that calcium along with Vitamin D supplementation can increase BMD, and decrease the risk of bone fractures.

Vitamin D plays a crucial role in the regulation of calcium and phosphorus metabolism and promotes calcium absorption from the gut and kidney tubules. The National Osteoporosis Foundation recommends a daily intake of 800 to 1,000 international units of vitamin D for adults age 50 and older. Vitamin D deficiency and insufficiency are important nutritional factors that require attention in all population groups, but especially in the elderly.

The effects of the aging process on Vitamin D status is well documented and both the institutionalized and free-living elderly are at greater risk for deficiency due to their decreased activity level and exposure to the sun. Supplementation with Vitamin D has been shown to improve calcium absorption, lower PTH levels, and reduce wintertime bone loss in post-menopausal women.

A combination of Vitamin D and calcium supplementation has been shown to reduce fracture rates, but Vitamin D alone did not appear to be as effective. Interestingly, a combination of Vitamin D and calcium supplementation has been shown to reduce the frequency of falls, as well as indices of body sway and blood pressure. This effect on factors associated with falling, as well as falling itself has great potential for decreasing fracture risk.

**Protein:** Insufficient intakes of dietary protein have been implicated in the development of osteoporosis, and protein supplementation has been shown to improve the clinical outcomes of hip fractures. Lack of protein affects sex hormone status and synthesis of key growth factors.

Very low protein intake has been associated with a decrease in bone formation and an increase in bone turnover, along with decreases in bone mass and overall bone strength.

Diets high in animal protein and poor in vegetable intake, however, are also implicated in the development of osteoporosis. Both animal protein and cereals are rich sources of phosphoric and sulfuric acid. On a Western diet, adults produce approximately 1 mEq of acid per day; 2 mEq of calcium are required to buffer this amount and prevent metabolic acidosis.

Removal of this amount of calcium from the skeletal stores would result in a 15% loss of inorganic bone in an average individual in a decade. Of course, the more acid precursors consumed, the greater the degree of systemic acidity and the greater potential loss of bone.

With increasing age, renal function declines and acidity increases; thus, humans become more acidic with age. Markers of meat intake, including zinc, magnesium, phosphorus, protein and fat, were statistically related to rapid bone loss in menopausal women. Women with a higher ratio of animal to vegetable protein intake had a higher rate of bone loss and a greater risk of hip fracture than did those with a low ratio of animal to vegetable protein intake.

**Fruits and Vegetables:** Recent population based studies have suggested a positive association between high levels of consumption of fruit and vegetables, with their high levels of potassium, magnesium, beta-carotene, fiber and Vitamin D, and bone mass and bone metabolism in women and elderly men.

This may be due to the beneficial effects of the alkaline environment resulting from a diet rich in fruit and vegetables, and its ability to counter the effects of the acid-producing consumption of animal proteins and fats. Increasing one's fruit and vegetable intake from 3 to 9 servings a day may decrease urinary calcium excretion by as much as 30%.

**Sodium:** Modest salt restriction (5 grams daily) has been shown to reduce urinary calcium losses in the elderly, potentially resulting in a reduction in the loss of bone mass of around 1.5% per year.

**Other Nutrients:** As more study is done on the brittle bone disease, researchers are learning about other trace elements that appear to play a yet undefined role in bone health.

**Magnesium** is a component of every body cell, vital to the enzyme system and important to nerve transmission. About half of the body's total supply is stored in the bones, and when osteoporosis depletes them, this mineral is also lost.

**Manganese**, found in whole grains, nuts, some fruits, vegetables and soybeans, may prevent osteoporosis. A recent study of osteoporotic women found them to have extremely low levels of this trace metal, but the way it protects the bones has not been clearly explained. If a supplement is taken,

it should not be taken with calcium since the two compete for absorption.

**Vitamin K** appears to be needed for bone health, as several key bone proteins found in the bone matrix depend on Vitamin K for their synthesis. Significant circulating levels of menaquinone (Vitamin K2) have also been found in healthy elderly women and following osteoporotic fractures of the spine and hip.

**Vitamin B12** may have an important role as well. A study of osteoporosis patients found that a large proportion of those with low levels of this vitamin had suffered a fracture. There is also evidence that Vitamin B12 suppresses osteoblastic activity.

## Building “Bone” Blocks

As discussed above, Calcium and Vitamin D are the cornerstone of osteoporosis prevention, yet they are not the whole story. Other minerals and nutrients that are vital to a healthy bone matrix include magnesium, potassium, vitamin C, vitamin K, vitamin B12, and others, including zinc, manganese, boron, copper, and silicon. It may also turn out that not only is supplementation vital to preventing and treating osteoporosis but that the timing of the supplementation is important. For example, in a study of healthy volunteers, two doses of 500 mg calcium and 400 IU vitamin D taken six hours apart produced a more prolonged decrease in serum parathyroid hormone levels (low levels of which indicate adequate calcium levels) than a single dose with the same total amounts of calcium and vitamin D.

**Magnesium:** Magnesium plays essential roles in bone formation and helps with calcium absorption. Studies have found that magnesium deficiency is associated with osteoporosis and bone fragility and that adequate magnesium intake is associated with increased bone mineral density among white men and women. Unfortunately, many people have magnesium deficiency, which may be caused by alcohol abuse or malabsorption.

Dietary magnesium deficiency in North Americans often occurs because people do not consume enough dark green, leafy vegetables, which are rich in magnesium. If not provided in the diet, magnesium should be taken as a supplement.

The optimum ratio of calcium to magnesium is believed to be 2:1, though extra magnesium may be needed to protect against atherosclerosis.

