

Understanding the Prevalence of Osteoporosis in Postmenopausal Women and
the Ayurvedic Relationship Between Asthi Dhatu and Vata Dosha

By Alessandra Bizzarri Todd

Osteoporosis is a disorder characterized by decreased bone mass and microarchitecture deterioration of bone tissue which increase skeletal fragility and often lead to fragility fractures in elderly men and women. It's a chronic metabolic bone disease meaning the strength of the bones have been compromised usually due to a depletion of vital minerals and irregularities in the body's homeostasis, over time. Many people go undiagnosed until they break a bone as bone mineral density (BMD) is not something that is routinely monitored. Osteoporosis is widespread meaning it is seen in all genders, ages and races, however, statistics show that it is most common in Caucasian women of older age groups. Currently, it has been estimated by the International Osteoporosis Foundation that more than two-hundred million people globally are suffering from osteoporosis. With an aging population and longer lifespan, osteoporosis is increasingly becoming a global epidemic.^{1,2} In America eighty percent of the estimated ten million people diagnosed with osteoporosis are women,³ making it one of the most important diseases women face.

In Ayurveda we are taught to live in harmony with the seasons, considering climate and time of life, we honor these cycles by intimately knowing our bodys' prakruti (constitution) and any given vikruti (imbalance) so that we may approach diet and lifestyle properly. It's a way of life in which we become devoted to simply paying attention to our internal and external landscapes. A practical practice of loving preventative care. The original Functional Medicine in which the unique individual is acknowledged and healing of the root cause of disease is facilitated. It is my intention for this paper to shed light on the prevalence of osteoporosis specifically in postmenopausal women, which happens to be our vata time of life, and how, with the ancient teachings of Ayurveda, a woman can reduce her chance of having this disease manifest in her body by understanding its samprapti (pathogenesis).

Traditional pathophysiological concepts of osteoporosis focus on endocrine mechanisms such as estrogen or vitamin D deficiency as well as secondary hyperparathyroidism. There is exciting research emerging that expands on our fundamental knowledge of osteoporosis, highlighting interactions between bone health and the immune system, the gut microbiome and cellular senescence.⁴ The classification of this disease falls under two categories-- type 1 or type 2. Type 1 osteoporosis typically affects women within the first 10 years after menopause and is characterized by fractures that occur at sites rich in cancellous bone, such as crush fractures of the vertebrae or the distal forearm. Type 2 osteoporosis is associated with age-related changes, such as impaired vitamin D metabolism, osteoblast function, and bone formation; secondary hyperparathyroidism; vitamin D deficiency; and inadequate calcium intake,

which occur as a late consequence of estrogen-deficiency osteoporosis. Type 2 fractures happen at sites containing substantial portions of both cortical and cancellous bone, including hip, proximal humerus, proximal tibia and pelvis. Anterior wedging of the vertebra of the dorsal spine is also common. Most nonvertebral fractures occur as a result of a fall in elderly people, while vertebral fractures can occur spontaneously with simple activities such as stepping off of a curb, or sitting down in the seat of a car, without a provoking cause.⁵

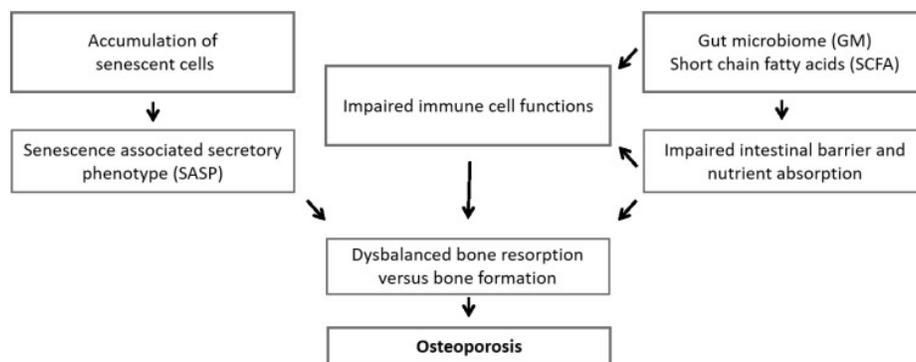
Bone is an exquisite example of the principle that form follows function. Bone provides structural support for ambulation and ventilation; a reservoir for calcium, phosphate, amino acids and bicarbonate; protection of interior organs; transmission of sound waves; and nurturing hematopoietic stem cells found in peripheral blood and bone marrow. Metabolic functions include secretion of hormones that regulate both mineral and energy metabolism. To accomplish these functions, the bone has cortical and trabecular compartments. Approximately eighty percent of the bone mass is in the cortical compartment. Vascular channels occupy about thirty percent of the volume. The surface to volume ratio in cortical bone is much lower than in trabecular/cancellous bone. On the topic of bone degeneration, we see that with aging or disease, the cortex becomes more porous, thus gaining surface area but losing strength. In the long bones, increased porosity near the periosteal surface causes more loss of strength than increased porosity near the endocortical surface⁶ which leads to thinning of the cortical wall. Age-related bone loss and postmenopausal osteoporosis are due to this deregulation of bone remodeling in which less bone is reformed than resorbed, which would not occur with equal strength in all bone regions.⁷

