Healthy Bones
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You deserve healthy bones. But your chances of having them are getting slimmer every year. If you are a Caucasian American woman over 50, you have a 50/50 chance of suffer a fracture from osteoporosis in you lifetime.

If you have bones, this message is for you.

Osteoporosis literally means 'porous bones'. It's a progressive degeneration of the structure, density and strength of the bone. Over time, bones contain less mineral, especially calcium, and become brittle, fragile and tend to break easily.[1]

Some people with osteoporosis suffer no symptoms at all, and don't know they have weak bones, until a crisis hits. Common symptoms include backache, loss of height or stooped back, fractures that occur easily, loss of bone in the jaw, blood clots and pneumonia.

The most common cause of osteoporosis is simple aging. From about age 35, everyone's bone structure becomes less dense. When this combines with other factors, such as inadequate calcium intake over the course of many years, poor childhood bone formation, or a reduced amount of calcium taken up into the bones after menopause, osteoporosis can be the unfortunate result. Diseases affecting the parathyroid gland also affect calcium levels and can increase osteoporosis risk.

Osteoporosis is a major health threat for up to 44 million Americans. Today in the United States, 10 million patients have osteoporosis. Between eighteen million and 34 million more are estimated to have low bone mass, placing them at increased risk for osteoporosis. Osteoporosis is usually thought of as an older person's disease, but it can, and does, strike at any age.

Osteoporosis occurs to a significant degree in nearly 25 percent of white, Asian and brown-skinned women after menopause. (Black women have been shown to have greater bone density, which reduces their osteoporosis risk.)

Several risk factors for osteoporosis have been identified. The 2 major risks are peak bone mass and rate of bone loss. The National Osteoporosis Foundation says that well established risk factors include being a Caucasian or Asian woman. White women 65 or older have twice the fractures as African-American women. Other factors are advanced age, early or surgically induced menopause, chronic corticosteroid use and maternal history of osteroporosis. Less well established factors are low weight/height ratio, prolonged bed rest, high alcohol consumption, low calcium intake, cigarette smoking, high protein/phosphate intakes, high caffeine consumption and physical inactivity.

Within the next 50 years, 25% of Americans will 65 or older. Women over 65 represent the fastest growing population segment in the United States.

As the U.S. population ages, medical conditions more common among elders are gaining prominence in the public eye. Osteoporosis is going to be a major public health concern. However, it's not inevitable, as osteoporosis is, to a great extent, preventable and treatable.[2]

Broken Bones

Osteoporosis leads to bone fragility and an increased susceptibility to fractures, especially of the hip, spine and wrist. If not treated, osteoporosis can progress painlessly until a bone breaks. Breaks occur in the ribs and forearm, though any bone is at risk. In America, 1.5 million osteoporotic fractures occur each year. Potentially devastating hip fractures, which almost always require hospitalization and major surgery, make up 20% of that figure. A hip fracture can impair the ability to walk unaided and may bring prolonged or permanent disability. Only 15% of hip fracture patients can walk across a room unaided six months later.

Spinal fractures are also very serious, creating loss of height, severe back pain, and deformity.[3] The direct costs of fractures now reach $10 to $15 billion dollars a year.

Sometimes something as simple as bumping a wall or tripping over a floor rug will cause a fracture. One out of 6 women will suffer a hip fracture during their lifetime, and for women who live into their late 80's, 1 out of 3 will suffer a hip fracture, as will 1 out of 6 men.[4] Up to 30% of elders who suffer hip fractures die within 6 months of their injury.

A woman's lifetime hip fracture risk is equal to the combined total risk of breast, uterine, and ovarian cancer. And hip fractures are very life threatening- women who suffer hip fractures are up to 20% more likely die within one year of the fracture than similar women who haven't suffered a hip fracture. One-fourth of those who were ambulatory before their hip fracture require long-term care afterward.

Interestingly, bone density is not a great predictor of fracture risk. Half of people with low bone density never suffer an osteoporotic fracture. Yet a considerable number of people with medium or high bone density unexpectedly fracture. Even if bone is dense, strong and resilient, it must also be able to heal and repair itself.

Boning up on osteoporosis

Susan E. Brown, Ph.D., CCN, of East Syracuse, NY, is a medical anthropologist and certified nutritionist. She holds a Ph.D. from the University of Michigan and is the recipient of two Fulbright-Hays Scholar Awards, an Organization of American States Research Fellowship and is a member of Sigma Xi, the honorary Scientific Research Organization of North America.

Dr. Susan E. Brown consults extensively on socioeconomic, cultural, educational and health issues. She has taught in North and South American universities and authored numerous articles in academic journals and popular magazines. Currently, Dr. Brown directs the Osteoporosis Education Project and the Nutrition Education and Consulting Service in East Syracuse, New York, where she conducts primary research and lectures on osteoporosis prevention and reversal. She also teaches the *Better Bones, Better Body Program*, a holistic, natural program for rejuvenating bone health.

Dr. Brown suggests that we rethink osteoporosis from a broader anthropological perspective. She says we will find that osteoporosis is somewhat different than we might think. If we take a more holistic perspective, we can see the true nature of osteoporosis.

Oeteoporosis is Not	**Instead, Osteoporosis is**
Thin Bones Only	Thin and Imperfect Bone Tissue
Normal Bone Loss	A Degenerative Disease of the Entire Body
Common Worldwide	Common only in the West
Only for Women	A Disease of Both Genders
Only for the Elderly	Increasingly Common in the Young
A Disorder Isolated in Bones	Result of Long Term Imbalances

Osteoporosis entails more than just weak bones. Mineral structures do give density to bones, true, but these mineral crystals are embedded in a living protein matrix which gives bone strength and resilience. This active connective tissue collagen matrix needs many assorted nutrients for its maintenance and repair.

In fact, cross-cultural studies from around the world do show that most people lose some bone mass as they age. But the remaining bone should be healthy and capable of constant self-repair. Normal aging bone loss should leave one with bones dense and robust enough to tolerate the daily stresses of life.

But in osteoporosis, bone weakness goes beyond normal aging. In this abnormal condition, bone becomes exceptionally thin from mineral loss and poor quality protein matrix.

The perception that low calcium and lower estrogen levels at menopause cause bone loss is, at best, only a part of the story. On the contrary, many countries have lower calcium intakes than the U.S., yet osteoporosis is less widespread in those locales. For example, the average Japanese calcium ingestion is only 540 mg, while Americans have twice as many hip fractures as the Japanese.

Medical authorities define several types of osteoporosis. Postmenopausal Osteoporosis (Type I) is associated with the onset of menopause and an increase in bone resorption, while bone tissue formation increases to a lesser degree. Women may lose up to 20 percent of bone mass in the five to seven years after menopause. The increase in osteoclast activity results in bone loss. Type I typically creates two types of fracture- vertebral and hip.

Age-Associated Osteoporosis (Type II) occurs in both men and women over 65 with bone loss over an extended period time (the 35 years since peak bone mass at age 30 to 35). As the body gradually fails to process

adequate vitamin D, secondary hyperparathyroidism sets in. Type II usually produces hip fracture and dorsal kyphosis ("dowager's hump").

Secondary osteoporosis is caused by an agent or disease, such as bone tumor, hyperthyroidism or inadequate gonadal function.

One of the most common causes of osteoporosis is steroid drugs. These anti-inflammatory agents, consumed mainly by women over 55 years of age, produce 20% of all osteoporosis. And approximately 11% of all hip fractures are attributed to the use of mood-altering medications. Certain other drugs also increase the risk of osteoporosis. Thyroxine, anticonvulsants, loop diuretics (furosemide) and long-term heparin are culprits. Long-acting psychotropic drugs like Valium and Librium bring a 70% to 80% greater risk of hip fracture from falls. Of course, any measure to reduce falls is a good idea.

Twenty percent of American osteoporosis patients are men. But worldwide, about 30% of hip fractures occur in men and one out of eight men over 50 will experience an osteporotic fracture in his lifetime. Two-thirds of those men with have one or more secondary causes, such as low testosterone or steroid use. More men than women die after a fracture, and gender is a strong predictor of mortality. Fractures in men account for about 20% of the total cost of osteoporosis in the United States.

Osteoarthritis (OA), or *degenerative joint disease* (DJG) is the most common form of joint disease. The disorder affects an estimated 20.7 million Americans, most of whom are over the age of 45. Osteoarthritis most often affects the knees, hips, spine, and hands, and sometimes other joints. Osteoarthrits and oeteoporosis are closely related, and the same people often have both conditions. Herbalists would describe both as "cold, dry" conditions. Thin people tend to get them.

In primary OA, no obvious causative factor can be identified. In secondary OA, the arthritis appears be the result of trauma, repetitive joint use, congenital or developmental defects, or metabolic disorders. Symptoms of OA include pain, stiffness, and reduced range of joint motion. In advanced cases, there may be significant disability. In the United States, 100,000 people are unable to walk because of severe OA of the hip or knee.

Previously, OA was thought to be a progressive, degenerative disorder, and was widely known as "wear and tear arthritis." It was presumes that everyone, if they lived long enough, would fall prey to OA. It is now known, however, that of the disease can be arrested or reversed. Drugs are often effective for symptom relief, but they don't slow the progression of the disease. Of course, they also have significant side effects. Several herbal medicines have been shown to be effective at relieving the symptoms of OA, and there is evidence that some of these medicines may improve the course of the disease.

Recent evidence changed the thinking about disease progress of OA. We now know that the joint cartilage of patients with OA is highly metabolically active. The damaged cartilage tissue actually tries to remodel and repair itself.

Though once thought to be impossible, arresting or reversing the disease occurs spontaneously in some OA patients. Pharmacological treatment of OA mainly involves nonsteroidal anti-inflammatory drugs (NSAIDs) and pain relievers. These drugs relieve symptoms, but they are far from perfect. NSAIDs, especially, cause serious side effects, including ulcer and liver or kidney failure. Neither of these types of medications cures the underlying cause of the disease. Evidence suggests, in fact, both in animals and in humans, that NSAIDs may actually accelerate joint destruction.

According to Dr. Brown, osteoporosis is really caused by the sum of numerous bone-depleting factors. She says that the best ways to prevent and treat osteoporosis is through a program that allows nature to restock the nutrients depleted by unhealthy lifestyle and diet. You may think of osteoporosis as a woman's disease, but bone health is critical for everyone. With a few obvious exceptions limited to women, most remedies can be used for both genders.

You know, it seems like people come to holistic practitioners to ask them to take away the symptoms of living busy, malnourished lives, so they can return to their busy, malnourished lives. But you can't approach osteoporosis that way. It is the end stage of a long degeneration, and you must have a lifelong plan to keep your bones healthy. Look at osteoporosis as an invitation to build a better body at any age.

Develop Healthy Bones

Your bone is not a hard and rigid structure. Rather, it is complex, living tissue that provides structural support, protects vital organs, and banks the calcium for bone density and strength. Bones are constantly changing, and they can heal. Before 30, you build and stock up bone mass efficiently.

As part of natural aging, your bones break down faster than new bone minerals can be formed. Your bones are like a bank account. The critical years for making deposits in the bone account are up to about age 30. Strong, healthy bone is continually recycled through bone remodeling, which includes resorption and formation. *Osteoclasts*, bone-resorbing cells, remove old bone by excavating small pits on the bone surface, releasing collagen and other proteins (Cross-linked N-telopeptides, NTx) and minerals in the bloodstream.

After the osteoclasts have done their job, cells called *osteoblasts* secrete protein and deposit new tissue. Bone mass remains constant when resorption and formation are in balance. Remodeling replaces approximately 20% of bone tissue throughout the skeleton annually. Osteoporosis occurs when resoprtion exceeds formation, producing a net bone loss.

Your skeleton grows most rapidly during infancy. Insufficient prenatal nutrition can lead to infants born with low bone mass. Breast-fed infants have greater bone-mineral density than those not breast-fed.

To deposit a healthy balance in your bone bank account:

- Breast-feed their infants as long as possible
- Vigorous exercise each day
- Eat fresh fruits, vegetables, beans and nuts in abundance.
- Limit sugar and processed foods
- Intake at least the RDA of all critical nutrients
- Refrain from smoking, recreational drugs and extreme dieting

Perimenopause, several years before the last menstrual period, can bring dramatic, puberty-like hormonal fluctuations- and considerable bone loss. Many, if not most, perimenopausal women drop spinal bone density at almost 2% per year. As a guide for future bone status, obtain baseline bone-density measurements. Compare that bone density with a urinary test of bone breakdown to see if your bone is stable or being lost.

To make it through this phase with the least bone loss, eat a mineral-rich alkaline diet (fruits, vegetables, nuts, seeds and lentils), do consistent strength-building exercise at least three times a week, and establish a program of appropriate dietary supplements.

Hormone replacement therapy (HRT) is sometimes used for postmenopausal bone protection, but Dr. Brown at The Osteoporosis Education Project concludes that for the average woman the risks of HRT outweigh any benefits. In 1992, a scientific study reported that postmenopausal women on estrogen replacement therapy gained no bone mass, but women using estrogen and a broad-spectrum multivitamin/mineral containing 26 nutrients, including 600 mg of magnesium and 500 mg of calcium citrate, gained 11% bone mass.

If you choose to pursue hormone therapy, have your levels checked to make sure the dose is correct. Use a urine test for bone resorption to verify that you do not have bone breakdown. If you diet, use bone-resorption tests to make sure you are not increasing bone loss.

Bone Mineral Density Measurements

Specialized tests- bone mineral density (BMD) tests- measure bone density in a variety of sites of the body. BMD is also correlated with fracture risk. BMD tests employ small amounts of radiation to assess the bone density of the spine, hip, wrist, heel, or total body. Measurements taken at the spine and hip are considered more sensitive than the wrist and heel. BMD at peripheral sites may still be normal when already significantly low at central sites.

DEXA scanning is usually performed at an outpatient facility, with the patient dressed. Radiation is less than that used for a chest X-ray.[5]

One test can't tell you how fast you are losing bone. BMD tests take sequential measurements (up to 24 months apart) to monitor bone loss of as little as 3-5% over time.

Currently, DEXA (Dual Energy X-ray Absorptiometry) is the most reproducible and accurate of the BMD techniques, and is the clinical method of choice. Test results reflect two standards: age-matched and young normal. "Age-matched" compares the BMD reading to the BMD expected in someone of the same age, and the "Young normal" compares the BMD to the estimated peak bone density of a healthy young adult. From a

clinical perspective, either a BMD measurement of more than 2.5 standard deviations (SD) below the "young normal" or a previous fragility fracture is accepted as osteoporosis.

Other common versions:

- pDXA (Peripheral Dual Energy X-ray Absorptiometry)- wrist, heel or finger
- SXA (single Energy X-ray Absorptiometry)- wrist or heel
- QUS (Quantitative Ultrasound) uses sound waves to measure density at the heel, shin bone and kneecap
- QCT (Quantitative Computed Tomography)- spine
- pQCT (Peripheral Quantitative Computed Tomography)- wrist
- RA (Radiographic Absorptiometry)- X-ray of the hand and a small metal wedge to calculate bone density

Biochemical markers are bone metabolism by-products of that are excreted in urine, which provide measure of bone turnover. They give immediate information on changes in bone metabolism as soon as three to six months, so you will know if your treatment plan is working.

The OSTEOMARK® test measures cross-linked N-telopeptides (NTx) of bone collagen. Another is the deoxypyridinium collagen crosslinks test. In adults, high levels suggest current excessive bone loss.

Roberta Lee, M.D. is the medical director of the Center for Health and Healing at Beth Israel Medical Center in New York City. She practices complementary medicine with a specialty in herbal therapeutics. Dr. Lee sees a lot of osteoporosis at her clinic. She reminds us that hypochlorhydria is common in aging people. To check out the possibility of urinary calcium loss, she suggests a test of NTx. Measuring the day's second urine, NTx is an assessment of urinary excretion of calcium, according to Dr. Lee. "If NTx is out of the normal range, it might indicate calcium loss from bone, and a person might be at risk. It is an inference of osteoporosis."[6]

There is no medical cure for osteoporosis, but a number of medications are approved by the FDA for postmenopausal women to prevent and/or treat osteoporosis. A number of drugs limit bone breakdown and reduce fracture risk. These, include Fosomax, the selective estrogen durg Estiva , the thyroid hormone Calcitonin, and others.

Estrogen and Hormone Replacement Therapy

Women all over the world experience a lowering of estrogen at menopause, but not all women experience osteoporosis. Attributing osteoporosis to the natural lowering of estrogen at menopause is too simplistic. The fact is that Mayan Indian women, Bantu women of Africa and the Japanese all have lower estrogen levels than US women, but they all experience much fewer fractures than US women.

Only a third of menopausal women in the United States actually try hormone replacement and half eventually drop it. Still, there are 10 million women every year opting to take the hormones, and that's big business. Wyeth-Ayerst's postmenopausal synthetic estrogen, Premarin®, is the nation's best-selling prescription drug.