**Phosphorus:** Phosphorus regulates bone formation, inhibits bone resorption, and also affects the regulation of calcium metabolism. Although there are few studies on the direct effect of phosphorus on bone mineral density, it is important to maintain a proper phosphorus-to-calcium intake because of the effect phosphorus has on calcium metabolism.

One researcher recommended a daily intake of 1000 mg calcium, with three-quarters as much (750 mg) phosphorus, as this intake was associated with higher bone mineral density among young women. It is also a good idea to reduce the consumption of soft drinks since they are high in phosphorus and can unfavorably alter the calcium/phosphorus balance.

**Other minerals and trace elements:** Few people are aware of the importance of balanced intake of minerals and trace elements, including copper, zinc, silicon, and boron. Recent research suggests that it is important to ensure adequate intake of these minerals.

**Copper** plays an essential role in bone metabolism and turnover. It modulates the differentiation and proliferation of osteoblast precursors, namely the mesenchymal stem cells. Women taking copper supplementation have shown improved bone density, while copper deficiency can produce osteoporosis in animal models of the disease.

**Boron** assists with calcium absorption and bone formation. It maximizes the body's utilization of calcium, vitamin D, and magnesium and has shown antiosteoporotic activity. It is especially effective in the presence of deficiencies in Vitamin D, magnesium, and potassium.

The role of **zinc** in osteoporosis is less well understood, but it is increasingly apparent that zinc deficiency is a risk factor for osteoporosis. It has been theorized that zinc deficiency may lead to the increase of natural anticoagulants in the blood. In alcoholics, zinc has also been shown to limit the damaging effects of alcohol on bone.

**Silicon** is also important to bone health. A study of both men and women in the large Framingham Heart Study found that silicon intake was positively related to increases in bone mineral density in the hip and spine.

**Strontium** is a mineral that could be an alternative bone builder in light of the recent negative discoveries of bisphosphonates. Naturally occurring in seafood,

whole grains, and legumes, albeit in amounts much smaller than recommended therapeutic doses.

A study published in the *New England Journal of Medicine* reported positive outcomes for women with osteoporosis, who consumed strontium supplements. During this study of postmenopausal women, strontium reduced the risk of vertebral fractures by 33% over a four year period.

Ask your natural health practitioner more about strontium. If you do take it, make sure you separate your intake of calcium and calcium-containing foods from the strontium by a few hours; the two minerals may compete for absorption.

**Vitamin C and Vitamin E:** Vitamin C, also known as ascorbic acid, is essential for the formation of collagen and the stimulation of proteins derived from osteoclasts. Studies show that Vitamin C contributes to increased bone mineral density by improving markers of bone turnover and that increased antioxidant intake, especially Vitamin E, is associated with reduced risk of hip fracture, especially among smokers. It is also necessary for the synthesis of steroid hormones and neurotransmitters, which are vital to bone formation.

In addition, this vitamin makes iron more available. Vitamin C is a powerful antioxidant and helps protect the body from cytokines that are produced during bone breakdown. Studies demonstrate a significant decrease in antioxidant defenses in older women.

**Bioflavonoids:** Bioflavonoids include rutin, quercetin, hesperidin, and eriodictyol. They are found in onions, peppers, garlic, black currants, blueberries, red berries, buckwheat, and green tea. These nutrients have been shown to stimulate bone morphogenetic proteins, which are known to increase bone formation, as much as 30%.

**Vitamin B12:** Recent evidence has implicated elevated homocysteine as a possible risk factor for osteoporosis, especially in women. Vitamin B12, together with folic acid and vitamin B6, can help lower homocysteine. Before the evidence connecting elevated homocysteine to osteoporosis emerged, vitamin B12 had already been identified as a possible strategy to reduce the risk of osteoporotic fracture, primarily because vitamin B12 deficiency has been associated with decreased bone-mineral density in the hip. Vitamin B12 and



## Diet Tips for Healthy Bones

1. Eat plenty of foods high in calcium and Vitamin D. Good sources of easily absorbable calcium include broccoli, chestnuts, clams, dandelion greens, most dark green leafy vegetables, flounder, salmon, shrimp, whole grains, oats, beans, tofu, soybeans, and wheat germ.
2. Oxalic acid in certain foods binds with calcium in the intestine and prevents its absorption. Spinach, parsley, collard greens and beet greens, almonds, cashews, and asparagus are high in oxalic acid and calcium absorption from these sources is limited. Turnip greens, kale, and endive are high in calcium and low in oxalic acid.
3. Consume whole grains and calcium at different times. Bran cereals and unleavened whole-wheat products contain phytate, which binds calcium in the intestine and prevents its absorption.
4. Many Oriental foods contain high amounts of calcium, including sesame seeds, seaweed, miso, and soy products. However, they may also contain a great deal of sodium.
5. Alligator meat is an excellent source of calcium. (A bit difficult to catch but still good for you.)
6. At least three servings of a dairy food are necessary to give 1000 mg of calcium. Cottage cheese, yogurt and sliced cheese are good sources.
7. Tolerance to lactose can be increased by adding lactase tabs to milk 18 to 24 hours before drinking it. Yogurt may be better tolerated than other dairy products since it's low in lactose.
8. Grated cheddar and Swiss cheese contain more calcium and less calories than Parmesan.
9. Use cottage cheese as a topper for bread instead of butter. Tofu also adds calcium to foods.
10. Include garlic and onions in the diet, as well as eggs. These foods contain sulfur, which is needed for healthy bones.
11. Marinating meat with the bone in it dissolves the bone and provides some calcium.
12. Fish bones (as in canned salmon, sardines) are rich sources of calcium.
13. Taking a calcium supplement with juice or food will provide acid to aid dissolution.
14. Oranges and papayas are high in calcium. Most other fruits aren't. Limit consumption of citrus fruits, as they may inhibit calcium intake.
15. Natural sweeteners, maple syrup and molasses contain a good deal of calcium, especially Blackstrap molasses.
16. More calcium is contained in broccoli leaves than in the stalks.
17. If you can eat shrimp, shells and all, you will get more calcium.
18. High fat foods bind calcium in the intestine.
19. A diet high in red meat and other animal proteins prevents calcium absorption.
20. Avoid phosphate-containing products such as soft drinks, high-protein animal products, and alcohol. Also avoid sugar and salt.
21. Avoid yeast products. Yeast is also high in phosphorus, which competes with calcium for absorption.