Bone undergoes continuous cycles of modeling and remodeling. During bone modeling either bone formation or bone resorption occur independently at distinct sites. Thereby, changes in dimensions and shape of bone during growth and adaptation of bone to altering mechanical demands are facilitated. Remodeling, in contrast is a highly coordinated process of concomitant resorption and formation at a distinct site and is responsible for the maintenance of skeletal integrity by renewing old and damaged bone. Additionally remodeling processes maintain calcium and phosphate homeostasis by targeted release and incorporation from and into the bone matrix. The crucial role of remodeling in overall bone homeostasis is highlighted by the fact that impaired remodeling, favoring bone resorption over bone formation, is a fundamental pathophysiological mechanism leading to bone pathologies such as osteoporosis.⁴

It is well accepted in the medical field that the gut microbiome (GM), which is the entirety of microorganisms living in the human digestive tract, influences development and homeostasis of gastrointestinal (GI) tract issues and also of tissues at extra-GI sites such as nutrient production and absorption, host growth and immune homeostasis. In fact, we know a well established function of the GM is to modulate immune function, hence, effects of the GM on intestinal and systemic immune responses,

which in turn modulate bone homeostasis, provide an important connection. Studies have shown specifically that the quality and pH of GM influences the absorption of nutrients required for skeletal development such as calcium, and thereby affect bone mineral density. Additionally, microbial fermentation of dietary fibers to short chain fatty acids (SCFAs) seems to play an important role in this process as it's been proven that diets that include various prebiotic foods increase calcium absorption. Beyond this influence of intestinal nutrient absorption, SCFAs have emerged as potent regulators of osteoclast differentiation and activity and of bone metabolism. Moreover, postmenopausal as well as inflammation-induced bone loss was prevented and the protective effect was associated with impaired osteoclast differentiation and bone resorption. SCFAs are therefore an example of gut-derived microbial metabolites that diffuse into systemic circulation where they can then regulate anatomically distant organs such as the skeletal system, creating what scientists are calling the microbiota-skeletal axis.⁸

One more compelling piece to this claim is in the exploration of cellular senescence which will support why improper management of stress induced inflammation can lead to bone loss. Cellular senescence describes a cell fate induced by various types of stress and is associated with irreversible cell cycle arrest and resistance to apoptosis, a process of programmed cell death. Cells entering senescence are characterized by proinflammatory cytokines, chemokines and extracellular matrix-degrading proteins.⁹ The number of these cells increases during the process of aging which has been evidenced to play a role in age-related tissue dysfunction and the development of age-related diseases such as diabetes mellitus, hypertension, atherosclerosis or osteoporosis.⁴ A recent study showed an accumulation of senescent cells in bone biopsy samples from older postmenopausal women compared to younger perimenopausal women.¹⁰



Peak bone mineral density (BMD) is typically achieved by about the age of thirty, after which time bone loss begins to outweigh bone formation, a process that accelerates with menopause. In the five to seven years immediately after menopause, women may lose as much as twenty percent of their BMD, largely as a result of declining estrogen levels and the resultant loss of the positive effects of estrogen on bone formation and protection against bone loss. The risk of osteoporotic fractures doubles every seven to

eight years after the age of fifty and by the age of eighty, women may have lost up to thirty percent of their BMD.⁵ In addition to menopausal contributing factors, aging itself is also associated with changes in bone integrity. Take kidney health for example, inefficient functioning of these organs can lead to poor calcium resorption, which can in turn lead to hyperparathyroidism and decreased BMD.