HRT means you increase your risk of several kinds of cancer, according to many studies. According to Judyth Reichenberg-Ullman, N.D., of Seattle, Washington, other possible side effects include liver and gallbladder disease, high blood pressure, blood clots, strokes and depression. Progesterone is used in HRT to counter some of these effects, but the synthetic progesterone that is almost always used has side effects of its own.

The three-year Postmenopausal Estrogen/Progestin Interventions (PEPI) Trial was the first large-scale study to include natural progesterone. In this study, 34 percent of women on unopposed estrogen (without accompanying progesterone) developed endometrial hyperplasia, considered a precursor to endometrial cancer.

Many breast cancers have been linked to high estrogen levels or estrogenic activity; estrogen has certainly been determined to play a role in some breast cancers. And a number of studies of demonstrate increased breast cancer risk in postmenopausal women who take estrogen.

To combat the endometrial cancer risk, doctors and drug companies began recommending the combination of estrogen and progesterone. That was when ERT became HRT. However, while progesterone (or, actually, Provera, a synthetic progestin) reduces estrogen's cancer-causing risk, it may increase breast cancer risk as well as depression, "weight" gain and high blood pressure.

Margo Woods, a Tufts University School of Medicine nutritional biochemist who is studying diet's influence on menopause, is quoted as saying "We're concerned about the literature saying that women at risk of

breast cancer should not take hormone replacement therapy. Our feeling is that, ideally, almost no woman should be on it."

Another issue to consider is the fact that synthetic estrogen replacement in HRT does not actually match that which is produced by your body; therein may lie some of the risks. Hormone "replacement" therapy implies that you are replacing your body's own hormones with the real thing, but you're not. These synthetics are drugs that mimic the real thing.

As noted women's physician Dr. Kaaren Nichols, M.D., explains: "A woman's body has three kinds of estrogen: estradiol, estriol and estrone. Estrone is the kind you get in hormone replacement therapy—basically, Premarin®. It's made from horse urine, because it's easy to make and available in large quantities. This is not the same as what's produced by the human ovary.

"Now, that's the kind they can get from horse urine, so that's the kind you get. What about the other two estrogens? You're replacing only one of three kinds your body makes." Dr. Nichols also notes that horse urine is composed of a high percentage of metabolytes that the human body can't use and must get rid of, placing stress on the liver which is saddled with this processing task.

Dr. Nichols asserts, "Premarin® is patented by a huge pharmaceutical company that has a lot of power and has gotten it established as the sole estrogen used for replacement therapy. No one gets estriol because it's not patentable. It's a natural substance. There's no money in it. Estriol is also the only estrogen proven not to cause breast cancer. And you can't get it. A few pharmacies will compound it, but that's it."

There is evidence that estriol is not only not carcinogenic, but actually anticarcinogenic. According to the Journal of the American Medical Association, studies have shown that a high estriol level protects against the tumor-producing effects of the other two estrogens—estrone and estradiol. One study linked high urinary levels of estriol to early first-child birth and low incidence of breast cancer. Estriol's safety advantage may be due to the fact that it does not lose its identity when given orally (i.e., it remains estriol).

Chi-Ling Chen, Ph.D., and colleagues, in a recent study in the *Journal of the American Medical Association*, showed that all forms of breast cancer were increased by 60 to 85 percent in women who were long-term (57 months or longer) users of HRT.

At least one study has found that postmenopausal women who had adequate calcium in their diets, who exercised and who averaged one alcoholic drink or less a week had bones that were just as strong as those of women who took hormones.[7]

On Oct. 15, 2002, the government said that women should not use estrogen and progestin in hopes of preventing bone loss. The clear risks of use outweigh the few long-term benefits, such as bone strength, the U.S. Preventive Services Task Force said.

Very recently, HRT fell out of favor with an expert committee. An international team of women's health experts is discouraging the use HRT for many postmenopausal conditions. As evidence from high-quality clinical trials accumulates, coronary heart disease, fractures, depression and urinary incontinence are losing favor as valid indications for it.

In 2002, previous recommendations seem quaint. At a symposium sponsored by the Office of Women's Health Research at the National Institutes of Health, experts on the Committee of the International Position Paper on Women's Health and Menopause outlined the dramatic shift in clinical practices for menopause. During the 4 years it took to compile the evidence that fills the report, dozens of experts drew similar conclusions about fractures involving osteoporosis. "[A]ggressive pharmacotherapy should be reserved for women who are at high risk" for fractures, according to report's executive summary. For more information on the report, go to the Office of Women's Health Research Web site, http://www4.od.nih.gov/orwh .

HRT helps prevent bone loss, said Foundation, but whether that benefit equals reduced fracture risk remains questionable. Furthermore, newer trials show that HRT must be taken continuously to maintain bone mass, not just for 5 or 10 years after menopause, as previously assumed. Given the hormone's risks, including a three-fold increase in blood clots, women are usually better off with other therapies, according to the report.

Stay Active for Healthy Bones

"Use it or lose it"- that's the secret for bone mass. Thousands of studies now confirm that bone grows in strength in response to the strain put upon it. But to begin rebuilding bone density, you need rigorous weight-bearing exercise. Even walking or exercise in a wheelchair helps bone mass.

At least four hours a week of regular physical activity and strength-building exercises help to maintain bone. Tufts University scientists found that strength-building exercises could make a 95-year-old as strong as a 50-year-old and a 65-year-old as physically able as a healthy 30-year-old.[8]

A 1995 study, also from Tufts, looked at weight lifting for 60 year old women and found that they still gained substantial, continual increases in strength.[9]

University of Washington scientists had women participating in regular exercise three times per week, with each supervised session lasting one hour. Strenuous exercise (aerobic and strength-building components) was paced at 70% of the maximum oxygen intake. Weight-bearing, strength-training activities (walking, jogging, stair climbing, weight lifting) and non weight-bearing aerobic activities (cycling, rowing) plus a total of 1500 mg calcium a day (from diet and supplements) and 50 IU vitamin D succeeded in building bone.[10] Women gained a 5.2% bone mineral increase after 9 months, and a 6.1% increase after 22 months.

If a person is suffering from osteoarthritis (OA), the last thing she often feels like doing is exercising. But, experts say, that's precisely what could help ease the pain.[11] About 50 percent of OA patients swim or exercise in other ways.

Through exercise, patients can improve overall health and fitness, as well as arthritis symptoms. Regular exercise can help manage pain, as well as keep joints moving. Other positive effects include strengthening muscles around joints, increasing energy, improving sleep, controlling weight and strengthening the heart. Authorities suggest that the program should include range-of-motion exercises to keep joints flexible. It also should include fitness exercises, such as water exercise or walking.

Regular exercise may be the best way to prevent pain from occurring in arthritic joints in the first place. Those who exercise regularly also recover faster from existing arthritic pain.[11] Without movement, general deconditioning leads to increased fatigue in even normal day-to-day activity, resulting in more pain and a downward spiral. Further, exercise triggers a process that protects and helps repair joints damaged by arthritis. It increases the pumping action of synovial fluid that protects, provides nutrients and takes away waste products from the articular cartilage.

A regime of physical therapy and home workouts considerably reduces the pain and stiffness of osteoarthritis of the knee, says to a recent study from U.S. military researchers. The combination of physical therapy and modest exercise also helped delay or prevent knee replacement surgery, say the scientists.[12] The program consisted of hands-on physical therapy twice a week for a month, plus custom exercise routines tailored to each patient. At one and two months of follow-up, patients who received the bodywork showed significantly fewer signs of arthritis than those who had no treatment. These patients could walk about 13 percent faster and they had more than a 55 percent improvement in pain, function and joint stiffness.

Yoga and relaxation techniques are used by nonmedical practitioners to help ease musculoskeletal symptoms. A recent study looked at the effect of yoga on the hands of patients with OA. Individuals with OA of the hands were assigned to receive either the yoga program or no therapy. The scientists measured pain, strength, motion, joint circumference, tenderness and hand function. The yoga treated group improved significantly more than the control group in pain during activity, tenderness and finger range of motion. This yoga program was effective in providing relief in hand OA, said the researchers.[13]

Patients with OA should continue to do activities that don't injure the joint. An activity such as stationary bicycling can keep the joints active and mobile, as well as conditioning the thigh muscles to protect the knee joint against further injury. Swimming is a good alternative activity, and the weightless water environment can contribute to the perfect exercise. OA sufferers should avoid high-impact exercises that put sudden sharp or very high forces across the joint. These include running and jumping.

Acid Balance and Healthy Bones

In any healthy person, the oxidation and utilization of foods creates a wide range of chemicals that immediately impact the acid-base balance (the pH measure of the tissues) When carbohydrates and fats oxidize in the cells, the result is an excessive quantity of carbon dioxide (CO_2). CO_2 is a toxic waste product, so this substantial production of CO_2 is potentially stressful, even toxic, to the whole body. Depending on how efficiently the respiratory system- one way out of the body for the waste CO_2 - is functioning, a trace amount of CO_2 will be exhaled out through the lungs. Contrary to what most of learned in high school, most of the remaining concentration of CO_2 does not in fact go out into the air, but will combine with water (H_2O) in the body's

tissues to produce carbonic acid (H_2CO_3), pushing the pH of the blood toward the acid range. To prevent this acid from immediately damaging tissues and disrupting cellular processes, this carbonic acid is buffered by alkaline buffering compounds, including, especially, calcium from bone. It is critical that the body maintain blood acidity in very tight control. The body will draw upon buffering compounds to the point of drastically exhausting its reserves to preserve this proper blood pH. Eventually, through a complex set of metabolic pathways, the carbonic acid is finally eliminated through the kidneys.

The body's three buffer systems that are the most metabolically active and therefore the most significant:

- The bicarbonate/carbon dioxide system (Bicarbonate buffers the acids, resulting in carbonic acid being formed from water and CO2)
- The extracellular system (mainly made up of the proportional concentration of phosphate)
- The skeletal bone system (bones continuously release their alkaline minerals -magnesium, manganese, sodium, potassium, zinc, boron, copper, strontium, calcium

Much of the carbonic acid that ends up in the blood from the buffering process finds its way into the red blood cells, where it becomes buffered by the potassium salt of hemoglobin. Another reason to keep potassium intake high!

Proper pH balance is particularly important in sustaining correct mineral status, and consequently, bone health.

"The most important of all bone-affecting balances, however, is the least well known. This equilibrium involves the delicate chemical balance of acid and alkaline within our bodies," says Dr. Brown. "Just as acid rain in the environment damages our external ecosystem, so internal acids damage our internal ecosystem. Without knowing it, most of us consume diets high in acid-forming foods."

According to Dr. Brown, this results in chronic, low grade acidosis. Dr. Brown says, "We see chronic metabolic acidosis as the major hidden cause of osteoporosis. Even a slight tilt toward acid produces osteoporosis. The average American labors under chronic acidosis." The body then has to take valuable alkaline salts of bone building minerals (magnesium, manganese, sodium, potassium, zinc, boron, copper, strontium and calcium) from bone tissue and use them for buffering those internal acids. Your skeleton, in addition to holding you up, is the reserve storehouse for these minerals, so they are withdrawn to fit the need. Dr. Brown: "Bone is the great mineral storehouse Nature never thought we would need. Acidosis <u>is</u> mineral deficiency- especially potassium."[14]

William Lee Cowden, M.D. practices integrated medicine at The Conservative Medicine institute in Richardson, Texas. Dr. Cowden says that acid neutralizing calcium comes first from blood and soft tissue. He says that this initial depletion of muscle calcium can cause muscle cramps. When calcium begins to be pulled from bone, it can end up being deposited as calcium salts in joints, eventually causing degenerative arthritis.[15]

Researchers at The Osteoporosis Education Project realized that our internal acid load is a major factor behind the osteoporosis epidemic. To counteract this, they encourage a diet high in alkalinizing foods, such as fruits, vegetables, nuts, spices, herbs, seasoning, seeds and pulses, including lentils.

Dr. Brown says, "People in other cultures, who eat closer to this type of diet, do better with less calcium, because less mineral is wasted for buffering."[16]

Approximately 6 to 8 percent of us are naturally high-alkaline producers. These folks are natural peak performers. They have excellent lung capacity. Their digestive systems function well. They have strong skeletons with large reserves of alkaline minerals in their bones. Believe it or not, they thrive on stress, can eat the standard American diet with less ill effects, require hard endurance exercise and rarely get ill. To top it off, they also age more slowly. Their natural edge on the rest of us shows up in extraordinary success. Where do I sign up?

Those are the fortunate few. But even if you didn't start with a naturally high-alkaline metabolism, you can regain the pH balance in your body. Would you like to regain your physical stamina, your optimism, and ability to be sociable? To say nothing about tolerating the stresses of career and family. When you're in pH balance, you can again think clearly. You resist disease. Even if illness and injury should strike, you recover rapidly. It just takes some study and determination to change the way you always do things.

Sugar and Bones

All authorities in the field are unanimous in their condemnation of refined carbohydrates, especially white sugar. Refined carbohydrates increase acidity and worsen the typical Western metabolic problem.

When you eat the typical American meal, the sugar rush comes at you so fast you nearly have to duck. The average American eats over 20% more sugar (about 25 more pounds per person per year) than she did in 1986. According to the Center for Science in the Public Interest, the current estimates for the average American's consumption of caloric sweeteners (sugar, corn syrup, and the like) is around 152 pounds a year.

Alan Gaby, M.D., is one of the pre-eminent holistic physicians in the United States. Dr. Gaby teaches at Bastyr University in Seattle, where I also teach. He is the author of many professional publications for physicians, and *Preventing and Reversing Osteoporosis: What You Can Do About Bone Loss--A Leading Expert's Natural Approach to Increasing Bone Mass.*

According to Dr. Gaby, eating sugar may also deplete our bodies of calcium. Numerous studies over the years have shown that a high sugar diet causes urinary calcium to increase.[17] [18] A recent French study showed that single chocolate bar, containing 55 grams of sucrose produced a striking increase in blood triglycerides and urine calcium.[19]

To make it worse, when 100 grams of sugar was given to people with a history of calcium oxalate kidney stones, or to their relatives, they excreted even more calcium. In your body, 99% of the stored calcium is in the bones, so the increase in calcium excretion caused by sugar probably means that it is leaching from bone. Therefore, this information suggests that a diet high in sugar may lower the calcium content in bone. To add to our scientific information, a 1987 study investigated the idea that eating sugar, known to increase blood insulin, would inhibit reabsorption of calcium through the kidney. In other words, the calcium would be excreted in the urine. You guessed it. That's just what happened- along with zinc and sodium, two other alkaline minerals.[20]

A significant study came out in *The American Journal of Clinical Nutrition* in 2002 that again gave support to this connection. In the Framingham Osteoporosis Study, scientists with the US Department of Agriculture Human Nutrition Research Center on Aging, at Tufts University in Boston, studied 907 older Americans to determine dietary factors connected with bone mineral density (BMD). They concluded, "High candy consumption was associated with low BMD in both men and women." In fact, candy consumption was the most substantial predictor of low BMD in the study.[21]

Other whole sweeteners- maple syrup, molasses, barley malt syrup, rice bran syrup- are considered to be far less acid producing, because they contain less total sugar, and they come with an assortment of alkalinizing minerals built in. But there is a limit. Ultimately, all carbohydrates break down into acids. Be moderate with your need to use sweeteners- they really are just treats, and not necessary on a daily basis.

Protein

According to Dr, Gaby, the American diet tends to contain too much protein. He claims that studies have indicated that excessive protein in the diet may encourage bone loss. When you eat more protein, the urinary excretion of calcium rises. The digestion of protein liberates acidic breakdown products. As we know, calcium is mobilized to buffer these chemicals. Dr. Gaby maintains that science has shown that people who eat a vegetarian diet have stronger bones later in life than those who eat meat.

The amino acid *methionine*, which is abundant in animal protein, is converted in the body to *homocysteine*. It now looks like this chemical, which has become known as a cardiovascular disease risk factor marker, is capable of causing bone loss.

Animal flesh contains phosphorous, a necessary nutrient. But Dr. Gaby says that excessive consumption contributes to osteoporosis. One of the breakdown products, phosphoric acid, must be buffered by calcium. This probably explains in part why excessive protein has an adverse effect on bone.

Dr. Brown's China Health Project suggests that we eat too much fat and protein- two noted anti-nutrients. As a group, we consume two and three times the ideal. On average, a woman would do well to limit fat to 35 to 40 grams per day, and a man 38 to 50 grams. As for protein, Dr. Brown feels that many of us (but not all of us) consume twice the amount we should. The U.S. RDA for protein averages 50 grams for females and 63 for males. Still, a large percentage of the elderly consume too little protein, and this also damages bone.

Beans, peas and other legumes, including lentils, are an excellent source of protein. If you include these in your diet, and also eat whole grains such as whole wheat, oats, corn, barley, millet, buckwheat and, rice,

these two food groups together will provide the entire assortment of essential amino acids you need for protein. Legumes are high in the alkaline minerals calcium, magnesium, potassium, iron, copper, zinc and vitamin B-complex, all nutrients that benefit strong bones. As an added benefit, legumes are high in soluble fiber, the kind that lowers cholesterol.