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## Can Herbs Help?

Alfalfa, barley grass, black cohosh, boneset, dandelion root, nettle, parsley, pokeroor, rose hips, and yucca help to build strong bones.

*Caution:* Do not use boneset on a daily basis for more than one week, as long-term use can lead to toxicity.

Feverfew is good for pain relief and acts as an anti-inflammatory. *Caution:* Do NOT use in pregnancy.

Horsetail and oat straw contain silica, which helps the body absorb calcium.

folate have been shown to reduce the risk of hip fracture in elderly Japanese people who have suffered stroke.

**Vitamin K:** Vitamin K facilitates the activity of calcium in bone building. Vitamin K is necessary for the activation of osteocalcin, a protein found in relatively high amounts in the bone, which allows calcium to bind to bone matrix. Osteocalcin that is not appropriately synthesized with vitamin K may lead to low bone mineral density and an increased risk of osteoporosis.

Diets with more vegetables and less meat are higher in vitamin K. One study examined the relationship between vitamin K intake and hip fracture. Using 10 years of data on 72,000 participants in the Nurses' Health Study, researchers found that study participants who received the most vitamin K were about a third less likely to get a hip fracture. Those who ate lettuce every day slashed their risk of hip fracture in half compared to those who ate it less than once a day (lettuce is a source of vitamin K). The effect of taking vitamin K was greater than the effect of taking synthetic estrogen, which did not protect the participants' bone density in this study, nor did vitamin D. In fact, women who took a lot of vitamin D but had low intakes of vitamin K had a doubled risk of hip fracture. Although vitamin D increases the amount of bone-friendly osteocalcin, only vitamin K can make it work properly.

Vitamin K shows remarkable results

against bone loss in postmenopausal women. A study of 1 mg vitamin K daily for two weeks demonstrated it increased the bone-building protein gamma-carboxyglutamic acid in women. Another study showed that vitamin K slowed calcium loss by one-third in people who have a tendency to lose it. Drugs containing vitamins K1 and K2 are being used to treat osteoporosis. The dosage used in Japan is 45 mg daily.

**Red Clover:** Lucky News for Bones-- Those little red clover flowers that pop up each spring aren't just weeds—they may contain a powerful tonic for your bones.

In recent years there's been tantalizing evidence that red clover supplements, which contain estrogen-like compounds similar to those in soy, may stem bone loss. Now, one of the largest and longest studies so far gives the herb the nod.

Researchers at the University of Belgrade School of Medicine gave 29 postmenopausal women doses of red clover-derived isoflavones daily, while the other 24 women was without medication. After a year, the supplement group showed a significant increase in bone composition markers and osteodensity. The researchers concluded that the positive bone turnover after red clover consumption helped to prevent the progression of osteoporosis.

## Diet and the Acid-Base Balance

Diet is clearly a major influence on the body's acid-base balance. Certain foods, such as proteins, grains, beans, coffee, white sugar and many processed foods, generate free acid as they are metabolized. Other foods contain mineral compounds such as potassium citrate and magnesium salts that generate bicarbonate, the body's main compound for detoxifying and removing metabolic acids from the body.

A diet balanced in base-forming and acid-forming foods creates little or no acid buildup and no threat to bone alkali reserves. A base-forming diet is familiar to humans; in fact, scientists calculate that during the vast majority of human evolution our diet was, indeed, overall base forming. The contemporary diet of industrialized countries, however, is uniformly acid forming or "acidogenic." Returning to an alkali-rich, base-forming diet provides the cornerstone of a new diet for healthy bones.

A diet that balances base- and acid-forming foods maintains the body's systemic pH balance. If acid-forming foods predominate, however, as is the case in the typical Western diet, the first-line alkali reserves in the blood and cells are soon exhausted and the body starts using minerals stored in the bones.

The body's goal here is basic survival, and if it becomes even slightly acidic, it willingly sacrifices the structural integrity (strength) of the bones in order to recover the systemic acid-base balance. Simply put, the body places its short-term need to survive above a long-term need for strong bones.

Back to Basics History provides key insights for a modern healthy bones diet. Our ancestral diet was nutrient dense—rich in vitamins, minerals, phyto-compounds, omega-3 fatty acids and protein. This balanced diet provided sufficient alkaline-forming foods to buffer the acids produced as a by-product of eating lots of animal flesh. Ancestral bones, it appears, were only infrequently sacrificed in order to maintain critical systemic pH balance. Following our ancestors' lead, a healthy bones diet should be nutrient rich and contain:

Fruits in a variety of colors 4–6 servings a day (one serving equals one fruit;

1½ cup frozen or canned fruit; 1¼ cup dried fruit or 6 oz fruit juice)

Vegetables of low and medium starch content (in a variety of colors) 2–4 cups mostly cooked vegetables 1–2 cups raw leafy salad vegetables

Grains/High-Starch Root Crops 7–8 servings a day (one serving equals one slice whole-grain bread, 1½ cup dry or hot cereal or 1½ cup cooked rice, other grain or pasta)

Animal protein (fish, poultry, meat, eggs) 2 or fewer servings a day

Nuts, Seeds and Dried Beans 1–2 servings (one serving equals 1/3 cup nuts, 2 tablespoons seeds or 1/2 cup dried beans, cooked). If you don't eat meat, include one or two servings of beans a day.