Prevention of osteoporosis should commence early in life while conventional approaches which include pharmacologic and nonpharmacologic treatments are recommended upon diagnosis. On the topic of prevention, it is significant to note that optimal bone building begins in childhood through the teen years when we are still actively laying down vital bone matrix, making it a great time to focus on education around bone health and prevention of osteoporosis through creating healthy habits in diet, lifestyle and exercise. Weight-bearing exercises can build healthy bone and strengthen the muscles around them for protection while balance training like callisthenic movements can prevent falls later in life. Ninety percent of bone density is achieved by the age of twenty, and bone remodeling continues to be optimal until the age of thirty-five, making it another crucial window of time for women to implement these healthy habits. Bone health cannot be viewed in isolation from the environment in which we live. The depletion of vital nutrients from our soil which extends to our food mixed with sedentary lifestyles and increased levels of stress all diminish bone density over time. For people diagnosed with osteoporosis, the point of therapy is to reduce fracture risk and further bone loss, prevent disability and control pain⁵, which may include the use of pharmacologic treatments. Corrections to diet, lifestyle and exercise are still very beneficial to preventing further damage and finding comfort in everyday life, encompassing a more holistic approach to health that is acknowledged by many medical resources.

Pharmacologic agents for the treatment of osteoporosis can be classified as either antiresorptive (i.e. targeting osteoclast-mediated bone resorption) or anabolic (i.e. stimulating osteoblasts to form new bone). Drugs for each type have been shown to improve BMD and reduce the risk of fractures¹¹ but do have associative side effects. Nonpharmacologic treatments include the intake of dietary supplements (specifically calcium, magnesium, zinc, vitamin D and K₂) to inhibit further bone resorption with proper calcium absorption, reduce systemic inflammation and balance hormones. This area of management would also include physical activity and modifiable risk factors like avoiding cigarette smoking which decreases BMD and limiting alcohol consumption which can increase the risk of falls.¹¹

The importance of calcium to our bone health is common knowledge in today's world. Most people assume that drinking enough milk should suffice. Just think back to the 90's GOT MILK? movement which entered our collective consciousness through constant advertisement to push consumption because it "does a body good." As a society we know it's important to our health, but we have lost the wisdom around why milk is so nourishing to our bodies, how to properly take it and why we should honor the animals that provide it. Many people today have dairy sensitivities, intolerances or

allergies because of the quality and quantity of milk in the modern American diet. It's a perfect example of how a nectar can become a poison when taken in excess-- just think of alcohol and the havoc it can wreak. The issue people face is the cow's milk we have today is unrecognizable to the milk described in the ancient texts of Ayurveda, for example. It's not organic, meaning the cows have been injected with growth hormones and fed unnatural and often GMO diets. Milk is ultra-pasteurized to kill potential pathogens "for our safety" but the real danger lies in the amount of mucus and puss that is allowed to enter the milk in the first place in the US, which is shockingly high. It's also homogenized so the fat doesn't rise to the top but in that process a potentially harmful protein called xanthine oxidase enters our bloodstream upon consumption that would otherwise not survive our digestive process. Then there is the conversation around A1 and A2 beta-casein proteins found in cow's milk. A1 is the milk pervading our grocery stores and restaurants here in America while A2, having a slightly different set up of their amino acid chains and therefore easier to digest, are found in European brown cows, goat, sheep, water buffalo and human breast milk.¹² It's a lot to unpack but the good news is the individual holds the power of knowledge and choice. A reduction in careless consumption of dairy and support of local, small batch farmers that provide high quality product can create a major shift in our health and Planet. Ayurveda describes harmonious ways to take milk, which I will cover later. There are also plenty of plant allies that can effectively deliver calcium to our bodies. Adequate intake of calcium and phosphorus in the appropriate ratio of 1-2:1 (Ca:P), found in many dark leafy greens, algae, nuts and seeds, in addition to magnesium and vitamin D, are vital for bone health.¹³ Vitamin K also plays an important role in its ability to help our bodies absorb vitamin D. A Framingham osteoporosis study also demonstrated that adequate protein intake, something like twenty grams per day, in women older than seventy-five years of age can help minimize bone loss.⁴ Checking for deficiencies in bloodwork panels run by healthcare professionals is a trusted way to get specific and guided protocols for supplementation, or better yet to see if one can get these vital nutrients into their whole-food diet.