Having said this, remember- it's still necessary to consume enough protein. Dr. August cites several studies correlating dietary protein and bone density. Decreased density of the femur density is associated with the amount of protein in the diet. The bone is composed of a very important protein matrix, in which minerals are deposited. Unless there is sufficient dietary protein, the body cannot preserve its protein matrix. So, as with most things, balance is the key- we need neither too much nor too little protein.

But wait. Is something a fowl here? Chicken- the flesh health nuts love to recommend. Ideas are all over the place on this one. It is clearly less acid producing than red meat, but to what degree, no one can agree- probably somewhere between beef and fish. So why make a flap over it?

Dr. Brown maintains, "Meat is fine. It just has to be balanced by more potassium, found in vegetables, at least two servings each at lunch and dinner, so that the pH balance is maintained. The average American can buffer about 50-60 grams per day of protein. Most are eating well over that, and 100 grams per day is common."

Dr. Brown mentions that the recently popularized Paleolithic diet does include more meat than most Americans currently eat, but that proponents of this approach claim that it actually is an alkalinizing diet on balance, because it incorporates a very large amount of high potassium fruits and vegetables.

From the pH point of view, fish flesh may be a lesser evil. Dr. Lark, even while suggesting eliminating red meat, points out that fish is less acidic and contains the beneficial anti-inflammatory oils. In her program, she includes it as an alternative to red meat as a source of protein.

Fish is acid producing- just less so that red meat. Depending on where you fall in the pH spectrum, you can make a decision about whether, and how much, to include in your diet.

Treat Brittle Bones with Diet

The recent Framingham Osteoporosis Study, published in 2002, measured bone mineral density in 907 subjects. The scientists also concluded that a diet high in fruit and vegetable intake appears to be protective in men. In fact, the men who ate the most fruits and vegetables had the best bone mass in the study, regardless of other dietary factors.[22]

There is substantial evidence that a diet high in vitamin C, vitamin E, and beta-carotene can slow the progression of degenerative joint disease, by as much as 70%.[65] Of course, these nutrients are found in fruits, vegetables, whole grains, nuts, and seeds.

You will notice that fruit and vegetable diet, while dramatically beneficial, has not been the focus of recent media and public awareness perception, which has gone instead toward the importance of calcium. While calcium is no doubt important, especially in a culture that eats a high acid diet, we need to take a holistic approach toward the total diet. Clearly something as simple as getting the nationally recommended five servings of vegetables and fruits (which almost no American actually does) may be equally as important as calcium status when it comes to preserving bone density.

And some vegetables do contain considerable calcium- particularly carrot. Leafy green vegetables pack a substantial dose of calcium, as well as iron and magnesium.

Fruits have high potassium content. This alkalizing mineral, in bananas, oranges, berries, peaches, apricots and melons, offsets the drain on the alkaline minerals in your bones, and decreases fluid retention.

No matter what the methodology, a diet that concentrates on fruits, vegetables and whole grains is likely to accomplish one valuable goal: weight loss. And it's well established that losing extra pounds often leads to at least some joint pain relief. One study found that overweight women can "significantly lower their risk for developing osteoarthritis of the knee by losing weight."[23,24]

Overweight people have a higher risk of developing knee degeneration. They may be at elevated risk of hand and hip OA. Additionally, excess body weight accelerates disease progression. Increased joint stress attending obesity may explain the strong connection between obesity and knee OA risk, but it does not explain very well why obese people have a high risk of hand degeneration. Also, obese women are unexplainably at higher risk of knee problems than obese men. And studies of metabolic factors connected with obesity have not

explained these discoveries. There is a scarcity of data on weight loss as a treatment for bone and joint disease, but preliminary investigation implies that it is principally effective in knee conditions and that a small degree of weight decrease may even have positive effects.[25]

Coffee has a pH of about 5.0, so it's closer to neutral than many acid fruits, but compared to pH readings of over 6.0 for grains, and close to 6.0 for many vegetables, coffee may be too acid for many. Also, it contains caffeine. A recent study done by researchers from the Bone Metabolism Unit of Creighton University's School of Medicine found that elderly women with high caffeine intakes showed much higher rates of spinal bone loss than those with low intakes. Low tissue vitamin D levels made it worse. They looked at several risk factors, including calcium intake, caffeine intake and smoking. The factor that had the most severe effect was caffeine intake. Researchers reported that taking in more than 300 mg per day of caffeine (the amount in about 3 cups of coffee) accelerated bone loss in elderly postmenopausal women.[26]

Supplements for Osteoporosis

There is now no doubt that an aggressive program of well selected supplements can help bone density. Studies show improved bone-mineral status in perimenopausal women who consume more zinc, magnesium, potassium and fiber. Unfortunately, teenage girls take in on average only 68% of the RDA for calcium, so it's unlikely they will generate full bone-mass development. Teenage boys consume more calcium, but are still short on other essential bone nutrients, such as magnesium, of which 30% of all adolescents consume less than two-thirds the RDA.

Osteoporosis can even be stopped, even after a fracture. Women, with an average age of 84, were studied. They improved 2.7% in hip density and reduced their hip fracture rate by 43% with daily 1,200 mg tricalciumphosphate and 800 IU vitamin D. Controls lost 4.6% hip density and had 67% more fractures.

Calcium

Calcium and osteoporosis is a very confusing subject. Osteoporosis is a disease of many factors. Drugs, exercise, hormones, heredity, smoking, alcohol, weight fluctuation, inactivity, and dietary variables all affect bone mass and strength, and are seldom, if ever, properly considered in calcium trials. For example, bone density can be increased without decreasing fracture rate. Fracture risk is due to bone fragility, and what determines bone fragility in osteoporosis is largely unknown. Some studies look at populations with small calcium intake. Confoundingly, the calcium intake in The Netherlands, for example, is high, and so is osteoporosis. Higher calcium intakes do not correlate with protection from bone loss.[27] Dr. Brown maintains that the counties, like Peru, Sri Lanka and China, that have the lowest calcium levels in the diet, have populations that maintain lifelong healthy bones, while Americans have brittle bones. This, she says, is because Western populations have overburdened themselves with an excessive acid load, throwing pH out of balance.

So far, we don't know much about a very large factor in the calcium and osteoporosis connection-inefficient absorption and high kidney loss. About 25 percent of changes in calcium balance result from absorption differences, and 50 percent is a result of urinary loss. In calcium balance studies, calcium absorption in postmenopausal women varied as much as 61 percent. And 40 percent of those women could not absorb enough calcium to stay in positive calcium balance even when they took 800 mg daily. High salt and high protein diets, common in the United States, promote high levels of calcium excretion via kidney losses.

Calcium makes up 1-2 % of the adult human body. Over 99 % of that calcium content is found in the teeth and bones.[28] Calcium, magnesium, zinc, fluoride, and phosphorous work in tandem to build and maintain bones and teeth.[29] The ratio of calcium to phosphorous and magnesium are critical to the absorption into the bone. Proper calcium absorption depends on adequate amounts of vitamin D.

Adolescence is the time to build bone mineral mass. Teenagers who take in the RDA of calcium have better bone-mineral density than those who consume less, while those eating a few hundred milligrams more than the RDA do even better. Still, though, only 10% of American girls and 25% of boys even meet the RDA for calcium.

A very large overview study of calcium and bone mass found beneift. Using 49 separate studies, the literature was consistent in showing a 1000 mg/day calcium supplement improved bone mass and would prevent loss of one percent of bone mass per year in postmenopausal women not on estrogen.

The term *elemental calcium* refers to the amount of calcium in the molecule. This can be tricky, because some brands list only the total weight of each tablet, not the actual amount of calcium.

Percentage of calcium in types of calcium	Amount to take to equal 200 mg elemental calcium
Calcium carbonate (40 percent)	500 mg
Calcium citrate (21 percent)	950 mg
Calcium lactate (13 percent)	1,550 mg
Calcium gluconate (9 percent)	2,200 mg

NIH "Optimal Calcium Intake"

Age	**Milligrams Calcium Per Day**
Birth to 6 months	400 mg
Infants 6 to 12 months	600 mg
Children 1 to 5 years	800 mg
Children 6 to 10 years	800 to 1,200 mg
Adolescents and young adults (11 to 24)	1,200 to 1,500 mg
Women 25 to 50	1,000 mg
Pregnant or lactating women	1,200 to 1,500 mg
Postmenopausal women on ERT	1,000 mg
Postmenopausal women not on ERT	1,500 mg

Lynne August, M.D. received her Medical Degree from Washington University School of Medicine in 1973. She is the founder and director of Health Equations, a service that offers health professionals and patients an innovative, objective evaluation of a standard blood test. Dr. August was a clinical researcher at the Institute of Applied Biology in New York, where she participated in research on non-toxic therapeutic lipids. She also draws upon her training and practice in Ayurveda.

The Health Equations evaluation tells each patient which metabolic track they are on and where it will lead- even decades from now. Based on thirty years of clinical medical research, the Health Equations Blood Test Evaluation provides a snapshot of a person's nutritional status with concise, personalized interpretations and recommendations. Follow-up evaluations examine a person's response to the recommendations.

Dr. August has an excellent system of classifying nutrients and body processes into two categories- anabolic and catabolic. This perspective is valuable in helping us get a handle on where to start with classifying foods and minerals.

According to Dr. August, many individuals are calcium deficient because they are not absorbing the calcium from the food they eat. She says patients lose calcium for a number of reasons:[30]

- When tissue and cell cholesterol is high, they lose calcium in their urine
- When the tissue and cell cholesterol is low, calcium is bound up and not available to the cells
- They are eating processed foods
- They are not eating or digesting enough protein and/or fat
- There is not enough calcium in the diet

There's a lot of debate about which type of calcium is best, and it gets pretty technical. Companies that produce different forms trot out studies to support their preparation. Dr. Ralph F. Shangraw, Chairman of the Department of Pharmaceutics at the University of Maryland School of Pharmacy, tested more than 80 calcium

carbonate supplements and determined that the "effectiveness of over half of the tablets is questionable" due to their slow rate of disintegration and dissolution.

Calcium carbonate - the form found in antacids - is less easily absorbed than more modern forms. But it's cheap, and has a high percentage of elemental calcium.

Calcium hydroxyapatite is a source of concentrated calcium in the form in which it occurs in living bone, with a 2:1 calcium to phosphorus ratio and additional natural trace minerals, matrix proteins and glycosaminoglycans. Initial studies looked promising, although better forms have since been developed. In one study, a group receiving calcium hydroxyapatite was compared with another group receiving calcium gluconate and a control group. Over the 14 month period, the control group lost significant bone and the gluconate group stayed level, while there was a significant bone gain of 11.6% in the calcium hydroxyapatite group.[31]

A cutting edge form, calcium citrate malate, appears to be winning the calcium absorption wars, at least for now.[32] Studies show that up to 42% of the calcium in calcium citrate malate is absorbed, compared to calcium carbonate at about half that rate. Scientists looked at all commercially available calcium forms (calcium oxalate, hydroxyapatite, calcium carbonate, tricalcium phosphate, calcium citrate, calcium citrate/malate and bisglycinocalcium). Calcium citrate malate was the most soluble and absorbed better.[33]

In another study of women aged 21 to 30, 250 mg of calcium citrate malate, given three times per day, was absorbed better (37.3 percent versus 29.6 percent, respectively) than an equivalent calcium carbonate supplement. In the same study, calcium citrate/malate was also absorbed better than calcium from milk.[34] Adolescents also did better with citrate malate than carbonmate.[35]

Vitamin D

Vitamin D functions as a hormone. It regulates the growth and development of many different cells, including maintaining strong bones. Postmenopausal women hospitalized for hip fractures were more likely to be deficient in vitamin D.

Mild abnormalities of increased secretion of parathyroid hormone (PTH) and decreased vitamin D production contributes to menopausal bone mass decrease. Vitamin D supplements suppress the increased PTH secretion, and increase intestinal calcium absorption. The need for vitamin D increases with age, but vitamin D deficiency is common among the elderly. Up to 80% of all hip fracture patients have vitamin D deficiency.

The body produces vitamin D when the skin is exposed to sunlight, so the elderly living in northern climates or exposed to little sunlight are at higher risk. For healthy bones, get daily direct sunlight. Take supplemental vitamin D at 800 to 1,000 IU per day.

Boron

Preliminary studies report that boron plays a role in the metabolism of calcium, phosphorus, and vitamin D. So far, though, research does not clearly demonstrate benefits in osteoporosis, but the implications are intriguing.

A 1986 study found that daily supplementation with 3 mg. of boron greatly reduced urinary excretion of calcium and magnesium. Another study on athletic college women showed that boron supplementation lowered serum phosphorus levels, and high phosphorus is another factor associated with osteoporosis.

Sulfur Compounds for the Bones and Joints

When degeneration starts to develop, the body responds by rebuilding healthy joint tissue. This natural process can delay the development of actual pain and loss of mobility for years. Glucosamine, which functions in cartilage formation and repair, is one of the best-known and most well researched nutrients for OA. Findings published recently in *The Lancet* suggested that the long-term combined structure-modifying and symptom-suppressing effects of glucosamine sulfate indicate that it could be beneficial in OA.[36] The medical director of the Arthritis Foundation called this a "landmark study providing evidence that glucosamine has an important role in the management of osteoarthritis."

Scientists speculate that the sulfur content of glucosamine increases its therapeutic effects. Patients with arthritis, particularly OA, have been shown to have lower than normal concentrations of serum and synovial sulfur.

Along with glucosamine sulfate, patients are also experiencing relief from two additional sulfur-containing supplements, MSM (methyl-sulfonyl-methane) and SAM-e (S-adenosylmethionine). MSM is known to pick up joint flexibility, diminish stiffness and swelling, enhance circulation and lessen pain, while the sulfur in SAM-e is used by the body in the production of proteoglycans and the manufacture of cartilage.

Herbal Medicine for the Bones and Joints

Because osteoporosis and related arthritis are among the leading ailments affecting aging baby boomers, the search for alternative methods of combating this looming duo is leading them to dietary supplements in droves. According to research conducted by *Prevention* magazine, 48 percent of consumers who regularly use specialty supplements are between the ages of 45 and 73. Arthritis/osteoporosis bestsellers glucosamine sulfate, chondroitin and soy isoflavones alone generated $414 million in sales last year.**Error! Bookmark not defined.**

In 1999, the two most celebrated introductions in the pharmaceutical arena were the COX-2 inhibitor drugs celecoxib (Celebrex) and rofecoxib (Vioxx). Used to treat the pain and inflammation of arthritis, their financial successes have already stimulated the development of natural alternatives.

COX-2 is short for cyclooxygenase-2. This key enzyme helps the body produce the inflammatory compounds prostaglandins and cytokines. The COX-2 enzyme is activated when tissue is injured or inflamed and is vital for fighting infections and healing injuries. When the body overproduces COX-2, though, the result is *chronic* inflammation and pain.

NSAIDs, such as ibuprofen and aspirin, ease inflammation by inhibiting the activity of COX-2. Certain herbs contain components that seem to safely and appreciably inhibit COX-2. Turmeric, holy basil (*Ocimum sanctum*) and green tea (*Camellia sinensis*) are examples.[37]

Noted herbalist James A. Duke, Ph.D., suggests more easy ways to up your intake of COX-2:

1. Replace some or all of your coffee with green tea and/or chamomile infusion. Spice them up with clove, lavender, marjoram, rosemary, sage and thyme.
2. Eat more cabbage, celery, chives, currants and rhubarb.
3. Use more celery seed, ginger and turmeric in your teas and cooking.
4. Enjoy grape juice or red wine for their resveratrol- better yet, enjoy stuffed grape leaves.[38]

Herbal medicine can be quite effective in treating bone disorders, and there is evidence that some of these medicines may improve the course of the disease. Scientific evidence is building for these remedies.

First, maximize your continued hormonal activity. The liver processes estrogen, so take herbs that support the liver. Liver herbs include burdock root, yellow dock and dandelion.

There are herbs that have estrogenic effects. They include dong quai, burdock root, blue cohosh, black cohosh (which has been studied extensively and is probably the most well-documented natural alternative to hormone replacement therapy), Chinese ox knee root, Chinese 3-edge root, sage, alfalfa concentrate, and motherwort. All of these are especially excellent for hot flashes. Blue cohosh is probably the best for hot flashes.

Michael Murray, N.D. feels the four most successful herbs for increasing estrogen activity are donq quai, licorice root, chaste berry and black cohosh.

Licorice root benefits the bones in children. It is sweet and quite tasty, but laxative. Small amounts should be used to bowel tolerance. Preliminary research suggests that long-term intake of black tea may improve bone mineral density in older women.[39]

Herbs for bone maintenance and mineralization include nettle, alfalfa, slippery elm, oatstraw and horsetail (these latter two are sources of silicon). Algaes are a rich broad-spectrum source of minerals as well. Ginger and cinnamon increase circulation to joints and turmeric is a joint builder.