Oils – The link below, published by United States Department of Agriculture demonstrates the appropriate daily allowance for oils.

**Visit [www.choosemyplate.gov/food-groups/oils\\_allowance\\_table.html](http://www.choosemyplate.gov/food-groups/oils_allowance_table.html).**

Fermented foods A daily serving of 1 cup yogurt or kefir or 2–3 oz. tempeh or sauerkraut. Use pickled vegetables, umeboshi plums or miso as condiments.

Wholesome Sweets 3–5 times per week (one serving equals 2 small whole grain cookies; ¾ cup pudding, compote, ice cream, sherbet or sorbet; 1 small piece fruit-based dessert or piece of cake) As near as possible, today's healthy bones diet should include adequate amounts of all the 19 key bone-building nutrients (See sidebar opposite), at least 64 ounces of water and a 15 minute "serving" of sunlight to maintain internal vitamin D production.

## Sample Meals

**Breakfast:** Alkalinizing whole-grain hot quinoa cereal with yogurt, milk or milk substitute, fruit and a few nuts. Green tea or grain/herb coffee substitute • Fresh mixed-fruit salad and cow, sheep, goat or soy yogurt or cottage cheese with roasted nuts and seeds sprinkled on top • Almond or cashew butter, ghee, avocado, sheep, goat or cow milk cheese on rice cakes or other whole-grain crackers/bread with fruit or fresh fruit juice • "Green rice" (avocado mixed with wild or brown rice and a pinch of sea salt or tamari) served with fruit and hot beverage • Left-over lentil or vegetable soup or stew accompanied by a toasted sheet of nori sea

vegetable • An omega-3 enriched egg omelet (or scrambled tofu omelet) with 1 cup of chopped vegetables, fresh cut fruit or a fruit salad • Vegetable miso soup with wakame sea vegetable and brown rice (optional) • Mashed sweet potatoes or yams served with eggs, or slices of tempeh and fruit, herb tea, green tea, grain/herb coffee substitutes, lemon or lime water and 100 percent fresh fruit or vegetable juices are healthful additions to any breakfast. Those with sensitive stomachs might want to heat or cook the fruit in these dishes, or eat it separately as a between-meal snack.

**Lunch:** Steamed vegetables (broccoli, bok choy, cut-up kale leaf, carrots, celery, red, yellow or orange peppers), grilled tofu or chicken (3–4 oz), brown rice, quinoa, millet • Whole-grain bread sandwich with avocado, cheese, lettuce and tomato, served with a salad of mixed field greens • Vegetable lentil soup with a salad of mixed greens, green olives and artichoke hearts • "Green rice" and steamed vegetables • Salad with pieces of leftover baked potato • Beans and brown or wild rice with avocado slices and steamed vegetables

**Dinner:** Stir-fry vegetables with tofu or meat and alkaline-forming root crop like sweet potatoes, yams or parsnips • Baked fish, turkey, chicken or other meat with baked squash and sautéed greens (collard, kale, dandelion or turnip greens) • Whole grains served with steamed or sautéed vegetables (2 cups) and a small amount of animal protein (e.g. meat or fish) or beans Dinners may occasionally include a "nourishing sweet" such as baked apples, fruit compotes or rice pudding made with maple syrup, rice syrup or raw cane sugar, naturally and lightly sweetened fruit pies or apple crisp with whole-grain topping or whole-grain cakes and cookies.

**Snacks:** Fresh fruit • Yogurt (cow, sheep or goat) • Whole-grain crackers with almond or cashew butter, cottage cheese or sheep or goat cheese • Lightly roasted almonds or pumpkin seeds • Fresh fruit or vegetable juices • Vegetable broth • Frozen fruit-juice treats, sherbets or smoothies • Frozen blueberries

## Pharmacologic Management of Osteoporosis

Current medications used to treat osteoporosis include antiresorptive agents, such as bisphosphonates, selective estrogen-

receptor modulators (SERM) or anabolic agents, such as teriparatide. No pharmacologic therapy should be administered for an indefinite time period. Moreover, the National Osteoporosis Foundation recommends that pharmacologic therapy should only be given to postmenopausal women and men aged 50 years or older, who meet the following conditions:

- Low BMD – T-score between -1.0 and -2.5 at the femoral neck
- A ten year probability of a hip fracture of 3% or higher, or a ten year probability of a major osteoporosis-related fracture of 20% or higher.
- A current hip or vertebral fracture

## Antiresorptive Agents

A number of drugs have been identified that slow the progression of bone loss by suppressing the remodeling process. They reduce the depth of bone resorption (perhaps by reducing the life span of the osteoclasts) and may increase bone formation (perhaps by prolonging the life span of the osteoblasts.) Bone mineral density may increase due to more complete filling of the remodeling space and more complete mineralization of the existing bone. No new bone is made, so use of these substances is unlikely to replace bone already lost or change the existing bone's architecture or overall strength.

Although there is no cure for osteoporosis, there are treatments available to help stop further bone loss and reduce the risk of fractures these include:

**Bisphosphonates** - Bisphosphonates are prescription drugs that interfere with osteoclast function and reduce the number of osteoclasts. These drugs are among the most thoroughly investigated in the field of osteoporosis. They offer an alternative treatment for those post-menopausal women who cannot or choose not to use HRT. They have been shown to prevent bone loss, increase bone mineral density, and reduce the risk of fracture of the spine, hip and forearm.