The first symptom of osteoporosis is usually dull, aching pain in the bones, particularly the back and chest. The pain may radiate down the leg and muscle spasms may be present. At the spinal column mass may diminish, dorsal kyphosis and cervical lordosis increase, which may lead to multiple compression fractures of the spine and reduction in height. Clinical evaluation reveals a complex of risk factors such as estrogen deficiency, androgen deficiency, hyperthyroidism, chronic malnutrition, long-term lack of calcium, long history of tobacco use, alcohol abuse, sedentary lifestyle, immobility, familial history and underlying skeletal disease.¹⁴ A number of disease states and additional factors can lead to osteoporosis, commonly as a result of increased bone loss or problems with mineral secretion, absorption or deposition are but not limited to genetic disorders, calcium balance disorders, endocrinopathologies, GI disorders, chronic renal disease and long term use of various medications.⁴

So, how does this common metabolic bone disorder remain so unrecognized in early stages while also remaining an increasingly significant problem? My findings show that it's in part due to its' nature of being a clinically silent disease until it manifests in the form of a fracture, only to reveal a late-stage progression, but also because nonmedical treatment in the form of prevention; including stress management, gut health and an active lifestyle; seem to be recommended but only recently backed by modern science. As far as finding comfort for those living with osteoporosis, nonmedical treatment is said to complement the appropriate pharmacologic treatment, and these treatments should be used together to maximize outcomes for these patients.¹⁵ Enter the wisdom of Ayurveda; although revered as a complete medical science in classical texts, in California it is recognized as a complimentary science, making it a great choice not only in disease prevention but also as a lifestyle companion for conventional healing modalities of osteoporosis in postmenopausal women.

Ayurveda is a "Divine Science" due to its' origin as well as its incredible strength. It's both a preventative and curative medical system that explains the human body as a congenial balance of dosha, dhatu (tissue) and mala (waste).¹⁶ The doshas, vata, pitta and kapha, govern all functions in our bodies and their dynamism is what makes life happen. The Sanskrit word dosha, actually means "faulty" or "to cause harm," but they only do so when they are functioning abnormally. In practice the balance of the doshas is measured through observation and various evaluation techniques to understand the unique nature (prakruti) and imbalance (vikruti) present in an individual. The doshas have a natural ebb and flow in the body that have sites in particular organs and relate to environmental variables like the time of day, seasonal influence, and a persons' time of life. When there is an accumulation of a dosha, that does not get relieved naturally or with treatment, then it will overflow from the digestive system to a weaker site in the body where it will settle in and eventually manifest into a diagnosable disease, presenting more intense symptoms over time. The basic principle of treatment in Ayurveda is simple, "like increases like and opposites reduce each other,"¹⁷ which becomes helpful in understanding how to manage doshic imbalance.

Among the seven dhatus, asthi dhatu, or bone tissue, is responsible for maintenance of the structural framework of the body; it gives shape to our bodies and protects our vital organs. Asthi dhatu is one of the main sites of vata dosha and are inversely proportional to each other regarding increase and decrease, therefore explaining that excess vata over time will decrease bone mass. On a cellular level vata is involved in absorption (samana vayu) and circulation of nutrients (vyana vayu); and excretion of waste (apana vayu), which proves the accumulative or degenerative effect vata may have on bone when not functioning properly. Osteoporosis is understood as asthi dhatu kshaya (degeneration of bone tissue) and is explained in the eighteen types of kshaya in the Caraka Samhita,¹⁶ one of the most highly respected classical texts of Ayurveda.

Ayurveda views the health of the body as the functioning of a biological fire governing metabolism, called agni.¹⁸ The quality of agni is influenced by the dominant dosha present in an individual and may be described as tikshnagni (high agni/pitta), mandagni (low agni/kapha) or vishmagni (variable agni/vata). Food is digested by jatharagni (the main digestive fire in the stomach) and is turned into ahara rasa, the nutritive substance that nourishes the dhatus. The quality of each dhatu is understood to form from the preceding dhatu; rasa (lymph), rakta (blood), mamsa (muscle), medas (fat), asthi (bone), majja (marrow), shukra (reproductive essence) and ojas (immune strength and resilience), respectively. Each dhatu contains its own agni which contributes to the quality of the next tissue formation. Since medas proceeds asthi formation we can infer a connection between the nourishment of medas and asthi as diseases of one often affect the other. The evidence for a connection between osteoporosis and obesity is strong, but an increase in fat does not increase bone mass, it actually appears to antagonize the asthiagni leading to a weak supply of transformed potential nutrition from the circulating rasa.¹⁹ When digestion occurs properly, a substance being digested is transformed into usable materials and normal waste.¹⁷