Natural Hormone Approaches

Red Clover

This is a legume, which, like soy contains "phytoestrogens". Red clover has been found to contain over 125 compounds, but the isoflavones are thought to be responsible for the majority of its effects. Isoflavone extracts of red clover are used to treat menopausal symptoms and oeteoporosis, as an alternative hormone replacement therapy. While soy, another source of isoflavones, has been extensively studied, there are no high-

quality human trials supporting the efficacy of red clover. Yet practitioners report success, and use of these products seems logical. Preliminary studies look good.

One report, published in 2001, in the journal *Menopause,* illustrates that a red clover isoflavone combination was associated with a significant increase in HDL (good) cholesterol and a significant increase in the cortical bones of the arm after 6 months of treatment.[40]

Soy

Soy has received a great deal of attention for its apparent ability to benefit several areas of health concerns, from breast cancer to heart disease to menopause. In numerous studies, several compounds in soy have been shown to lower cholesterol, slow bone loss and block breast cancer activity.

Whole soy, soy products and soy isoflavones have been studied for various health conditions. Since genistein and other isoflavones have estrogen-like effects in the body, they are called "phytoestrogens". A small number of studies report benefits, especially in the lumbar spine. There is some safety concern with the use of isolated isoflavones. They may slightly lower lymphocytes, and there is a possibility that they interact with some drugs, in the manner as does grapefruit juice. Dr. Brown says that isoflavones modestly support bone health. However, she does not endorse the use of more than 50 mgs of soy isoflavones given potential risks. Perhaps it would be better to stick to whole soy products.

Studies show that feeding sulfur containing amino acids (cysteine and methionine), which are abundant in animal protein, to animals, causes a reduction in bone density.[41] Rats fed a diet of 15% soy protein as a control did not have this bone loss. Researchers theorize that homocysteine reacts with collagen in the body's connective tissues, hastening bone breakdown.

Horsetail *(Equisetum spp.)*

Studies indicate that silicon plays a role in bone development, may enhance bone mineralization, and may promote calcium deposition in bone. Horsetail, which contains silicon, is a traditional joint medicine. According to preliminary evidence, it may be an effective natural treatment for osteoporosis. In a 1999 Italian randomized trial, 122 women took placebo, no treatment, horsetail dry extract or a horsetail-calcium combination. After 40, 80, and 365 days, both the horsetail and calcium groups had a statistically significant improvement in bone density.

Ashwaganda root (*Withania somnifera*)

This herb has a wide range of effectiveness. Considered to be the main long-term stamina-enhancing tonic for men in Ayurveda, it can be taken by women, too. Ashwaganda is said in Ayurveda to build and solidify tissue growth- it is "anabolic." Since osteopoosis is a disease of "catabolism," or tissue destruction, ashwaganda seems like a good choice. Modern research is bearing this out.

The demonstrated effectiveness of ashwaganda in a variety of rheumatologic conditions may be due in part to its anti-inflammatory properties, which have been established in several studies.[42] One clinical trial supports the use of ashwaganda for arthritis. In a double blind, placebo-controlled cross-over study, 42 patients with osteoarthritis received a formula containing ashwaganda or placebo for three months. Patients were evaluated for one month, pretreatment, during which time all previous drugs were withdrawn. During both the pretreatment and treatment phase, pain and disability scores were evaluated weekly. The herbal formula significantly reduced the severity of pain and disability scores.[43]

Take ashwaganda at a dose of up to several grams per day for symptom relief. A maintenance dose is about 1 gram per day.

Salai Guggul gum (*Boswellia serrata*)

This herb has become well known in North America over the last decade for its pronounced effects on joint disorders. The medicine is a gum resin that exudes from the bark of a large branching tree that grows in semi-arid areas of South Asia. Extracts of this gummy exudate have been traditionally used in the Ayurvedic system of medicine as an anti-arthritic. In Ayurvedic herbalism, medicinal preparations of gum are known as "gugguls."

This gum contains components, boswellic acids, that inhibit inflammation producing substances, leukotrienes, in the body. In fact, boswellia gum inhibits inflammation through several mechanisms in the body. Scientific experiments showed that the herb "was found to exhibit marked sedative and analgesic effects."

Boswellia gum treats pain, another reason it should be used for advanced osteoporosis. While Ayurveda recommends this herb for arthritis, studies in humans for this disease are lacking. Boswellia has been shown to reduce inflammatory processes in humans with colitis, however, which is a step in the right direction.

Boswellia is usually administered as a purified extract. For the initial treatment dose, 400 mg three times per day is suggested.

Turmeric root (*Curcuma longa*), nature's remedy for inflammation

Turmeric, the herb that gives the yellow color to curry powder, is far more than a simple cooking spice. This herb has profound anti-inflammatory properties, and is a staple in Ayurvedic bone treatment.

The active ingredient, curcumin, has been shown to be equivalent to NSAIDs as an anti-inflammatory. Curcumin is nonsteroidal, so it has none of the destructive side effects of steroid anti-inflammatories.[44] Recently, turmeric has been shown to enhance wound healing, further evidence of its potential benefit in bone health.[45] Curcumin directly treats pain, as well. Like cayenne, another medicinal spice, it depletes substance P, the pain receptor neurotransmitter, in the nerve endings.[46] Research now shows that curcumin and related compounds suppress pain through a mechanism similar to many drugs (COX-1 and COX-2 enzymes).[47]

Turmeric has very low toxicity, and is, after all, a common food spice, so the dose can be quite large. Generally, patients find that, the larger the dose, the greater the relief. For acute inflammation, such as a sore knee after exercise, the dose can be as high as one ounce (4 Tbs.) per day. Stir the powder into water and swallow, or make it into a paste with honey or a bite of oatmeal. For continuing health benefit, use 1 gram per day as a spice, or in capsules. Standardized extract is available. The dose is 1500 mg of total curcumin content per day.

Recently, a study looked at a combination of these exceptional herbs. Forty-two patients with OA were randomly assigned to receive an Ayurvedic preparation, containing (per capsule) 450 mg of ashwaganda, 100 mg of Boswellia, 50 mg of turmeric, and 50 mg of a zinc complex, or a placebo, for three months. The dosage was two capsules three times per day, after meals. The treatments were then crossed over. Compared with placebo, the herb combination significantly reduced the severity of pain and the disability score. No side effects required discontinuation of treatment.

California Poppy aerial parts (leaf, flower and stem) *(Eschscholtzia californica)*- Nature's remedy for joint degeneration

This charming backyard plant is in fact potent medicine. A Native American herb, it is a distant relative of opium poppies. California poppy contains pain-relieving isoquinoline alkaloids. In Europe, the German Commission E lists it as an antispasmodic and sedative,[48] and the herb has become a popular pain medicine. [49]

Since California poppy is relaxing, it works particularly well for people experiencing pain with nervousness and sleeplessness. A French animal study from 1991 indicated a clear-cut anti-anxiety effect. Higher doses were sedative.[50] A German test tube study indicated that the plant extract of had analgesic properties.[51] A key alkaloid (chelerthyrine) inhibits a body protein (kinase C) that contributes to persistent pain.[52, 53]

California poppy is calming and promotes sleep, so don't take it when driving, and surpass the recommended dose only with care. Raise the dose gradually until you are accustomed to the pain relieving and sedative effects.

As tea, a usual dose is 3-5 tsp. of chopped dry herb, brewed, taken when necessary. As a tincture, start with 5 ml when pain begins, and then adjust as effective.

Willow bark (*Salix alba* and other species)- Nature's aspirin for joint pain

A traditional pain reliever, willow has an outstanding reputation. It contains salicin and other related compounds (salicylates), which are the herbal predecessors of aspirin.[54] Plant salicylates relieve pain, reduce fever and curb inflammation. The American Herbal Pharmacopoeia says, "in modern herbal therapy, willow is

predominantly used as an anti-inflammatory for symptomatic relief of gouty arthritis and as an analgesic for mild neuralgic pains, toothaches and headaches."[58]

In Europe, willow is extensively used for low back pain treatment. An Israeli blinded study from 2000 substantiated this benefit. The 191 back pain sufferers took a willow extract, standardized to contain either 120 mg or 240 mg of salicin, per day. The herb turned out to be a great deal more effective than a placebo in this trial, with the higher dose being considerably more effective. In the high-dose group, the response was apparent after only 1 week of treatment.[55]

A 2001 German study looked at willow bark for OA. It assessed the clinical efficacy of a standardized willow bark extract. A dose corresponding to 240 mg salicin per day was compared with placebo in a 2-week, double blind, randomized controlled trial. The main outcome measured was pain, along with stiffness and physical function. The investigators concluded that the willow bark extract showed a moderate analgesic effect in osteoarthritis and appeared to be well tolerated.[56]

Herbs often work effectively in combination. A combination of willow, black cohosh and other herbs, given for two months, produced a significant decline in pain scores in a 1996 study of chronic arthritic pain.[57]

Willow bark does not thin the blood like aspirin, so don't use it for heart disease.[58] Fortunately, though, it won't cause the bleeding problems common with aspirin. You won't go through aspirin's characteristic digestive disturbance when using willow.

Use a tea brewed from up to 1 oz., dry weight, of the raw herb, per day, or an extract containing 240 mg total salicin per day. Use willow for as long as necessary.

Saint Johnswort flowering tops *(Hypericum perforatum*- Nature's medicine for connective tissue and nerve injury

Saint Johnswort is an ancient medicine, and has been used in Europe for hundreds of years. Clinically, European and North American herbalists use the herb to benefit mild painful conditions including arthritis, neuralgia, sciatica and muscle inflammation.[59]

The typical dose is 2-5 g of raw herb, 10-15 ml of tincture, or 900 mg of standardized extract (0.3% hypericin), per day.

Feverfew leaf (*Tanacetum parthenium*)- The aspirin of the eighteenth century

This popular decorative relative of the daisy, sometimes called "the aspirin of the eighteenth century," has been rediscovered. Traditionally it was used in European herbalism for all types of pain including arthritis. Some herbalists are now reviving the historical use, recommending feverfew for acute pain, including arthritis flares and headache.[60, 61] Often it is taken in doses of 300 mg every 15 minutes for an hour when the pain starts. Feverfew can produce a little queasiness, so work the dose up cautiously.

Corydalis tuber *(Corydalis yanhusuo)*- Nature's medicine for aches

Corydalis ("yan hu suo") is the main herb used in Traditional Chinese Medicine for treating pain. It is another relative of the poppy, containing isoquinoline alkaloids, mainly tetrahydropalmatine. The raw herb is about 1% the strength of opium.[62] Like morphine, it promotes relaxation and relieves pain. While morphine is addictive and creates tolerance, tetrahydropalmatine doesn't have these problems. Chinese herbalists value yan hu suo as a muscle relaxant, and particularly use it for menstrual pain.

Several studies in animals have confirmed the benefits.[63, 64] A 1999 animal study performed at The University of Maryland Dental School demonstrated that yan hu suo significantly reduced pain and inflammation.[65]

Corydalis is relaxing and promotes sleep, a particularly relevant benefit for OA, considering the sleep connection. Don't take it while driving, and exceed the recommended dose only with caution. Increase the dose gradually until you are familiar with the pain relieving and sedative effects. As a tea, start with ½ ounce, dry weight, of chopped herb, brewed, per day.

Devil's Claw root *(Harpagophytum procumbens)*

Devil's claw is a South African traditional herb, so named because its peculiar appearance is claw-like. Its large tuberous roots are chopped up and dried in the sun for 3 days before being used medicinally. Native

South Africans use the herb to reduce pain. European colonists took note and brought devil's claw back to Europe, where it became a popular arthritis treatment.

In modern European herbalism, devil's claw is used to treat all types of joint pain, including osteoarthritis, rheumatoid arthritis, gout and soft-tissue pain, such as back pain.

A recent double-blind study compared devil's claw to a European arthritis drug. In this trial, 122 patients with arthritis of the hip and/or knee were given either devil's claw or the drug for 4 months. The results showed that devil's claw was equally as effective as the drug, as measured by pain levels, mobility, and need for pain-relief medications (such as acetaminophen or ibuprofen).[66]

A typical European dosage of devil's claw is 750 mg 3 times daily of a preparation standardized to contain 3% iridoid glycosides.

Essential Oils, Salves and Ointments

Topical treatments are very popular with patients in pain. The Arthritis Foundation says that almost 45 percent apply ointments or rubs.**Error! Bookmark not defined.** Selected and used properly, they can be very beneficial.

Herbalist Chanchal Cabrera, NIMH, of Vancouver, BC, suggests that with salves it "may be possible to get better access to a poorly vascularized area from the outside (eg, capsicum over an painful area)."[67]

Since, as we have discussed, this is a "cold" disease, most salves and packs for the disorder have a warming effect on the tissue, increasing circulation in the joint, promoting increased movement and enhancing the delivery of nutrients.

Marsha Aker's feet hurt so bad, she thought she had "pieces of glass cutting into my feet." Barely into middle age, Marsha, a massage therapist, who had to stand on those feet all day long, thought her career was over. Finally, she was diagnosed medically as having bone spurs.

Desperate to try anything, Marsha soaked her feet in a warm salve of castor oil each night for 30 minutes. The relief was immediate, and continued to increase each night. Two weeks later, she was pain free for the first time in months.

Now, nine years later, Marsha's pain hasn't returned. She feels great, and best of all, she still keeps going full time in her busy Gresham, Oregon, massage practice, where she's known for her liberal use of—you guessed it—castor oil salve!

Many therapists will be familiar with the use of castor oil as a pack. Castor oil *(Rincinis communis)* is widely used in Western herbalism, Ayurveda and Chinese medicine. According to noted herbalist Michael Tierra, the oil is recommended in Ayurveda, externally and internally, to relieve all *vata* (cold and dry, nervous system-oriented) derangements, including pains, constipation and arthritis. The Chinese also use it for joint pain. [68]

Saint Johnswort may be more known in Europe as an external remedy, which is where it really excels. It is one of the most popular European remedies, as an oily preparation, such as an ointment, for joint pain.[69]

Arnica flower *(Arnica montana)* salve is clearly the winner in the sore joint category. This remedy is widely respected in Europe and North America. As the remedy par excellence for sprains and bruises and injured tissues, widely esteemed German herb authority Rudolf Fritz Weiss, MD claims, "Arnica has excellent pain relieving properties."[70] For traumatic injury to the tissues of all kinds, this is the one.

My personal experience with arnica is very impressive. In my two decades of concocting herbal salves, the absolute standout has been a formula of arnica flower, witch hazel, turpentine, pine tar and sulfur. This combination is a general trauma healing salve, which is used for bruises, sprains, strains, rashes, wounds and eczema.

Cayenne peppers have become one of the hottest remedies around. Capsaicin, the active compound in hot peppers, applied topically, is a widely available and effective treatment for pain.

Capsaicin formulations have increased in just a few years to capture almost 20 percent of the topical analgesics category, because of their efficacy in providing relief.[71]

A double blind clinical study found that topical capsaicin reduces the pain of arthritis by depleting nerves of "substance P," a neurotransmitter that carries pain sensations to the brain. This study of 70 patients with osteoarthritis of the knee had them apply a 0.025 % capsaicin cream or a placebo to their diseased knee four times daily. After four weeks, those using capsaicin cream reported significant improvements in pain.[72]

Another double blind randomized, controlled trial showed that 0.025 % capsaicin cream applied four times daily was effective in pain management of osteoarthritis of the knee, ankle, wrist and shoulder.[73]

Cayenne ointments do not provide immediate pain relief. Often they take several applications to penetrate to the nerve and block the substance P. It's important not to give up too soon. I often suggest 2-3 daily applications for at least two weeks to expect results.

At first, capsaicin might produce a mild burning sensation, but this discomfort quickly fades, as does the pain. Wash your hands after applying capsaicin cream, as it can be painful if residual cream comes into contact with your eyes or other sensitive tissues.

Counter-irritation, the experience of "fooling" pain by generating a superficial feeling of cold or heat over a sore area, has been known for centuries. Counter-irritants include menthol, wintergreen oil, camphor, eucalyptus oil, turpentine oil and methylnicotinate, which arouse or irritate nerve endings, distracting the body's attention from musculoskeletal pain. Generally quite safe, counter-irritants sometimes cause rashes and blisters. Be careful of sensitive areas, such as behind the knees.

Warming preparations are generally better for degenerative bone diseases. In my experience, clove oil preparations, when applied topically, are superb for enhancing the neurological response of the muscle, and can also be used in neuromuscular diseases. Ginger oil is the counterpart to clove, suppressing the nerve signal, and is used on muscles in spasm.

Putting Together a Lifestyle

In addition to these effective natural treatments, authorities have identified lifestyle adjustments that can help people make the most of their life as the work to conquer bone and joint degeneration.[74]

First and foremost, individuals should maintain ideal body weight. The more the weight, the more stress on bones, especially hips, knees, back and feet. Movement and exercises are critical. Good posture will protect the joints in the neck, back, hips and knees. When lifting or carrying, the largest and strongest joints and muscles should be used. Alternating periods of heavy activity with periods of rest will avoid repetitive stress. Pain is an important message- it should not be ignored. Changing positions regularly will decrease stiffness in muscles and joints. New activities should be started slowly and safely to feel how the body will react to them. Appropriate safety equipment should be part of activities. Patients must ask for help whenever they need it- no heroics allowed.