The reduction in fracture risk is usually seen within the first 12 to 18 months of treatment. It is not clear, however, whether the risk reduction is comparable across all age groups, and whether or not it is sustained or lessens with time. Risedronate and Alendronate are approved by the FDA for the prevention and treatment of postmenopausal and steroid-induced osteoporosis.

Etidronate is also a member of this class. An advantage of these drugs is that they are not hormones and have no significant effect on tissues other than bone. The bisphosphonates are poorly absorbed, so should be taken on an empty stomach with plain water, at least 30 minutes before eating, drinking other liquids, or taking other medications. They have been reported to cause upper gastrointestinal irritation, and studies are underway to determine the relative safety of risedronate and alendronate.

Esophageal irritation and other GI symptoms may be reduced by remaining upright after taking the medication. Once weekly dosing and other dosing schedules are being evaluated.

Natural medicine physician Jonathan Wright, MD, coauthor of *Natural Medicine, Optimal Wellness: The Patient's Guide to Health and Wellness*, explains that unfortunately, these drugs don't create new bone. And there is a recently publicized link between bisphosphonate drugs and jaw osteonecrosis (bone death), as well as the possibility of severe esophagus damage when these medications aren't completely swallowed, make matters worse. Given such concerns, restoring balance between breaking down old and creating new bone seems a far better solution. Examples of bisphosphonate drugs include:

- alendronate (Fosamax)
- risedronate (Actonel)
- ibandronate (Boniva)
- Zoledronic acid (Reclast)
- alendronate plus vitamin D (Fosamax Plus D),
- risedronate with calcium (Actonel with Calcium),
- risedronate delayed-release (Atelvia)
- etidronate (Didronel)

### Calcitonin (Miacalcin or Fortical)

This medication is approved for women who are more than five years post menopause. This drug is a hormone that decreases osteoclast activity, which impedes bone loss. Research has indicated that this drug reduces vertebral fractures by as much as 30% in patients with prior vertebral fractures. It is administered as a daily intranasal spray in a dose of 200 IU. It is also available subcutaneous by injection.

Selective Estrogen Receptor Modulators (SERMs): This class of drugs acts in

different ways on the estrogen receptors of various organ systems. The first compound of this class that was shown to affect bone metabolism was tamoxifen, which prevented bone loss in post-menopausal women and appeared to prevent fractures in women treated with it for prevention of breast cancer.

Although tamoxifen stimulates the endometrium, increasing the risk of endometrial cancer, its discovery spurred the search for a similar compound that would not cause cancer but retained its bone protective effects. One such compound is raloxifene (Evista), the most extensively studied SERM in the bone field today.

Both animal and human studies suggest that raloxifene reduces the rate of bone turnover, but to a lesser degree than estrogens or bisphosphonates do. When administered to post-menopausal but not osteoporotic women for two years, raloxifene increased bone mineral density and prevented bone loss at all skeletal sites; this effect was sustained for up to four years. It also reduced the risk of vertebral fractures in osteoporotic women with and without previous fractures. It did not, however, alter the risk of nonvertebral fractures.

Raloxifene increases the incidence of certain post-menopausal symptoms such as hot flashes, especially when given soon after the menopause. It may also decrease the incidence of breast cancer. Raloxifene does not stimulate the endometrium and thus should not increase risk for endometrial cancer.

### Other Non-FDA Approved Medications

**Tibolone** is not a SERM but shares some of the properties of the class. It is a synthetic steroidal compound with combined estrogenic, progestogenic, and androgenic properties. It should be noted that this medication has not been approved by the FDA for treatment of post-menopausal symptoms. However, research indicates that it appears to suppress bone turnover and increase bone mineral density to a degree similar to that achieved by HRT. This compound may present an option for the management of postmenopausal women, and appropriate clinical trials appear warranted.

**Protelos (strontium ranelate)** was the first dual-action osteoporosis drug on the market. It works by both increasing

new bone formation and decreasing bone resorption, thus rebalancing bone turnover in favor of bone creation and strengthening the bones.

Two large, multinational studies have been conducted on the safety and effectiveness of Protelos at 2 g daily. The Spinal Osteoporosis Therapeutic Intervention trial found that Protelos reduced the risk of a new vertebral fracture by 41 percent after three years, compared with placebo. The Treatment of Peripheral Osteoporosis study found a 16 percent reduction in the risk of non-vertebral fractures in all patients and a 36 percent reduction in hip fractures among high-risk patients. In both studies, Protelos was well tolerated with a lower side-effect profile than existing osteoporosis drugs.

**Genistein** is an isoflavone phytoestrogen that has had preliminary research indicating positive bone health benefits in postmenopausal women.

**Sodium fluoride** has been used to motivate the formation of bone growth. However, further research is needed to fully understand its effects on bone health.

There is also some indirect evidence that **NSAIDs** may decrease bone turnover and be associated with greater bone mineral density. The effect seems to be related to their inhibition of COX-2 activity, and the recent availability of better-tolerated COX-2-specific inhibitors may open a new area of investigation for the treatment of osteoporosis.

### Receptor Activator of Nuclear Factor kappa-B ligand (RANK)

**Denosumab (Prolia)** is a Receptor Activator of Nuclear Factor kappa-B ligand (RANK), which plays a key role in bone remodeling. This medication, approved by the FDA, decreases bone resorption by inhibiting osteoclast activity. In a randomized placebo-controlled trial, denosumab reduced hip fractures by 40%, vertebral fractures by 68% and non-vertebral fractures by approximately 20%. In another recent randomized controlled trial, denosumab increased BMD in women within a 12 month period.