The purusha dhara kala is the membrane in which asthiagni resides, that which hold bone; and the membrane that holds feces. In the body the purishavaha srotas is the large intestine, where vata is metabolized. However, the lining of the large intestine is considered homologous to the lining of the bone called periosteum. Ayurveda understands an important relationship between the bones and the colon. In the colon, positive aspects of air and earth are absorbed while negative aspects are released as feces and gas. When the colon is not functioning properly and too much gas is produced, the negative aspects of air are absorbed. This results in the excess air residing in the bones. Hence colon abnormalities damage the bones and we see the special relationship between vata and asthi.¹⁷ Elimination is the third stage of digestion in which food remains go through a drying process where water is absorbed and the remaining indigestible food remains are discarded. When vata is vitiated one may experience gas, bloating and constipation as a result. When digestion is not healthy, ama (toxins) build up in the body and causes inflammation. This combination together with things like insufficient exercise, hormonal imbalance, excess stress, exposure to chemicals, and intake of caffeine, alcohol or tobacco, will lead to osteoporosis.

Postmenopausal women, by nature are dealing with a lot of vata. Upon the cessation of menses, a woman enters the vata stage of life. Menstruation itself is governed by vata as the uterus is a hollow organ, filled with air and ether which once held the potential for life, located in a vata region of the body, the pelvis. There may have been symptoms in perimenopause like stress-related insomnia, stubborn weight gain in the vata region (hips and thighs), gut issues, hot flashes, vaginal dryness, mood fluctuations, all of which are driven by vata imbalance. Chronic stress uses more resources than the body can supply in a day which leads to bone loss therefore managing stress is one of the most important ways to strengthen bones and prevent osteoporosis.

In an Ayurvedic approach to healing, you consider vata's elemental make-up of vayu (air) and akasha (ether), and their combined gunas (qualities) which are cold, light, dry, mobile, rough, subtle, flowing, sharp, rough and clear. Every concept in Ayurveda should be understood in the light of the gunas as they demonstrate a huge matrix on which the entire universe is suspended.²⁰ Vata's positive influence on us include movement (without which the other doshas would be inert), creative flow, connection to the Divine, communication between cells and humans alike and circulation of prana, which is our very breath and life-force energy. Thinking of the principle that like increases like, it can be expected that a vata dominant person will chase movement in life which, if not managed properly, will lead to physical and mental exhaustion. The way to balance vata is by regulating agni and introducing the rest of the panchamahabhutas (five elements) into ones' sensorial, daily experience. The five elements are akasha (ether), vayu (air), tejas (fire), apas (water) and prithvi (earth). So to reduce stress we balance air and ether by grounding with earth, we regulate agni by stoking the fire to properly digest food and burn ama, and introduce moisture with each meal to soothe bodily tissues, thus reducing systemic inflammation.

Most menopausal and postmenopausal women would greatly benefit from a vata pacifying diet, which focuses on the sweet, sour and salty tastes.²⁰ An emphasis on the use of the tonifying sweet taste is encouraged as it helps to support all of the dhatus and increases ojas. Tonic therapy at this time of life slows the rate of depletion allowing a gradual transition to take place.²¹ The sweet taste is made up of earth and water elements with qualities that are heavy, moist, cool, stable, smooth and soft-- quite the opposite of our vata qualities which is what makes them balancing. Sweet taste foods include ripe fruits, root vegetables, whole grains, healthy fats like avocado, nuts and seeds; raw dairy and meats. Organic, raw dairy is considered a sattvic (peaceful) food when sourced and taken properly. The ancients advised that we spice and boil the milk, which does not destroy prana or the enzymes necessary to digest the milk in the same way that the industrial pasteurization of milk will-- adding cardamom can help digest the protein and cinnamon will help digest the carbohydrates.¹² Milk can also be an excellent anupana (carrier) of herbs when tonification of bone tissue is indicated.²² It is best taken alone but can be paired successfully with grains, dried fruits or nuts. It should not be taken with fresh fruits, vegetables or any other protein, especially fish.¹² If you have corrected digestion and dairy still cannot be tolerated then home-made plant-based milks are an excellent choice; specifically from almonds¹⁹ or oats, on the topic of bone health. A good rule of thumb is that animal products in one's diet should be sourced ethically and locally or not taken at all, to avoid an undesirable transfer of energy. Eating local, seasonal foods that make you feel good, are at the very heart of this way of life. The Ayurvedic sage Caraka taught that for food to be digested in a timely manner, thus promoting energy, healthy complexion, strength and longevity, it must not only be imbibed in proper measure, but must also be of wholesome quality.^{23,24}