Sidebar

Two Newcomers to watch for

Thunder God Vine root (*Tripterygium wilfordii*) ("Lei Gong Teng")

This herb is used in the modern practice of Chinese medicine. As evidence for its effectiveness mounts, it is gradually gaining attention from herbalists. Although still obscure here, over 200 studies of thunder god vine have been published in the scientific literature.

Thunder god vine is used primarily in treating autoimmune diseases, such as rheumatoid arthritis, in which it is a successful substitute for corticosteroid drugs.[75] In one multi-center study of rheumatoid arthritis involving 226 patients, overall effectiveness of the remedy surpassed 90%.[76] Studies have shown the herb to inhibit the function of immune effector cells such as neutrophils, macrophages and B lymphocytes. A recent study from China sought to investigate the mechanism of action for Tripterygium. In this test tube study, researchers determined that the herb was immunosuppressive in autoimmune diseases.[77]

A 2000 scientific article written by the Arthritis Center at The University of Texas Southwestern Medical Center in Dallas confirmed that accumulated data from the clinical trials suggest efficacy of this treatment in a number of rheumatic diseases, including rheumatoid arthritis and systemic lupus erythematosus.[78] They go on to say that its immunosuppressive and anti-inflammatory effects suggest that diterpenoid compounds account for the therapeutic effects.

While most of the research on this herb suggests uses for inflammatory arthritis, some OA patients find that it benefits their condition.

Thunder god vine contains some components, apparently unrelated to the active ingredients, which make the herb tricky to use. Chinese herbalists process the herb specially to remove these compounds. Many modern herbalists are administering the isolated diterpenes, which seems to work just as well and to avoid the

adverse reactions. Preparations of Tripterygium are just beginning to show up in American markets. Use the dose suggested on the package, or recommended by a qualified herbalist.

Butterbur root and leaf *(Petasites hybridus)*

Butterbur has been used in European traditional herbalism to treat pain. Modern research is confirming this use.[79] Research reveals that the herb is anti-inflammatory and antispasmodic.[80]

In scientific studies, butterbur produced significantly fewer migraine attacks, fewer migraine days and a reduction in migraine pain.[81] The qualities of this herb lend it to treatment of OA.

Standardized extracts of butterbur are now available in the United States. The adult dosage ranges from 50-100 mg of standardized extract twice daily with meals. When used to treat chronic conditions, administration is preventive and supplementation should be carried out daily until symptoms improve and then tapered to a maintenance dose that gives the best results.

Resources

National Osteoporosis Foundation
1232 22nd Street N.W.
Washington, D.C. 20037-1292
(202) 223.2226
http://www.nof.org

Internatinal Osteoporosis Foundation
71, cours Albert-Thomas
69447 Lyon Cedex 03
France
Phone: +33 472 91 41 77
Fax: +33 472 36 90 52
E-mail: info@osteofound.org
http://www.osteofound.org

International Society for Clinical Densitometry
1200 19th St, NW Suite 300
Washington DC 20036
USA
202 828 6056
Iscd@dc.sba.com

The National Institutes of Health
Osteoporosis and Related Bone Diseases National Resource Center
1232 22nd Street, NW
Washington, DC 20037-1292
800-624-BONE
orbdnrc@nof.org
http://www.osteo.org

Ostex International Inc.
2203 Airport Way South, Suite 400
Seattle, WA 98134, U.S.A.
800-996-7839
www.osteomark.com

Osteoporosis Education Project
605 Frankin Park Drive
East Syracuse, NY 13057-1610
315.437.9384
info@betterbones.com
http://betterbones.com

[1] http://www.osteo.org
[2] About Osteoporosis www.osteomark.com
[3] http://www.nof.org/osteoporosis/index.htm
[4] Osteoporosis Education Project www.betterbones.com
[5] Indiana Menopause Center http://www.indianamenopause.com/DEXA_Bone_Density_Scan.htm
[6] Personal communication
[7] NY TIMES July 12, 1995
[8] Frontera WR, Meredith CN, O'Reilly KP, Knuttgen HG, Evans WJ.
J Appl Physiol 1988 Mar;64(3):1038-44 Strength conditioning in older men: skeletal muscle hypertrophy and improved function.
United States Department of Agriculture Human Nutrition Research Center on Aging, Tufts University, Boston, Massachusetts 02111.
The effects of strength conditioning on skeletal muscle function and mass were determined in older men. Twelve healthy untrained volunteers (age range 60-72 yr) participated in a 12-wk strength training program (8 repetitions/set; 3 sets/day; 3 days/wk) at 80% of the one repetition maximum (1 RM) for extensors and flexors of both knee joints. They were evaluated before the program and after 6 and 12 wk of training. Weekly measurements of 1 RM showed a progressive increase in strength in extensors and flexors. By 12 wk extensor and flexor strength had increased 107.4 (P less than 0.0001) and 226.7% (P less than 0.0001), respectively. Isokinetic peak torque of extensors and flexors measured on a Cybex II dynamometer increased 10.0 and 18.5% (P less than 0.05) at 60 degrees/s and 16.7 and 14.7% (P less than 0.05) at 240 degrees/s. The torque-velocity relationship showed an upward displacement of the curve at the end of training, mainly in the slow-velocity high-torque region. Midthigh composition from computerized tomographic scans showed an increase (P less than 0.01) in total thigh area (4.8%), total muscle area (11.4%), and quadriceps area (9.3%). Biopsies of the vastus lateralis muscle revealed similar increases (P less than 0.001) in type I fiber area (33.5%) and type II fiber area (27.6%). Daily excretion of urinary 3-methyl-L-histidine increased with training (P less than 0.05) by an average 40.8%. Strength gains in older men were associated with significant muscle hypertrophy and an increase in myofibrillar protein turnover.
[9] Morganti CM, Nelson ME, Fiatarone MA, Dallal GE, Economos CD, Crawford BM, Evans WJ.
Med Sci Sports Exerc 1995 Jun;27(6):906-12 Strength improvements with 1 yr of progressive resistance training in older women.
Human Physiology Laboratory, Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University, Boston, MA, USA.
Thirty-nine healthy women (59.5 +/- 0.9 yr) were randomized to either a control group (CON) or a progressive resistance training group (PRT) that trained twice weekly for 12 months. PRT trained at 80% or more (average of 84%) of their most recent one repetition maximum (1RM) on the lateral pull-down (LPD), knee extensor (KE), and double leg press (DLP) apparatus. One RM was measured for each exercise once monthly in PRT and at baseline, midstudy, and end of study in CON. One RM significantly increased in PRT for all muscle groups trained compared to CON ($P < 0.0001$). Increases of 73.7 +/- 12%, 35.1 +/- 3%, and 77.0 +/- 5%, respectively, for KE, DLP, and LPD in PRT and 12.7% +/- 8%, 3.7% +/- 3%, and 18.4% +/- 4%, respectively, in CON were observed. Approximately 50% of the gains in KE and LPD and 40% in the DLP were seen in the first 3 months of the study. In all three exercises, strength gains in PRT continued over the entire 12-month period. These data indicate that high-intensity strength training results in substantial, continual increases in strength in postmenopausal women for at least 12 months, with the greatest gains seen in the first 3 months of training.
[10] Dalsky GP, Stocke KS, Ehsani AA, Slatopolsky E, Lee WC, Birge SJ Jr. Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. Ann Intern Med 1988 Jun;108(6):824-8
Washington University School of Medicine, St. Louis, Missouri.
STUDY OBJECTIVE: To assess the effect of weight-bearing exercise training and subsequent detraining on lumbar bone mineral content in postmenopausal women. DESIGN: Non-randomized, controlled, short-term (9 months) trial and long-term (22 months) exercise training and detraining (13 months). SETTING: Section of applied physiology at a university school of medicine. PATIENTS: Thirty-five healthy, sedentary postmenopausal women, 55 to 70 years old. All women completed the study. There was 90% compliance with exercise training. INTERVENTIONS: All women were given calcium, 1500 mg daily. The exercise group did weight-bearing exercise (walking, jogging, stair climbing) at 70% to 90% of maximal oxygen uptake capacity for 50 to 60 min, 3 times weekly. MEASUREMENTS AND MAIN RESULTS: Bone mineral content increased 5.2% (95% confidence interval [CI], 2.0% to 8.4%; $P = 0.0037$) above baseline after short-term training whereas there was no change (-1.4%) in the control group. After 22 months of exercise, bone mineral content was 6.1% (95% CI, 3.9% to 8.3% above baseline; $P = 0.0001$) in the long-term training group. After 13 months of decreased activity, bone mass was 1.1% above baseline in the detraining group. CONCLUSIONS: Weight-bearing exercise led to significant increases above baseline in bone mineral content which were maintained with continued training in older, postmenopausal women. With reduced weight-bearing exercise, bone mass reverted to baseline levels. Further studies are needed to determine the threshold exercise prescription that will produce significant increases in bone mass.
[11] Melville Nancy A. Exercise can help you recover faster from the discomfort of arthritis
http://www.healthscout.com/template.asp?page=newsDetail&ap=1&id=500317
[12] Deyle GD, Henderson NE, Matekel RL, Ryder MG, Garber MB, Allison SC. Effectiveness of manual physical therapy and exercise in osteoarthritis of the knee. A randomized, controlled trial. Ann Intern Med 2000 Feb 1;132(3):173-81
Brooke Army Medical Center and US Army-Baylor University, Fort Sam Houston, Texas 78234-6200, USA.
gail.deyle@amedd.army.mil
BACKGROUND: Few investigations include both subjective and objective measurements of the effectiveness of treatments for osteoarthritis of the knee. Beneficial interventions may decrease the disability associated with osteoarthritis and the need for more invasive treatments. OBJECTIVE: To evaluate the effectiveness of physical therapy for osteoarthritis of the knee, applied by experienced physical therapists with formal training in manual therapy. DESIGN: Randomized, controlled clinical trial. SETTING:

Outpatient physical therapy department of a large military medical center. PATIENTS: 83 patients with osteoarthritis of the knee who were randomly assigned to receive treatment (n = 42; 15 men and 27 women [mean age, 60 +/- 11 years]) or placebo (n = 41; 19 men and 22 women [mean age, 62 +/- 10 years]). INTERVENTION: The treatment group received manual therapy, applied to the knee as well as to the lumbar spine, hip, and ankle as required, and performed a standardized knee exercise program in the clinic and at home. The placebo group had subtherapeutic ultrasound to the knee at an intensity of 0.1 W/cm2 with a 10% pulsed mode. Both groups were treated at the clinic twice weekly for 4 weeks. MEASUREMENTS: Distance walked in 6 minutes and sum of the function, pain, and stiffness subscores of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC). A tester who was blinded to group assignment made group comparisons at the initial visit (before initiation of treatment), 4 weeks, 8 weeks, and 1 year. RESULTS: Clinically and statistically significant improvements in 6-minute walk distance and WOMAC score at 4 weeks and 8 weeks were seen in the treatment group but not the placebo group. By 8 weeks, average 6-minute walk distances had improved by 13.1% and WOMAC scores had improved by 55.8% over baseline values in the treatment group ($P < 0.05$). After controlling for potential confounding variables, the average distance walked in 6 minutes at 8 weeks among patients in the treatment group was 170 m (95% CI, 71 to 270 m) more than that in the placebo group and the average WOMAC scores were 599 mm higher (95% CI, 197 to 1002 mm). At 1 year, patients in the treatment group had clinically and statistically significant gains over baseline WOMAC scores and walking distance; 20% of patients in the placebo group and 5% of patients in the treatment group had undergone knee arthroplasty. CONCLUSIONS: A combination of manual physical therapy and supervised exercise yields functional benefits for patients with osteoarthritis of the knee and may delay or prevent the need for surgical intervention.

[13] Garfinkel MS, Schumacher HR Jr, Husain A, Levy M, Reshetar RA. Evaluation of a yoga based regimen for treatment of osteoarthritis of the hands. J Rheumatol 1994 Dec;21(12):2341-3

Division of Rheumatology, University of Pennsylvania School of Medicine, Philadelphia.

OBJECTIVE. Yoga and relaxation techniques have traditionally been used by nonmedical practitioners to help alleviate musculoskeletal symptoms. The objective of this study was to collect controlled observations of the effect of yoga on the hands of patients with osteoarthritis (OA). METHODS. Patients with OA of the hands were randomly assigned to receive either the yoga program or no therapy. Yoga techniques were supervised by one instructor once/week for 8 weeks. Variables assessed were pain, strength, motion, joint circumference, tenderness, and hand function using the Stanford Hand Assessment questionnaire. RESULTS. The yoga treated group improved significantly more than the control group in pain during activity, tenderness and finger range of motion. Other trends also favored the yoga program. CONCLUSION. This yoga derived program was effective in providing relief in hand OA. Further studies are needed to compare this with other treatments and to examine longterm effects.

[14] Personal communication

[15] Cowden, William Lee, M.D., *The Harmful Effects of an Imbalanced pH on the Body Systems*, Nature's Sunshine Products National Convention, 2003.

[16] Personal communication

17 Li MK, Kavanagh JP, Prendiville V, Buxton A, Moss DG, Blacklock NJ. Does sucrose damage kidneys? Br J Urol 1986 Aug;58(4):353-7

There is evidence to suggest that sucrose ingestion can cause renal parenchymal changes as well as increasing the urinary saturation index for calcium oxalate. Ten stone formers and 10 normal subjects received 250 gm of sucrose daily over a period of 7 days. Observations on the risk factors for calcium stone formation and urinary N-acetyl-B-glucosaminidase (NAG), a marker of renal tubular cell damage, were made. Oxalate excretion increased. Urinary calcium levels were unchanged but the pattern of response was different between the two groups, as with magnesium and phosphate. NAG was spontaneously higher in the patient group and increased significantly after sucrose ingestion in both groups.

[18] Holl MG, Allen LH. Sucrose ingestion, insulin response and mineral metabolism in humans. J Nutr 1987 Jul;117(7):1229-33

The effects of sucrose ingestion on the excretion of urinary calcium, zinc, phosphorus, sodium and potassium have been investigated and compared among 13 individuals to the magnitude of their postprandial serum insulin response. Fasted subjects consumed a beverage containing 2 g sucrose/kg, and urine and blood samples were taken at intervals during the next 3 h. As a result of sucrose consumption there were significant increases in serum insulin and decreases in serum phosphorus, but no change in serum total or filterable calcium, zinc, sodium or potassium. Urine calcium peaked at 1.5 h and was significantly increased from 10 through 2.5 h. Sucrose-induced increases in serum insulin and urine calcium were highly variable among subjects, and within the group were significantly correlated ($r = 0.82$, P less than 0.01). Urine calcium excretion was correlated with serum phosphorus ($r = 0.41$, P less than 0.05) and urine zinc ($r = 0.80$, P less than 0.01). Sucrose consumption also increased the urinary excretion of zinc and sodium, although renal reabsorption was not impaired. The effects of sucrose on urinary calcium are consistent with the hypothesis that insulin inhibits renal calcium reabsorption.

[19] Nguyen NU, Henriet MT, Dumoulin G, Widmer A, Regnard J. Increase in calciuria and oxaluria after a single chocolate bar load. Horm Metab Res 1994 Aug;26(8):383-6

Explorations Fonctionnelles Renales Metaboliques et Endocriniennes, Centre Hospitalier Universitaire de Besancon, France.

Chocolate, a foodstuff rich in sucrose, fat and oxalate, is considered unsuitable in cases of obesity, diabetes mellitus, urolithiasis and postprandial hypoglycemia. However the pathophysiological effects of chocolate are poorly documented. Therefore we investigated the effects of ingestion of 100 g dark chocolate bar (45 g cocoa and 55 g sucrose) on carbohydrate, calcium and oxalate metabolisms in 10 healthy subjects. Results were compared to those of 55 g sucrose intake (control group) performed on another day. Chocolate caused i) a lesser but longer increase in plasma glucose, insulin, and C-peptide than sucrose (respectively +23% of baseline vs +60%, $p < 0.001$; +436% of baseline vs +755%, $p < 0.01$ and +200% of baseline vs +331%, $p < 0.01$), ii) a striking increase in triglyceridemia, calciuria and oxaluria (respectively +96%, $p < 0.01$; +147%, $p < 0.01$ and +213%, $p < 0.001$). Thus, chocolate

(cocoa+sucrose) causes a lesser pancreatic stimulation than sucrose. However, the increases in both calciuria and oxaluria (induced respectively by sucrose and cocoa) following chocolate ingestion might contribute to urinary conditions favoring the development of calcium oxalate calculi.