### Parathyroid Hormone

There has been a recent flurry of interest in the use of parathyroid hormone (PTH) as an anabolic treatment for osteoporosis.

Recent studies have shown that PTH reduces the risk of spinal fractures in postmenopausal women with and without previous fractures, and that this effect may be above and beyond that achieved by HRT in these same women. Fracture-related pain and mean height loss were also reduced. In contrast to the antiresorptive agents, PTH appears to be able to alter the architecture of existing bone and thereby increase its strength. Animal studies have shown that PTH is able to increase the thickness of both cortical and trabecular bone, and perhaps trabecular connectedness as well. The structural changes are accompanied by increased bone strength and seem to diminish after cessation of treatment; however, coadministration or later addition of antiresorptive agents to the treatment regimen may maintain the structural changes achieved by PTH.

**Teriparatide (Forteo)** is a man-made form of a hormone called parathyroid that exists naturally in the body. Teriparatide increases bone density and increases bone strength to help prevent fractures. Teriparatide is used to treat osteoporosis in men and women who have a high risk of bone fracture.

Note: Forteo has been shown to increase the rate of bone tumors in lab animals. It is unknown if there is a higher risk of bone tumors in humans. Forteo should not be used in patients who are at risk for bone tumors, including patients with Paget's disease, open epiphyses, prior radiation involving the skeleton, or unexplained high levels of alkaline phosphatase in their blood.

Note: Brand names included in this course are provided as examples only, and their inclusion does not mean that these products are endorsed by the National Center of Continuing Education, Inc. Also, if a particular brand name is not mentioned, this does not mean or imply that the product is unsatisfactory.

## Osteoporosis in Men

Although osteoporosis has generally been considered a disease of women, primarily because of its association with the physiological changes of menopause, men suffer from osteoporosis, too. Of the nine million Americans who have osteoporosis, 2 million are men, and an additional 3.5 million men are at risk for development of the disease. Thus, osteoporosis in men is a significant but understudied problem. Up

until recently, assessment tools were only applicable to women. Progress has been made in the identification of osteoporosis. Case in point is the FRAX® tool, which is now applicable to both men and women.

Although men are less likely to fracture than women are, the lifetime risk of fracture in men is still 13% to 25%, and men have higher morbidity and mortality rates due to hip fractures than women. At age 50, the lifetime risk of hip fracture for men is about a third that of women, at 6% and 18% respectively, but the gap narrows considerably over subsequent decades. As in women, the risk of hip fracture in men is influenced by weight and physical activity: men who are overweight are 40% less likely, and those who are physically active are 16% less likely, to sustain a fracture. The majority of men with a diagnosis of osteoporosis will have a clearly identifiable cause: alcohol or tobacco abuse, a specific hormonal abnormality or other underlying disease, or secondary to corticosteroids or other medication use.

The incidence and risk of osteoporosis and fracture in men is less for a number of reasons. Men attain a higher peak bone mass during early adulthood, and their skeletons differ in dimension and strength as well. There is no dramatic hormonal change equivalent to a woman's menopause, although hormonal changes are implicated in the development of osteoporosis in men. Men have a shorter life expectancy, and they appear less likely to sustain a bone-fracturing fall. Interestingly, however, men are twice as likely to die during the year following hip fracture as are women.

After achieving peak bone mass, men generally maintain their bone mineral density (BMD) through middle age then lose it gradually, with an average lifetime decrease of approximately 40%. Like women, men tend to lose more trabecular bone than cortical bone, but the pattern of loss may be somewhat different. In women, the loss is due primarily to increased bone resorption, and results in diminished connectivity. In men, the loss appears to be due primarily to reduced bone formation; connectivity is maintained, with relatively less fracture risk as a result.

Many of the risk factors for development of osteoporosis are similar in men and women, including low dietary calcium and Vitamin D intake, diets high in animal protein and fat, smoking and alcohol use,

slight thin build, and lack of physical activity.

Overt hypogonadism in men has long been associated with low bone mass; bone loss results from hypogonadism in adults, whereas failure to achieve peak BMD is a consequence of hypogonadism of prepubertal onset. Beyond that fact, the specific relationship between hormone levels and osteoporosis in men is not clear. Testosterone appears to have a significant role in attainment and maintenance of BMD in men.

Although there is no abrupt cessation of testicular function in healthy males, total and free testosterone levels may decline with age, or remain in the normal range throughout the life span. Relatively low correlations have been found, however, between testosterone levels and BMD in older men.

It should be noted that androgens are partially converted to estrogens in older males, and higher correlations have been observed between estradiol (an endogenous estrogen) and BMD. The relative contribution of these hormones is still unknown, and both have been suggested to have a role in osteocyte apoptosis in both men and women. Recent treatment of prostate problems has included androgen deprivation: if this therapy proves to be successful, treated men may evidence an increased incidence of osteoporosis and fracture.

Genetic factors include higher overall risk in Caucasian and Japanese men, and may explain at least in part the 80% variance in peak BMD among young men. Familial factors such as maternal history of vertebral fracture have also been shown to contribute to osteoporosis in men. Genetic studies of the disease in men have focused on male hormone receptors and collagen genes.

Treatment of osteoporosis in men focuses on lifestyle modifications to prevent further bone loss and reduce fracture risk, treatment of disease-related pain, and various pharmacological approaches. Few drugs have been specifically approved for treatment of osteoporosis in men, but off-label use of those drugs indicated for women is not uncommon.

Clinical trials have shown that the bisphosphonates are generally effective in increasing BMD and lowering the risk of fracture among men with both idiopathic and corticosteroid-induced osteoporosis.

Alendronate has been found to increase BMD at the hip and spine, and to decrease the risk of spine fracture; it has been approved by the FDA for treatment of steroid-related bone loss in men.

New data suggest that alendronate is equally effective in men with low testosterone levels. Recent studies of risendronate have suggested that it, too, may be effective in reducing bone loss and preventing fractures. Several interesting studies have addressed the possibility of using SERMs to treat osteoporosis in men, given the apparent role of estradiol in bone health. Effects appear to be dependent on endogenous estradiol levels: the effects of raloxifene on biochemical markers of bone remodeling in men were determined by their circulating level of estrogen.

One of the most promising recent studies involves teriparatide therapy. Teriparatide is classified as a parathyroid hormone (PTH) and is known to increase bone mass in men. During this study, 83 men with low BMD received 10 mg/day alendronate and or 40 mcg/day teriparatide. The results showed that lumbar spine and neck BMD increased significantly in men, who were administered teriparatide monotherapy. Based on the results of this investigation, researchers stated that teriparatide appears to be a viable option for men with osteoporosis.

## Patient Education

Isn't osteoporosis something that happens only to older people?

No. Osteoporosis can begin when you are a child and is often not found until you are much older. That is why it is so important to eat well and get lots of exercise throughout your life to keep your bones healthy and strong.

### What causes osteoporosis?

A family history of osteoporosis, the foods you eat, your hormone make-up, your age, and how you live your life all play a role in causing osteoporosis. The strength of your bones depends on their mass and density. And bone density depends in part on the amount of calcium, phosphorus and other minerals bones contain. When your bones contain fewer minerals, their strength is decreased and they lose their internal supporting structure. Scientists have yet to learn all the reasons this occurs, but the process involves how bone is made. Bone is continuously changing — new bone is

made and old bone is broken down, a process called remodeling, or bone turnover.

A full cycle of bone remodeling takes about 2 to 3 months. When you're young, your body makes new bone faster than it breaks down old bone, and your bone mass increases. You reach your peak bone mass in your mid-30s. After that, bone remodeling continues. But you lose slightly more than you gain — about 0.3 percent to 0.5 percent a year. Not getting enough vitamin D and calcium in your diet can accelerate the process.

Your risk of developing osteoporosis depends on how much bone mass you attained between ages 25 and 35 (peak bone mass) and how rapidly you lose it later. The higher your peak bone mass, the more bone you have "in the bank" and the less likely you'll be to develop osteoporosis as you lose bone during normal aging. Getting enough calcium and vitamin D in your diet, which is essential for absorbing calcium, and exercising regularly can help ensure that your bones stay strong throughout your life.

### How do I find out if I have osteoporosis?

In the early stages of bone loss, you usually have no pain or symptoms. But once bones have been weakened by osteoporosis, you may have symptoms that include:

- Back pain
- Loss of height over time, with an accompanying stooped posture
- Fracture of the vertebrae, wrists, hips or other bones

Talk with your doctor to find out if you should be tested for osteoporosis. Doctors can detect early signs of osteoporosis with a simple, painless bone density test (densitometry) or through other assessment tools such as the previously discussed Fracture Risk Assessment FRAX® method.

If you're a woman, the National Osteoporosis Foundation recommends that you have a bone density test if you aren't taking estrogen and any of the following conditions apply to you:

- You use medications that can cause osteoporosis.
- You have Type 1 diabetes (the type of diabetes that generally affects young people and requires treatment with the hormone insulin), liver disease, kidney disease or a family history of osteoporosis.

- You experienced early menopause.
- You're postmenopausal, older than age 50 and have at least one risk factor for osteoporosis.
- You're postmenopausal, older than age 65 and have never had a bone density test.

Doctors don't generally recommend osteoporosis screening for men because the disease is far less common in men than in women.

### How is osteoporosis treated?

There are medications and hormones (chemicals that are made in your body) that are used to slow down the bone loss and even help build the strength of the bones back up. These treatments help keep your bones from breaking. Right now, hormone replacement therapy (HRT) is the best way to prevent osteoporosis in women.

If you're considering HRT, you should know that estrogen therapy might cause side effects, including uterine bleeding and breast tenderness. There are treatments, however, that can reduce these effects. If you can't or don't want to take estrogen, other prescription drugs can help slow bone loss and may even increase bone density over time. Talk with your doctor to find out which treatments are best for you.

### What can I do to help keep my bones healthy?

Here are some ways you can help keep your bones healthy.

- Get enough calcium. Calcium builds strong bones. You can get calcium by drinking lots of milk; eating yogurt, cheese, and other dairy products; and eating dark green and leafy vegetables. You also can eat foods that have calcium added to them such as orange juice and cereals. Many women take calcium pills to make sure they get enough calcium. Talk to your doctor about what is the right amount of calcium for you to get every day.
- Get exercise. Exercise is very important for good health and strong bones. Some of the best exercises for your bones are called weight-bearing exercises. Walking, running, dancing, climbing stairs, or using weights in a gym are all examples of weight-bearing exercise. Talk with your doctor about what kind and how much exercise is best for you.
- Don't smoke. People who smoke have more of a chance of getting osteoporosis.

- Don't drink lots of alcohol. Alcohol can hurt the cells that build your bones. It also lowers the amount of calcium in your body.
- Don't skip meals or go on fad diets. Skipping meals and going on fad diets can take away the foods your bones need to stay healthy.
- Talk to your doctor about hormone replacement therapy. Even before you go through menopause (the change of life), talk with your doctor about hormone replacement therapy. This is a treatment that puts hormones back in your body after your body stops making them. Hormone replacement therapy can help keep bone loss from taking place.
- Talk to your doctor to find out if you are at an increased likelihood of having osteoporosis than other people. Your doctor can help you find ways to keep your bones healthy and do tests to check the health of your bones. If you do get osteoporosis, your doctor can treat you with medicines and hormones.