[20] Holl MG, Allen LH. Sucrose ingestion, insulin response and mineral metabolism in humans. J Nutr 1987 Jul;117(7):1229-33

The effects of sucrose ingestion on the excretion of urinary calcium, zinc, phosphorus, sodium and potassium have been investigated and compared among 13 individuals to the magnitude of their postprandial serum insulin response. Fasted subjects consumed a beverage containing 2 g sucrose/kg, and urine and blood samples were taken at intervals during the next 3 h. As a result of sucrose consumption there were significant increases in serum insulin and decreases in serum phosphorus, but no change in serum total or filterable calcium, zinc, sodium or potassium. Urine calcium peaked at 1.5 h and was significantly increased from 10 through 2.5 h. Sucrose-induced increases in serum insulin and urine calcium were highly variable among subjects, and within the group were significantly correlated (r = 0.82, P less than 0.01). Urine calcium excretion was correlated with serum phosphorus (r = 0.41, P less than 0.05) and urine zinc (r = 0.80, P less than 0.01). Sucrose consumption also increased the urinary excretion of zinc and sodium, although renal reabsorption was not impaired. The effects of sucrose on urinary calcium are consistent with the hypothesis that insulin inhibits renal calcium reabsorption.

[21] Tucker KL, Chen H, Hannan MT, Cupples LA, Wilson PW, Felson D, Kiel DP. Bone mineral density and dietary patterns in older adults: the Framingham Osteoporosis Study. Am J Clin Nutr 2002 Jul;76(1):245-52

BACKGROUND: Several nutrients are known to affect bone mineral density (BMD). However, these nutrients occur together in foods and dietary patterns, and the overall effects of dietary choices are not well understood. OBJECTIVE: We evaluated associations between dietary patterns and BMD in older adults. DESIGN: Of the original Framingham Heart Study subjects, 907 aged 69-93 y completed food-frequency questionnaires as part of an osteoporosis study. We defined dietary patterns by cluster analysis. BMD was measured at the proximal right femur (femoral neck, trochanter, Ward's area) with a dual-photon absorptiometer and at the 33% radial shaft with a single-photon absorptiometer. We regressed BMD measures onto the cluster variable, adjusting for potential confounders. RESULTS: Six dietary patterns were identified, with relatively greater proportions of intake from meat, dairy, and bread; meat and sweet baked products; sweet baked products; alcohol; candy; and fruit, vegetables, and cereal. After adjustment for multiple comparisons, men in the last group had significantly (P = 0.05) greater BMD than did 2-4 other groups at the hip sites and the candy group at the radius. Men in the candy group had significantly (P < 0.05) lower BMD than did those in the fruit, vegetables, and cereal group for 3 of the 4 sites. Women in the candy group had significantly (P < 0.01) lower BMD than did all but one other group at the radius. CONCLUSIONS: Dietary pattern is associated with BMD. High fruit and vegetable intake appears to be protective in men. High candy consumption was associated with low BMD in both men and women.

[22] Tucker KL, Chen H, Hannan MT, Cupples LA, Wilson PW, Felson D, Kiel DP. Bone mineral density and dietary patterns in older adults: the Framingham Osteoporosis Study. Am J Clin Nutr 2002 Jul;76(1):245-52

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[23] Messier SP, Loeser RF, Mitchell MN, Valle G, Morgan TP, Rejeski WJ, Ettinger WH. Exercise and weight loss in obese older adults with knee osteoarthritis: a preliminary study. J Am Geriatr Soc 2000 Sep;48(9):1062-72

Department of Health and Exercise Science, Wake Forest University, Winston-Salem, North Carolina 27109, USA.

OBJECTIVE: The purposes of this pilot study were to determine if a combined dietary and exercise intervention would result in significant weight loss in older obese adults with knee osteoarthritis, and to compare the effects of exercise plus dietary therapy with exercise alone on gait, strength, knee pain, biomarkers of cartilage degradation, and physical function. DESIGN: Single-blind, two-arm, randomized clinical trial conducted for 24 weeks. SETTING: A university health and exercise science center. PARTICIPANTS: Twenty-four community-dwelling obese older adults aged > or = 60 years, body mass index > or = 28, knee pain, radiographic evidence of knee osteoarthritis, and self-reported physical disability. INTERVENTION: Randomization into two groups: exercise and diet (E&D) and exercise alone (E). Exercise consisted of a combined weight training and walking program for 1 hour three times per week. The dietary intervention included weekly sessions with a nutritionist utilizing cognitive-behavior modification to change dietary habits to reach a group goal of an average weight loss of 15 lb (6.8 kg) over 6 months. MEASUREMENTS: All measurements were conducted at baseline and 3 and 6 months, except for synovial fluid analysis, which was obtained only at baseline and 6 months. In addition, weight was measured weekly in the E&D group. Physical disability and knee pain were measured by self-report and physical performance was measured using the 6-minute walk and stair climb tasks. Biomechanical testing included kinetic and kinematic analysis of gait and isokinetic strength testing. Synovial fluid was analyzed for levels of total proteoglycan, keratan sulfate, and interleukin-1 beta. RESULTS: Twenty-one of the 24 participants completed the study, with one dropout in the E&D group and two in

the E group. The E&D group lost a mean of 18.8 lb (8.5 kg) at 6 months compared with 4.0 lb (1.8 kg) in the E group (P = .01). Significant improvements were noted in both groups in self-reported disability and knee pain intensity and frequency as well as in physical performance measures. However, no statistical differences were found between the two groups at 6 months in knee pain scores or self-reported performance measures of physical function. There was no difference in knee strength between the groups, with both groups showing modest improvements from baseline to 6 months. At 6 months, the E&D group had a significantly greater loading rate (P = .03) and maximum braking force (P = .01) during gait. There were no significant between-group differences in the other biomechanical measures. Synovial fluid samples were obtainable at both baseline and 6 months in eight participants (four per group). The level of keratan sulfate decreased similarly in both groups from an average baseline of 96.8 +/- 37.1 to 71.5 +/- 23 ng/microg total proteoglycan. The level of IL-1 decreased from 25.3 +/- 9.8 at baseline to 8.3 +/- 6.1 pg/mL. The decrease in IL-1 correlated with the change in pain frequency (r = -0.77, P = .043). CONCLUSIONS: Weight loss can be achieved and sustained over a 6-month period in a cohort of older obese persons with osteoarthritis of the knee through a dietary and exercise intervention. Both exercise and combined weight loss and exercise regimens lead to improvements in pain, disability, and performance. Moreover, the trends in the biomechanical data suggest that exercise combined with diet may have an additional benefit in improved gait compared with exercise alone. A larger study is indicated to determine if weight loss provides additional benefits to exercise alone in this patient population.

[24] Felson DT, Zhang Y, Anthony JM, Naimark A, Anderson JJ. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. The Framingham Study. Ann Intern Med 1992 Apr 1;116(7):535-9

Boston University Arthritis Center, Boston City Hospital, Massachusetts.

OBJECTIVE: To evaluate the effect of weight loss in preventing symptomatic knee osteoarthritis in women. DESIGN: Cohort analytic study. SETTING: The Framingham Study, based on a sample of a defined population. PATIENTS: Women who participated in the Framingham Knee Osteoarthritis Study (1983 to 1985): Sixty-four out of 796 women studied had recent-onset symptomatic knee osteoarthritis (knee symptoms plus radiographically confirmed osteoarthritis) were compared with women without disease. MEASUREMENTS: Recalled date of symptom onset was used as the incident date of disease. Historical weight was defined as baseline body mass index up to 12 years before symptom onset. Change in body mass index was assessed at several intervals before the current examination. Odds ratios assessing the association between weight change and knee osteoarthritis were adjusted for age, baseline body mass index, history of previous knee injury, habitual physical activity level, occupational physical labor, smoking status, and attained education. RESULTS: Weight change significantly affected the risk for the development of knee osteoarthritis. For example, a decrease in body mass index of 2 units or more (weight loss, approximately 5,1 kg) over the 10 years before the current examination decreased the odds for developing osteoarthritis by over 50% (odds ratio, 0.46; 95% Cl, 0.24 to 0.86; P = 0.02). Among those women with a high risk for osteoarthritis due to elevated baseline body mass index (greater than or equal to 25), weight loss also decreased the risk (for 2 units of body mass index, odds ratio, 0.41; P = 0.02). Weight gain was associated with a slightly increased risk for osteoarthritis, which was not statistically significant. CONCLUSION: Weight loss reduces the risk for symptomatic knee osteoarthritis in women.

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Boston University School of Medicine, Arthritis Center, MA 02118, USA.

Overweight people are at high risk of developing knee osteoarthritis (OA) and may also be at increased risk of hand and hip OA. Furthermore, being overweight accelerates disease progression in knee OA. While the increased joint stress accompanying obesity may explain the strong linkage between obesity and knee OA risk, it does not necessarily explain why obese people have a high risk of disease in the hand nor why obese women are at higher comparative risk of knee disease than obese men. Unfortunately, studies of metabolic factors linked to obesity have not provided an explanation for these findings. There are a paucity of data on weight loss as a treatment for OA, but preliminary information suggests it is especially effective in knee disease and that even small amounts of weight reduction may have favourable effects.

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BACKGROUND: The role of caffeine as a risk factor for bone loss is controversial. OBJECTIVE: Our goals were 1) to compare in both a cross-sectional study and a 3-y longitudinal study the bone mineral density (BMD) of postmenopausal women consuming high or low amounts of caffeine and 2) to study the interaction between caffeine intake, vitamin D receptor (VDR) polymorphism, and BMD in the longitudinal study. DESIGN: The results are derived from cross-sectional measurements of BMD in 489 elderly women (aged 65-77 y) and from longitudinal measurements made in 96 of these women who were treated with a placebo for 3 y. Changes in BMD were adjusted for confounding factors and were compared between groups with either low (< or =300 mg/d) or high (>300 mg/d) caffeine intakes and between the VDR genotype subgroups of the low- and high-caffeine groups. RESULTS: Women with high caffeine intakes had significantly higher rates of bone loss at the spine than did those with low intakes (-1.90 +/- 0.97% compared with 1.19 +/- 1.08%; P = 0.038). When the data were analyzed according to VDR genotype and caffeine intake, women with the tt genotype had significantly (P = 0.054) higher rates of bone loss at the spine (-8.14 +/- 2.62%) than did women with the TT genotype (-0.34 +/- 1.42%) when their caffeine intake was >300 mg/d. CONCLUSIONS: Intakes of caffeine in amounts >300 mg/d (approximately 514 g, or 18 oz, brewed coffee) accelerate bone loss at the spine in elderly postmenopausal women. Furthermore, women with the tt genetic variant of VDR appear to be at a greater risk for this deleterious effect of caffeine on bone.

[27] Patrick L. Comparative absorption of calcium sources and calcium citrate malate for the prevention of osteoporosis. Altern Med Rev 1999 Apr;4(2):74-85
Anthropologically speaking, humans were high consumers of calcium until the onset of the Agricultural Age, 10,000 years ago. Current calcium intake is one-quarter to one-third that of our evolutionary diet and, if we are genetically identical to the Late Paleolithic Homo sapiens, we may be consuming a calcium-deficient diet our bodies cannot adjust to by physiologic mechanisms. Meta-analyses of calcium and bone mass studies demonstrate supplementation of 500 to 1500 mg calcium daily improves bone mass in adolescents, young adults, older men, and postmenopausal women. Calcium citrate malate has high bioavailability and thus has been the subject of calcium studies in these populations. Positive effects have been seen in prepubertal girls, adolescents, and postmenopausal women. The addition of trace minerals and vitamin D in separate trials has improved the effect of calcium citrate malate on bone density and shown a reduction of fracture risk.
[28] Cashman KD. Calcium intake, calcium bioavailability and bone health. Br J Nutr 2002 May;87 Suppl 2:S169-77
Calcium accounts for 1-2 % of adult human body weight. Over 99 % of total body Ca is found in the teeth and bones. Therefore, in addition to the obvious structural role of the skeleton, it also serves as a reservoir for Ca. Dietary Ca intake has an important impact on bone metabolism and bone health. Chronic Ca deficiency resulting from inadequate intake or poor intestinal absorption is one of several important causes of reduced bone mass and osteoporosis. It is vital, therefore, that adequate dietary Ca is consumed at all stages of life - in early life so that the genetically programmed peak bone mass can be reached and in later adulthood so that the skeletal mass can be maintained and age-related bone loss minimised. Unfortunately, there is wide variation in the estimates of daily Ca requirements made by different expert authorities. Furthermore, there is evidence that many individuals are not consuming these recommended levels. The consequence of this for bone health will be discussed in the present review. Besides the amount of Ca in the diet, the absorption of dietary Ca in foods is also a critical factor in determining the availability of Ca for bone development and maintenance. Thus, there is a need to identify food components and/or functional food ingredients that may positively influence Ca absorption in order to ensure that Ca bioavailability from foods can be optimised. This approach may be of particular value in individuals who fail to achieve the dietary recommended level of Ca.
[29] Somer, 1995; Mahan et al, 1996
[30] Health Equations http://healthequations.com/products.html
[31] Epstein O, Kato Y, Dick R, Sherlock S. Vitamin D, hydroxyapatite, and calcium gluconate in treatment of cortical bone thinning in postmenopausal women with primary biliary cirrhosis. Am J Clin Nutr 1982 Sep;36(3):426-30
Women with primary biliary cirrhosis malabsorb calcium, phosphate and vitamin D, and develop accelerated cortical bone thinning. We have assessed the value of parenteral vitamin D, oral hydroxyapatite (HA), and calcium gluconate (CG) in the treatment of cortical bone thinning in primary biliary cirrhosis. Sixty-four postmenopausal women with primary biliary cirrhosis were assigned randomly into three groups: one group receiving no mineral supplements (control), one group receiving HA, and one group receiving CG. All patients received parenteral vitamin D2 (100,000 IU monthly). Eleven patients withdrew from the study and 10 withdrew due to poor compliance (six HA, four CG). Over a 14-month follow-up period, none of the groups showed a significant change in serum calcium or inorganic phosphate levels. Pre- and posttreatment hand radiographs were used to assess changes in metacarpal cortical thickness using the technique of caliper radiogrammetry. Cortical bone loss occurred in the control group (p less than 0.01). The HA group showed a significant gain in cortical bone thickness (p less than 0.01), while no significant change occurred in the CG group. This study indicated that vitamin D2 does not halt metacarpal cortical bone thinning in primary biliary cirrhosis. The addition of CG prevents bone thinning, and HA promotes positive cortical bone balance.
[32] Lloyd T, Martel JK, Rollings N, Andon MB, Kulin H, Demers LM, Eggli DF, Kieselhorst K, Chinchilli VM. The effect of calcium supplementation and Tanner stage on bone density, content and area in teenage women. Osteoporos Int 1996;6(4):276-83
Department of Obstetrics and Gynecology, College of Medicine, Pennsylvania State University, Hershey 17033, USA.
One hundred and twelve Caucasian girls, 11.9 +/- 0.5 years of age at entry, were randomized into a 24-month, double-masked, placebo-controlled trial to determine the effect of calcium supplementation on bone mineral content, bone area and bone density. Supplementation was 500 mg calcium as calcium citrate malate (CCM) per day. Controls received placebo pills, and compliance of both groups averaged 72%. Bone mineral content, bone mineral area and bone mineral density of the lumbar spine and total body were measured by dual energy X-ray absorptiometry (DXA). Calcium intake from dietary sources averaged 983 mg/day for the entire study group. The supplemented group received, on average, an additional 360 mg calcium/day from CCM. At baseline and after 24 months, the two groups did not differ with respect to anthropometric measurements, urinary reproductive hormone levels or any measurement of pubertal progression. The supplemented group had greater increases of total body bone measures: content 39.9% versus 35.7% ($p = 0.01$), area 24.2% versus 22.5% ($p = 0.15$) and density 12.2% versus 10.1% ($p = 0.005$). Region-of-interest analyses showed that the supplemented group had greater gains compared with the control group for bone mineral density, content and area. In particular, in the lumbar spine and pelvis, the gains made by the supplemented group were 12%-24% greater than the increases made by the control group. Bone acquisition rates in the two study groups were further compared by subdividing the groups into those with below- or above-median values for Tanner score and dietary calcium intake. In subjects with below-median Tanner scores, bone acquisition was not affected by calcium supplementation or dietary calcium level. However, the calcium supplemented subjects with above-median Tanner had higher bone acquisition rates than the placebo group with above-median Tanner scores. Relative to the placebo group, the supplemented group had increased yearly gains of bone content, area and density which represented about 1.5% of adult female values. Such increases, if held to adult skeletal maturity, could provide protection against future risk of osteoporotic fractures.
[33] Heaney RP, Recker RR, Weaver CM. Absorbability of calcium sources: the limited role of solubility. Calcif Tissue Int 1990 May;46(5):300-4

Center for Hard Tissue Research, Creighton University, Omaha, Nebraska 68178.
Fractional absorption of seven chemically defined calcium sources was measured in normal adult women under standardized load conditions. Solubility of the sources in water at neutral pH ranged from a low of 0.04 mM to a high of 1500 mM. The relationship of solubility to absorbability was weak. In the range from 0.1 to 10 mM, within which most calcium supplement sources fall, there was no detectable effect of solubility on absorption. Data from four food sources are presented for comparison. Absorbability of food calcium was not clearly related to absorbability of the dominant chemical form in the food concerned. These findings suggests that (1) even under controlled, chemically defined conditions, solubility of a source has very little influence on its absorbability; and (2) absorbability of calcium from food sources is determined mainly by other food components.

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Procter & Gamble Company, Miami Valley Laboratories, Cincinnati, Ohio.
Absorption of calcium from a highly soluble form of calcium, a mixed calcium citrate-malate salt (CCM), was tested against calcium carbonate and milk in both rats and humans. The rat method estimated absorption from the 6-day retention of an oral tracer, and the human method employed the standard double-isotope procedure. CCM was given both as a dry powder and in an orange juice beverage. In two experiments in rats calcium from CCM was absorbed at least as well as, if not better than from calcium carbonate or milk. In two separate experiments in humans, calcium from CCM was absorbed significantly better than from calcium carbonate or milk. We conclude that CCM exhibits excellent bioavailability and that this formulation is a useful addition to the forms of calcium now available either for direct supplementation or for food fortification.

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Department of Medicine, Indiana University School of Medicine, Indianapolis 46223.
Calcium absorption from two Ca salts was investigated in a crossover design using stable isotopic tracers in 12 healthy adolescents (6 males, 6 females). A Ca supplement in the form of Ca carbonate or Ca citric and malic acids (CCM) was ingested with a standardized breakfast and the order of administration was randomized. The oral supplement contained 250 mg elemental Ca, 21.8 mg of which was highly enriched 44Ca tracer. Thirty minutes later subjects received 3.6 mg 42Ca tracer intravenously. The molar concentrations of 42Ca and 44Ca tracers in a urine sample obtained 24 h after tracer administration were quantified by fast-atom-bombardment mass spectrometry and used to determine fractional absorption of the Ca from the supplement. Ca in the form of CCM had an increased fractional absorption (p less than 0.03) relative to Ca carbonate in healthy adolescents (36.2 vs 26.4%). This increase was not related to body size, sex, or indices of Ca metabolism.

[36] Reginster JY, Deroisy R, Rovati LC, Lee RL, Lejeune E, Bruyere O, Giacovelli G, Henrotin Y, Dacre JE, Gossett C. Long-term effects of glucosamine sulphate on osteoarthritis progression: a randomised, placebo-controlled clinical trial. Lancet 2001 Jan 27;357(9252):251-6
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BACKGROUND: Treatment of osteoarthritis is usually limited to short-term symptom control. We assessed the effects of the specific drug glucosamine sulphate on the long-term progression of osteoarthritis joint structure changes and symptoms. METHODS: We did a randomised, double-blind placebo controlled trial, in which 212 patients with knee osteoarthritis were randomly assigned 1500 mg sulphate oral glucosamine or placebo once daily for 3 years. Weightbearing, anteroposterior radiographs of each knee in full extension were taken at enrolment and after 1 and 3 years. Mean joint-space width of the medial compartment of the tibiofemoral joint was assessed by digital image analysis, whereas minimum joint-space width--ie, at the narrowest point--was measured by visual inspection with a magnifying lens. Symptoms were scored by the Western Ontario and McMaster Universities (WOMAC) osteoarthritis index. FINDINGS: The 106 patients on placebo had a progressive joint-space narrowing, with a mean joint-space loss after 3 years of -0.31 mm (95% CI -0.48 to -0.13). There was no significant joint-space loss in the 106 patients on glucosamine sulphate: -0.06 mm (-0.22 to 0.09). Similar results were reported with minimum joint-space narrowing. As assessed by WOMAC scores, symptoms worsened slightly in patients on placebo compared with the improvement observed after treatment with glucosamine sulphate. There were no differences in safety or reasons for early withdrawal between the treatment and placebo groups. INTERPRETATION: The long-term combined structure-modifying and symptom-modifying effects of gluosamine sulphate suggest that it could be a disease modifying agent in osteoarthritis.

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www.healthwellexchange.com/nutritionsciencenews/NSN_backs/Jan_01/shelfstockers.cfm

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www.findarticles.com/cf_0/m1279/2001_Jan/66961755/print.jhtml

[39] Black Tea Monograph, Natural Standard, naturalstandard.com

[40] Clifton-Bligh PB, Baber RJ, Fulcher GR, Nery ML, Moreton T. The effect of isoflavones extracted from red clover (Rimostil) on lipid and bone metabolism. Menopause 2001 Jul-Aug;8(4):259-65
Department of Endocrinology, Royal North Shore Hospital, St. Leonards NSW 2065, Australia.
OBJECTIVE: This study was undertaken to evaluate the effects of varying doses of phytoestrogens on lipid and bone metabolism in postmenopausal women. DESIGN: A novel red clover isoflavone preparation (Rimostil) containing genistein, daidzein, formononetin, and biochanin was administered to 46 postmenopausal women in a double-blind protocol after a single-blind placebo phase and followed by a single-blind washout phase. Patients were randomized to receive either 28.5 mg, 57 mg, or 85.5 mg of phytoestrogens daily for a 6-month period. RESULTS: At 6 months, the serum high-density lipoprotein cholesterol had risen significantly by 15.7-

28.6% with different doses (p = 0.007, p = 0.002, p = 0.027), although the magnitude of the response was independent of the dose used. The serum apolipoprotein B fell significantly by 11.5-17.0% with different doses (p = 0.005, p = 0.043, p = 0.007) and the magnitude of the response was independent of the dose used. The bone mineral density of the proximal radius and ulna rose significantly by 4.1% over 6 months with 57 mg/day (p = 0.002) and by 3.0% with 85.5 mg/day (p = 0.023) of isoflavones. The response with 28.5 mg/day of isoflavones was not significant. There was no significant increase in endometrial thickness with any of the doses of isoflavone used. CONCLUSION: These results show that the administration of an isoflavone combination extracted from red clover was associated with a significant increase in high-density lipoprotein cholesterol, a significant fall in apolipoprotein B, and a significant increase in the predominantly cortical bone of the proximal radius and ulna after 6 months of treatment. Interpretation of the results is undertaken cautiously because of the absence of a simultaneously studied control group.

[41] Whiting SJ, Draper HH. Effect of a chronic acid load as sulfate or sulfur amino acids on bone metabolism in adult rats. J Nutr 1981 Oct;111(10):1721-6

Diets containing an acid load as either sulfur amino acids (SAA) or inorganic sulfate were fed to 45Ca-labeled adult male rats for 10 months. Radioisotope excretion and bone composition data (femur, tibia, mandibles) were compared with those for rats fed a control (15% soy protein) diet. Rats fed the SAA supplement (1.28% cystine plus 0.19% methionine) exhibited a significant reduction in femoral weight and A:R ratio and a tendency toward lower specific gravity, dry weight, fat-free weight and calcium content. Femoral radioautographs indicated a reduction in metaphyseal bone density in six of eight animals. We have postulated that the osteopenia produced by feeding excess free SAA may be due to decreased bone formation caused by a reaction between homocysteine and the aldehyde groups of collagen, as in genetic homo-cystinuric osteoporosis. Sulfate feeding (1.42% of the diet) produced a significant increase in 45Ca excretion, indicative of enhanced bone resorption, lasting about 2 months. There was a tendency for bone mass measurements to be lower than controls after 10 months, but the differences were not significant. Two of eight sulfate-fed rats showed radiographic evidence of osteopenia. No evidence of osteopenia was seen in the controls or in rats previously fed a high protein diet containing the same concentration of SAA.

[42] Lakshmi-Chandra Mishra Alternative Medicine Review. Scientific Basis for the Therapeutic Use of Withania somnifera (Ashwagandha): A Review. 2000;5(4) 334-346

OBJECTIVE: The objective of this paper is to review the literature regarding Withania somnifera (ashwagandha, WS) a commonly used herb in Ayurvedic medicine. Specifically, the literature was reviewed for articles pertaining to chemical properties, therapeutic benefits, and toxicity. DESIGN: This review is in a narrative format and consists of all publications relevant to ashwagandha that were identified by the authors through a systematic search of major computerized medical databases; no statistical pooling of results or evaluation of the quality of the studies was performed due to the widely different methods employed by each study. RESULTS: Studies indicate ashwagandha possesses anti-inflammatory, antitumor, antistress, antioxidant, immunomodulatory, hemopoetic, and rejuvenating properties. It also appears to exert a positive influence on the endocrine, cardiopulmonary, and central nervous systems. The mechanisms of action for these properties are not fully understood. Toxicity studies reveal that ashwagandha appears to be a safe compound. CONCLUSION: Preliminary studies have found various constituents of ashwagandha exhibit a variety of therapeutic effects with little or no associated toxicity. These results are very encouraging and indicate this herb should be studied more extensively to confirm these results and reveal other potential therapeutic effects. Clinical trials using ashwagandha for a variety of conditions should also be conducted.

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[45] Sidhu GS, Singh AK, Thaloor D, Banaudha KK, Patnaik GK, Srimal RC, Maheshwari RK Enhancement of wound healing by curcumin in animals. *Wound Repair Regen* 1998 Mar-Apr;6(2):167-77

[46] Patacchini R, Maggi CA, Meli A Capsaicin-like activity of some natural pungent substances on peripheral endings of visceral primary afferents. *Naunyn Schmiedebergs Arch Pharmacol* 1990 Jul;342(1):72-7

1. The effects of some naturally occurring pungent substances, piperine, mustard oil, eugenol and curcumin, were compared to those of capsaicin in the rat isolated urinary bladder. 2. All test compounds dose-dependently contracted the rat bladder and produced desensitization toward capsaicin (1 mumol/l). Development of cross-tachyphylaxis among the natural pungent substances on one hand and capsaicin on the other, suggested a common site of action on visceral primary afferents. 3. Contractile responses to piperine, mustard oil and eugenol were partially tetrodotoxin and ruthenium red-sensitive, suggesting that activation of sensory terminals by these agents takes place indirectly, as well as by a direct action on sensory receptors. 4. The presence of the secondary acrylamide linkage (present in the backbone of capsaicin, but not in that of test compounds) does not appear to be essential to produce desensitization of sensory nerve terminals.

[47] Ramsewak RS, DeWitt DL, Nair MG Cytotoxicity, antioxidant and anti-inflammatory activities of curcumins I-III from Curcuma longa. *Phytomedicine* 2000 Jul;7(4):303-8

Curcumin I, curcumin II (monodemethoxycurcumin) and curcumin III (bisdemethoxycurcumin) from Curcuma longa were assayed for their cytotoxicity, antioxidant and anti-inflammatory activities. These compounds showed activity against leukemia, colon, CNS, melanoma, renal, and breast cancer cell lines. The inhibition of liposome peroxidation by curcumins I-III at 100 microg/ml were 58, 40 and 22%, respectively. The inhibition of COX-I and COX-II enzymes by the curcumins was observed. Curcumins I-III were active against COX-I enzyme at 125 microg/ml and showed 32, 38.5 and 39.2% inhibition of the enzyme, respectively. Curcumins I-III also showed good inhibition of the COX-II enzyme at 125 mg/ml with 89.7, 82.5 and 58.9% inhibition of the enzyme, respectively.

[48] Blumenthal, Mark, *The Complete Commission E Monographs*, The American Botanical Council, Austin, Texas, 1998

[49] *PDR for Herbal Medicines*, Medical Economics Company, Montvale, New Jersey, 1998

[50] Rolland A, Fleurentin J, Lanhers MC, Younos C, Misslin R, Mortier F, Pelt JM Behavioural effects of the American traditional plant Eschscholzia californica: sedative and anxiolytic properties. *Planta Med* 1991 Jun;57(3):212-6

Eschsholzia californica Cham. is a traditional medicinal plant of the Indians used by the rural population of California for its analgesic and sedative properties. Our study on the aqueous extract shows that this plant reduced the behavioural parameters measured in a familiar environment test in mice (novelty preference, locomotion and rearings in two compartments test) at doses above 100 mg/kg and in non-familiar environment tests (staircase test) at doses above 200 mg/kg. This finding validates its traditional sedative properties confirmed by the sleeping induction at doses above 100 mg/kg. Furthermore, when administered at a dose a of 25 mg/kg, E. californica appeared to also have an anxiolytic action since it produced an increase of the number of steps climbed by mice in the staircase test (anticonflict effect) and that of the time spent by animals in the lit box when they were confronted with the light/dark choice situation. Before evaluation of the behavioural effects, it was verified that our aqueous extract did not induce any toxic effect when administered i.p. and p.o.

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The endogenous pentapeptides, met-enkephalin and leuenkephalin, similar to their parent structures, beta-endorphin or dynorphin, bind to opioid receptors of the nociceptive system thus provoking analgesic responses. Peroxidases and phenolases (tyrosinase, catecholase) were shown to dimerize these pentapeptides thus possibly modulating their activity and/or lifetime. Extracts from plants from the order of the Papaverales contain isoquinoline alkaloids. Since the benzoisoquinolines are known to possess sedative-hypnotic activities, the potential effects of extracts from two species from this plant group, Eschscholtzia californica (Papaveraceae) and tyrosinase-catalyzed dimerization and/or oxidation of met-enkephalin were investigated. The results of the study show that the peroxidase-catalyzed dimerization via the tyr-residues is especially inhibited by the C. cava extract. The tyrosinase-catalyzed reaction yields five different products A-E, according to their HPLC-retention times. Consisting of the 4:1 (v/v) combination of the extracts from E. californica and C. cava, Phytonoxon N (abbreviated as PN) stimulates the formation of minor products A, B and E, whereas the formation of the major products C and D is inhibited. Only products C and D exhibit properties similar to the peroxidase-derived dimer. Product A is likely to be identical to DOPA-enkephalin.

[52] Herbert JM, Augereau JM, Gleye J, Maffrand JP Chelerythrine is a potent and specific inhibitor of protein kinase C. *Biochem Biophys Res Commun* 1990 Nov 15;172(3):993-9

The benzophenanthridine alkaloid chelerythrine is a potent, selective antagonist of the Ca++/phospholopid-dependent protein kinase (Protein kinase C: PKC) from the rat brain. Half-maximal inhibition of the kinase occurs at 0.66 microM. Chelerythrine interacted with the catalytic domain of PKC, was a competitive inhibitor with respect to the phosphate acceptor (histone IIIS) (Ki = 0.7 microM) and a non-competitive inhibitor with respect to ATP. This effect was further evidenced by the fact that chelerythrine inhibited native PKC and its catalytic fragment identically and did not affect [3H]- phorbol 12,13 dibutyrate binding to PKC. Chelerythrine selectively inhibited PKC compared to tyrosine protein kinase, cAMP-dependent protein kinase and calcium/calmodulin-dependent protein kinase. The potent antitumoral activity of celerythrine measured in vitro might be due at least in part to inhibition of PKC and thus suggests that PKC may be a model for rational design of antitumor drugs.

[53] Meller ST, Dykstra C, Gebhart GF Acute thermal hyperalgesia in the rat is produced by activation of N-methyl-D-aspartate receptors and protein kinase C and production of nitric oxide. *Neuroscience* 1996 Mar;71(2):327-35

There is general agreement that activation of the N-methyl-D-aspartate receptor is involved in thermal hyperalgesia. However, there is less agreement on the specific intracellular events subsequent to receptor activation and the involvement of other excitatory amino acid receptors in thermal hyperalgesia. In the present study, we found that the intrathecal administration of N-methyl-D-aspartate produced a dose- (1 fmol-1 pmol) and time-dependent thermal hyperalgesia. In contrast, over the dose range tested, intrathecal administration of either alpha-amino-3-hydroxy-5-methylisoxazole-4-proprionate (AMPA; 10 fmol-100 pmol), 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (10 fmol-100 pmol), quisqualate (10 pmol-5 nmol) or a 1:1 combination of AMPA and 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (total dose 20 fmol-200 pmol) did not produce any evidence of thermal hyperalgesia; greater doses produced a caudally-directed biting and scratching behavior that precluded testing in the paradigm used. A fixed dose of 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (100 pmol) did, however, potentiate the effects of N-methyl-D-aspartate (1-100 fmol). Thermal hyperalgesia produced by N-methyl-D-aspartate (1 pmol) was attenuated by intrathecal administration of the N-methyl-D-aspartate receptor-selective antagonist 2-amino-5-phosphonopentanoate (100 pmol), but not by the AMPA receptor-selective antagonist 6,7-dinitroquinoxaline-2,3-dione (1 nmol) or the metabotropic receptor antagonist 2-amino-3-phosphonoproprionate (10 nmol). In a second series of experiments, we examined the role of different signal transduction systems in acute N-methyl-D-aspartate-produced thermal hyperalgesia. N-Methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was attenuated by intrathecal hemoglobin (1-100 pmol) and dose-dependently by intrathecal N(G)-nitro-L-arginine methyl ester (10 pmol-l nmol), Methylene Blue (10 pmol-l nmol) and chelerythrine (1-100 pmol), suggesting that acute N-methyl-D-aspartate-mediated thermal hyperalgesia involves activation of nitric oxide synthase and protein kinase C. In contrast, N-methyl-D-aspartate-produced thermal hyperalgesia was unaffected by intrathecal administration of the phospholipase A2 inhibitor mepacrine (10 nmol) or the phospholipase C inhibitor neomycin (10 nmol). While prostaglandins and leukotrienes have been suggested to play a role in hyperalgesia, N-methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was unaffected by the non-selective eicosanoid inhibitor nordihydroguaiarate (1 nmol), the cyclo-oxygenase selective inhibitor indomethacin (10 nmol) or the lipoxygenase selective inhibitor baicalein (1 nmol). The results of the present study suggest that acute thermal hyperalgesia can be produced by activation of N-methyl-D-aspartate receptors. Activation of AMPA, metabotropic or co-activation of AMPA and metabotropic glutamate receptors, at the doses tested, did not produce an acute thermal hyperalgesia. The thermal hyperalgesia produced by N-methyl-D-aspartate is mediated by activation of nitric oxide synthase

and protein kinase C, but not by phospholipase C, phospholipase A2, cyclo-oxygenase or lipoxygenase. Collectively, the results are consistent with a role for spinal N-methyl-D-aspartate receptors, nitric oxide and protein kinase C in thermal hyperalgesia.