### What if I already have osteoporosis?

These suggestions may help you relieve symptoms and maintain your independence if you have osteoporosis:

- Maintain good posture. Good posture — which involves keeping your head held high, chin in, shoulders back, upper back flat and lower spine arched — helps you avoid stress on your spine. When you sit or drive, place a rolled towel in the small of your back. Don't lean over while reading or doing handwork. When lifting, bend at your knees, not your waist, and lift with your legs, keeping your upper back straight.
- Prevent falls. Wear low-heeled shoes with nonslip soles and check your house for electrical cords, throw rugs and slippery surfaces that might cause you to trip or fall.
- Manage pain. Discuss pain-management strategies with your doctor. Don't ignore chronic pain. Left untreated, it can limit your mobility and cause even more pain.

### Future Impact of Osteoporosis

As our population continues to age, osteoporosis will have an even more profound impact upon the health of our nation. The economic consequences are

enormous. Our rapidly expanding population is susceptible to this crippler and we should plan to care for 1.7 billion osteoporotic fractures by early in this century. Since everyone is in danger of developing the disease, steps must be taken now. Life-style and personal risk factors must be evaluated, and this becomes a highly personal process. What can YOU do to practice early prevention for yourself and your family? If the disease has already attacked your bones, what can be done to diminish the devastation? What can be done to delay its progression? For many, osteoporosis is a preventable disease, if they take action early. Just sitting back and waiting for the inevitable is foolish. Prevention of osteoporosis requires YOUR knowledge, YOUR participation and YOUR commitment.

### Selected Resources

You can find out more about osteoporosis by contacting the National Women's Health Information Center (800-994-9662) or the following organizations:

National Osteoporosis Foundation  
Phone: (202) 223-2226 or  
[www.nof.org](http://www.nof.org)

Osteoporosis and Related Bone Diseases National Resource Center  
Phone: (202) 223-0344  
(202) 466-4315 (TDD)  
(800) 624-2663 (BONE)  
<http://www.osteoporosis.org>

National Institute on Aging  
(800) 222-2225 (Information Center)  
(800) 222-4225 (TTY)  
<http://www.nih.gov/nia>

Best Bones Forever  
<http://www.bestbonesforever.gov/>  
This girl-friendly Website helps girls understand how weight-bearing physical activity and calcium can be a fun and important part of everyday life.

Bone Health and Osteoporosis:  
A Surgeon General's Report  
[http://www.surgeongeneral.gov/library/reports/bonehealth/ppt\\_html.html](http://www.surgeongeneral.gov/library/reports/bonehealth/ppt_html.html)

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# Prevention of Falls

Osteoporosis is a major health problem because it contributes to the risk for fractures and associated medical complications. Fractures are usually caused by falls, and fall prevention may be especially important for the person with osteoporosis. Many elders are aware of the complications of a serious fall, and this can lead to a fear of falling that saps one's confidence, lessens one's independence, and decreases one's activity level.

*Here are some specific risk factors for falls, and ways to address them*

## Decreased Vision

Eyesight in general deteriorates with age, and vision may be further impaired by the development of cataracts, glaucoma, and macular degeneration. Vision may become blurred, and glare may become a problem. Older eyes often have difficulty adapting to significant changes in illumination, too. Changes in elevation, even slight ones like cracks in the sidewalk, can create a significant hazard. You can help an older person compensate for these vision problems by:

- keeping rooms lit brightly, but without glare
- removing throw rugs and other difficult to see obstacles, and creating clear paths from room to room
- increasing contrast on steps, between floor and carpet by use of white paint or tape
- reminding him to get regular eye exams and wear the correct prescription lenses.

## Limited Mobility

Age and disease-related changes in gait, posture, and balance can contribute to falls. Muscle strength may be reduced, and arthritis may lead to decreased flexibility of the joints. Associated pain may further decrease activity level, leading to even greater losses of strength and flexibility. Age-related changes in the inner ear may affect balance. Older folks often lean forward as they walk, changing their center of gravity and making their stride shorter and slower. A shuffling gait may also add risk of tripping over unexpected hazards. The older person should:

- exercise regularly
- try to maintain participation in activities such as walking, gardening, and housework for as long as possible
- use a cane or other assistive device to improve balance (usually an unpopular alternative)
- consider the use of hip pads to reduce the risk of hip fracture (even less popular!)

## Dementia

Persons with dementia, especially Alzheimer's disease, are generally at greater risk of falling than are other older adults. Safety awareness becomes limited, and the patient may lack insight into the consequences of potentially dangerous behaviors. Wandering is also common. While increased ambulation may provide

much-needed exercise, it must be done in a safe, hazard-free environment. Restraints such as wheelchair seat belts and bed rails may prevent some falls, but attempts by the patient to circumvent them may lead to even more serious injury. The safest alternative for the patient with dementia may be close, consistent supervision.

## Medications

*Prescribed and Otherwise.* Drug reactions in the elderly may include dizziness, lethargy, weakness, impaired balance, and blurred vision, and all of these are major risk factors for falls. Sedatives and psychotropic drugs are the primary culprits, along with other hypnotics, tranquilizers and some hypertensive agents. Even diuretics and laxatives can contribute to falls, however, because they lead to more frequent trips to the bathroom; and the urgency of each trip may create additional likelihood of stumbling. Polypharmacy, especially with different prescribers, can compound the problem. Encourage the older person to:

- review all of their medications, prescribed and over the counter, with their primary health care provider on a regular basis, and report any of the above symptoms as soon as possible but especially after starting a new medication
- consider use of a bedside commode or protective bed pads or undergarments as needed.