[54] Willow Bark Monograph, European Scientific Cooperative on Phytotherapy Monographs, Exeter, UK, 1997

[55] Chrubasik S, Eisenberg E, Balan E, Weinberger T, Luzzati R, Conradt C Treatment of low back pain exacerbations with willow bark extract: a randomized double-blind study. *Am J Med* 2000 Jul;109(1):9-14

PURPOSE: Herbal medicines are widely used for the treatment of pain, although there is not much information on their effectiveness. This study was designed to evaluate the effectiveness of willow (Salix) bark extract, which is widely used in Europe, for the treatment of low back pain.SUBJECTS AND METHODS: We enrolled 210 patients with an exacerbation of chronic low back pain who reported current pain of 5 or more (out of 10) on a visual analog scale. They were randomly assigned to receive an oral willow bark extract with either 120 mg (low dose) or 240 mg (high dose) of salicin, or placebo, with tramadol as the sole rescue medication, in a 4-week blinded trial. The principal outcome measure was the proportion of patients who were pain-free without tramadol for at least 5 days during the final week of the study.RESULTS: The treatment and placebo groups were similar at baseline in 114 of 120 clinical features. A total of 191 patients completed the study. The numbers of pain-free patients in the last week of treatment were 27 (39%) of 65 in the group receiving high-dose extract, 15 (21%) of 67 in the group receiving low-dose extract, and 4 (6%) of 59 in the placebo group (P <0.001). The response in the high-dose group was evident after only 1 week of treatment. Significantly more patients in the placebo group required tramadol (P <0.001) during each week of the study. One patient suffered a severe allergic reaction, perhaps to the extract. CONCLUSION: Willow bark extract may be a useful and safe treatment for low back pain.

[56] Schmid B, Ludtke R, Selbmann HK, Kotter I, Tschirdewahn B, Schaffner W, Heide L. Efficacy and tolerability of a standardized willow bark extract in patients with osteoarthritis: randomized placebo-controlled, double blind clinical trial. Phytother Res 2001 Jun;15(4):344-50

Pharmazeutisches Institut, Universitat Tubingen, Germany.

This study assessed the clinical efficacy of a chemically standardized willow bark extract in the treatment of osteoarthritis. Willow bark extract, in a dose corresponding to 240 mg salicin/day, was compared with placebo in a 2-week, double-blind, randomized controlled trial. The primary outcome measure was the pain dimension of the WOMAC Osteoarthritis Index. Secondary outcome measures included the stiffness and physical function dimensions of the WOMAC, daily visual analogue scales (VAS) on pain and physical function, and final overall assessments by both patients and investigators. A total of 78 patients (39 willow bark extract, 39 placebo) participated in the trial. A statistically significant difference between the active treatment and the placebo group was observed in the WOMAC pain dimension (d = 6.5 mm, 95% C.I. = 0.2-12.7 mm, p = 0.047); the WOMAC pain score was reduced by 14% from the baseline level after 2 weeks of active treatment, compared with an increase of 2% in the placebo group. The patient diary VAS confirmed this result, and likewise the final overall assessments showed superiority of the willow bark extract over the placebo (patients' assessment, p = 0.0002; investigators' assessment, p = 0.0073). It is concluded that the willow bark extract showed a moderate analgesic effect in osteoarthritis and appeared to be well tolerated.

[57] Mills SY, Jacoby RK, Chacksfield M, Willoughby M. Effect of a proprietary herbal medicine on the relief of chronic arthritic pain: a double-blind study. Br J Rheumatol 1996 Sep;35(9):874-8

Centre for Complementary Health Studies, University of Exeter, Devon.

Eighty-two subjects with chronic arthritic pain were randomly assigned for 2 months without cross-over to either Reumalex, a licenced over-the-counter (OTC) herbal medicine, or a placebo. Entry characteristics were determined by a previous survey of arthritic customers at pharmacy and healthfood shop outlets. The AIMS2 questionnaire was completed at monthly intervals throughout and for 2 months prior to the trial, and a modified Ritchie Index provided clinical scores. Subjects also completed diary recordings of their use of self-prescribed analgesics and events they considered significant. There was a small but statistically significant improvement in pain symptoms, less so in sufferers from osteoarthritis. There were no other significant changes in any other measures nor in the use of other self-prescribed analgesics. There were few side-effects noted. It is concluded that Reumalex has a mild analgesic effect in chronic arthritis at a level appropriate to self-medication.

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[62] Bensky, Dan, and Gamble, Andrew, *Chinese Materia Medica, Eastland Press, Seattle, 1986*

[63] Kubo M, Matsuda H, Tokuoka K, Ma S, Shiomoto H Anti-inflammatory activities of methanolic extract and alkaloidal components from Corydalis tuber. *Biol Pharm Bull* 1994 Feb;17(2):262-5

A methanolic extract (CM-ext) from Corydalis tuber (Corydalis turtschaninovii Besser forma yanhusuo Y. H. Chou et C. C. Hsu) has been screened for activity in experimental models of inflammation. CM-ext (200 or 500 mg/kg, p.o.) inhibited an increase in vascular permeability in mice induced by acetic acid, and reduced acute paw edema in rats induced by compound 48/80 or carrageenin. CM-ext suppressed the development of adjuvant-induced edema in arthritic rats. CM-ext and its alkaloidal components, dehydrocorydaline, d-glaucine and l-tetrahydrocoptisine inhibited compound 48/80-induced histamine release from peritoneal mast cells of rats. Since these substances from C. tuber were found to be effective in both the acute and chronic phases of inflammation, the crude drug C. tuber can be considered to exert anti-inflammatory activity.

[64] Liu L, Li G, Zhu F, Wang L, Wang Y[Comparison of analgesic effect between locally vinegar-processed preparation of fresh rhizoma Corydalis and traditionally vinegar-processed rhizoma Corydalis]. *Chung Kuo Chung Yao Tsa Chih* 1990 Nov;15(11):666-7, 702

Hot plate and writhing methods were used in the comparison of the analgesic effect of vinegar-processed fresh tuber corydalis and the traditionally vinegar-processed Rhizoma Corydalis. The result shows that the effect of the former is stronger than that of the latter.

[65] Wei F, Zou S, Young A, Dubner R, Ren K Effects of four herbal extracts on adjuvant-induced inflammation and hyperalgesia in rats. *J Altern Complement Med* 1999 Oct;5(5):429-36

OBJECTIVE: To evaluate the effects of four herbal medicine extracts on a rat model of inflammatory hyperalgesia. DESIGN/INTERVENTIONS: Inflammation was induced by injecting complete Freund's adjuvant (CFA) into one hindpaw of each rat. Four herbs that are routinely prescribed in Traditional Chinese Medicine for treatment of pain were used: Duhuo (Radix Angelicae Pubescentis), Bai jiang cao (Patriniae Herba cum Radice), Yan hu suo (Rhizoma Corydalis) and Sanqui (Panax Notoginseng). The crude water extracts of the herbs were inected intraperitoneally following a repeated treatment profile. OUTCOME MEASURES: Thermal hyperalgesia was assessed by testing each rat's paw withdrawal response to a noxious thermal stimulus. The magnitude of edema was determined by measuring the maximal thickness of the paw with a caliper. The effect of herb extracts on motor performance was assessed by using an accelerating rotarod test. RESULT: Duhuo, Bai jiang cao, and Yan hu suo significantly attenuated CFA-induced hyperalgesia at 2 hours and facilitated the recovery from hyperalgesia ($p < 0.05$), when compared to saline-treated rats. The CFA-induced edema was reduced by Duhuo at 24 hours, 72 hours and 168 hours; Bai jiang cao at 24 hours, and Yan hu suo at 24 hours and 168 hours. Sanqi did not produce any significant effect on inflammation and hyperalgesia. The rotarod performance was slightly reduced by Bai jiang cao, Yan hu suo, and Sanqi ($p < 0.05$) but not by Duhuo treatment. CONCLUSION: The present study identified Duhuo as a selective and effective herbal agent in attenuating persistent hindpaw inflammation and hyperalgesia in rats. These results indicate that some herbal agents may provide an alternative approach to the treatment of persistant inflammatory pain and hyperalgesia.

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Laboratoires Arkopharma, Carros, France.

In a double-blind, randomized, multicentre clinical study, the efficacy and tolerance of a herbal medicine product, Harpadol (6 capsules/day, each containing 435 mg of powdered cryoground powder Harpagophytum procumbens), was compared with diacerhein 100 mg/day in the treatment, for 4 months, of 122 patients suffering from osteoarthritis of the knee and hip. Assessments of pain and functional disability were made on a 10 cm horizontal visual analogue scale; severity of osteoarthritis was evaluated by Lequesne's index. Spontaneous pain showed a significant improvement during the course of the study and there was no difference in the efficacy of the two treatments. Similarly, there was a progressive and significant reduction in the Lequesne functional index and no statistical difference was found between Harpadol and diacerhein. At completion of the study, patients taking Harpadol were using significantly less NSAIDs and antalgic drugs. The frequency of adverse events was significantly lower in the Harpadol group. The most frequent event reported was diarrhea, occurring in 8.1% and 26.7% of Harpadol and diacerhein patients respectively. The global tolerance assessment by patients at the end of treatment favoured Harpadol. The results of this study demonstrate that Harpadol is comparable in efficacy and superior in safety to diacerhein.

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http://www.planetherbs.com/articles/introduction_to_planetary_herbol.htm

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Case Western Reserve University, Cleveland, Ohio.

The neuropeptide substance P has been implicated in the pathogenesis of inflammation and pain in arthritis. In this double-blind randomized study, 70 patients with osteoarthritis (OA) and 31 with rheumatoid arthritis (RA) received capsaicin (a substance P depletor) or placebo for four weeks. The patients were instructed to apply 0.025% capsaicin cream or its vehicle (placebo) to painful knees four times daily. Pain relief was assessed using visual analog scales for pain and relief, a categorical pain scale, and physicians' global evaluations. Most of the patients continued to receive concomitant arthritis medications. Significantly more relief of pain was reported by the capsaicin-treated patients than the placebo patients throughout the study; after four weeks of capsaicin treatment, RA and OA patients demonstrated mean reductions in pain of 57% and 33%, respectively. These reductions in pain were statistically significant compared with those reported with placebo ($P = 0.003$ and $P = 0.033$, respectively). According to the global evaluations, 80% of the capsaicin-treated patients experienced a reduction in pain after two weeks of treatment. Transient burning was felt at the sites of drug application by 23 of the 52 capsaicin-treated patients; two patients withdrew from treatment because of this side effect. It is concluded that capsaicin cream is a safe and effective treatment for arthritis.

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[76] Li RL, Liu PL, Wu XC[Clinical and experimental study on sustained release tablet of Tripterygium wilfordii in treating rheumatoid arthritis]. *Chung Kuo Chung Hsi I Chieh Ho Tsa Chih* 1996 Jan;16(1):10-3

Adopt to the prospective, multi-center, random, single-blind, equal rank-control methods, 226 patients of rheumatoid arthritis diagnosed according to the ARA criteria, were divided into 2 groups. One hundred and fourteen patients of test group were treated with sustained release tablets of Tripterygium wilfordii (TW-SR) orally, 2 tablets, twice a day for 4 weeks, 112 patients of control group received tablets of Tripterygium wilfordii (TW) orally, 2 tablets 3 times per day for 4 weeks. Results showed that the total effective rate of the two groups were 92.11% and 90.65%, respectively ($P > 0.05$). The adverse reaction rate of TW-SR group was 20.18%, which was lowered than that of TW group (70.54%, $P < 0.01$). Results of pre-clinical pharmacologic experimental study showed that the TW-SR has obvious anti-inflammatory, analgesia and immunosuppress'ive action as the TW has, while its toxicity was less than the latter significantly.

[77] Ho LJ, Chang DM, Chang ML, Kuo SY, Lai JH Mechanism of immunosuppression of the antirheumatic herb TWHf in human T cells. *J Rheumatol* 1999 Jan;26(1):14-24

OBJECTIVE: To investigate the immunosuppressive mechanism of Tripterygium wilfordii Hook-F (TWHf) in human T cells. TWHf, a traditional Chinese medicinal herb for rheumatoid arthritis, has been shown to inhibit the function of immune effector cells such as neutrophils, macrophages, and B lymphocytes. METHODS: T cell survival was evaluated with trypan blue exclusion assay, morphologic changes with Wright's stain, the induction of endonuclease activity with DNA fragmentation assay, and the subdiploid DNA content with flow cytometry. T cell activation was measured with interleukin 2 (IL-2) ELISA and the expression of several surface molecules with flow cytometry. RESULTS: At high dosages, TWHf caused inhibition of T cell proliferation and this mechanism was mediated through the induction of apoptosis. TWHf, in noncytotoxic dosages, was as potent as cyclosporin A and more potent than prednisolone and cyclophosphamide in inhibiting IL-2 production from activated T cells. TWHf also inhibited both phorbol 12-myristate 13-acetate induced IL-2Ralpha expression and ionomycin induced CD40 ligand expression. TWHf did not reverse downregulated expression of CD3 and CD4 by phorbol ester stimulation. CONCLUSION: This is the first evidence that the immunosuppressive mechanism of TWHf in T cells was mediated through both downregulation of T cell receptor signaling pathway and induction of cellular apoptosis, which is defective in autoimmune diseases.

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Various preparations of Tripterygium wilfordii Hook F (TwHF) have been used in the treatment of a number of autoimmune and inflammatory diseases since the 1960s. Accumulated data from the clinical trials suggest efficacy of this treatment in a number of rheumatic diseases, including rheumatoid arthritis and systemic lupus erythematosus. Studies on the relationship of the chemical components of TwHF and its immunosuppressive and anti-inflammatory effects suggest that diterpenoid compounds with epoxide groups account for the therapeutic effects of this herbal remedy. This herbal remedy is therefore a unique and powerful alternative therapy for autoimmune and inflammatory diseases.

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Department of Neurology, Municipal Hospital, Munchen-Harlaching, Germany.

OBJECTIVE: Migraine is still an unsolved problem. This clinical trial investigates the efficacy and tolerance of Petasites hybridus in the prophylaxis of migraine. METHODS: A randomized, group-parallel, placebo-controlled, double-blind clinical study was carried out with a special CO2 extract from the rhizome of Petasites hybridus. Following a four-week run-in phase, 60 patients received either the special Petasites hybridus extract Petadolex or placebo at a dosage of two capsules (each capsule contains 25 mg) twice daily over 12 weeks. Outcome variables included the frequency, intensity and duration of migraine attacks as well as any accompanying symptoms. RESULTS: The frequency of migraine attacks decreased by a maximum of 60 percent compared to the baseline. This reduction in migraine attacks with Petadolex was significant ($p < 0.05$) compared to placebo. No adverse events were reported. Petasites was exceptionally well tolerated. CONCLUSIONS: The results suggest that migraine patients can benefit from prophylactic treatment with this special extract. The combination of high efficacy and excellent tolerance emphasizes the particular value that Petasites hybridus has for the prophylactic treatment of migraine.

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Petasides hybridus (butterbur) is a perennial shrub, found throughout Europe as well as parts of Asia and North America, that has been used medicinally for centuries. During the Middle Ages butterbur was used to treat plague and fever; in the 17th century its use was noted in treating cough, asthma, and skin wounds. The plant can grow to a height of three feet and is usually found in wet, marshy ground, in damp forests, and adjacent to rivers or streams. Its downy leaves can attain a diameter of three feet, making it the largest of all indigenous floras, and their unique characteristics are responsible for the plant's botanical and common names. The genus name, Petasites, is derived from the Greek word petasos, which is the felt hat worn by shepherds. The common name of butterbur is attributed to the large leaves being used to wrap butter during warm weather. Other common names include pestwurz (German), blatterdock, bog rhubarb, and butter-dock. Currently, the primary therapeutic uses for butterbur are for prophylactic treatment of migraines, and as an anti-spasmodic agent for chronic cough or asthma. It has also been used successfully in preventing gastric ulcers, and in treating patients with irritable bladder and urinary tract spasms.

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