Sweet herbs are those that are cooling and demulcent in nature like licorice (*glycyrrhiza glabra*) and slippery elm (*ulmus fulva*). The salty taste is made up of water and fire and includes natural salts of course, but also foods and herbs like algae, celery, nettle (*urtica dioica*) and oatstraw (*avena fatva and sativa*) because of their high mineral content. The sour taste is made up of earth and fire and includes foods like citrus fruits, berries, ferments and tomato. Sour herbs will have high vitamin C, oxalic and malic acids and tannins like elderberry (*sambucus nigra*) and schisandra (*schisandra chinensis*).²⁷ Bone, however, is supremely nourished by hard, resinous herbs such as guggulu (*commiphora mukul*) and frankincense (*boswellia serrata*) that work via the blood (rakta) to feed the bones. These two herbs have a prabhava (special action) for repairing bone tissue.¹⁹ Ashwaganda (*withania somnifera*) is a noteworthy herb in both prevention and management of osteoporosis as it has been shown to increase the number of cells that produce bone tissue and aid calcification, it reduces inflammation and helps keep hormones stable. Ashwaganda is known as panacea in Ayurvedic medicine and is one of the most extensively used indigenous herbs to India. It is mentioned as a nutritive tonic by Caraka²⁶ and is widely known today as a primary adaptogen. The following herbs may also be beneficial to nourish rakta and asthi dhatu by acting on the blood to improve mineral absorption, reduce systemic inflammation, and aid in tissue repair and rejuvenation; these include, but are not limited to, dandelion root (*taraxacum officinale*)²⁷, tumeric (*curcuma longa*), ginger (*zingiber officinale*), vidarikand (*pueraria tuberosa-radix*), bala (*sida cordifolia-radix*) and amla (*embilica officinalis*).^{19,22} When formulating, the individual is always honored in this system of medicine. Ayurvedic treatment is to cleanse the body of ama, balance the constitution and promote rejuvenation. It does not treat diseases as specific entities but as by-products of aggravated doshas.¹⁸

Depending on the condition of the patient with osteoporosis, various panchakarma treatments may be applied seasonally to remove ama and excess dosha from the body and mind, all in service of preventing further bone loss and fractures. Sushutra writes, in the classical text the Sushruta Samhita, "depleted doshas may be restored by brhamana therapy; excessive doshas may be reduced by langhana therapy; vitiated doshas may be eliminated by the shodhana therapies; and while in a state of balance, they may be maintained by shamana therapies."²⁸ Brhamana therapies are tonifying in nature and chosen in a clinical context when strength, volume or mass of a tissue needs increasing,¹⁷ making it an excellent choice for degenerative conditions like osteoporosis. The only contraindications include presence of ama, kapha imbalance or fever, in which case a practitioner may want to administer palliative, or shamana therapies before or after for mild detoxification. If a patient is in need of stronger detoxification then reduction, or langhana therapy may be appropriate, followed by rasayana (rejuvenation).

Of all the treatments in this profound body of knowledge, a postmenopausal women with osteoporosis may want to get familiar with rest and snehana (oleation therapies). Rest is the most

important of all rejuvenators. When the body is resting, all of its energies go toward healing.¹⁷ A lifestyle that also includes yoga, meditation, pranayama and time connecting with nature have a deeply nourishing effect on our physiology and Spirit. The word sneha in Sanskrit means both oil and love, thus reminding us that when we participate in oleation therapies we are in fact engaged in something sacred and holy, as love, dear reader, is the ultimate healer. Abhyanga is the anointing of one's entire body with oils or ghee, that have been infused with Ayurvedic herbs, and should be practiced on a daily basis. The synergistic blends of herbs and oils force the negative elements from the body and lubricate the passages of memory. This re-opens the channels through which information and energy flow.²⁸ Shirodhara is another snehana treatment in which oil is poured in a continuous stream onto the third eye, with great precision. The rhythm of the oil trickling onto the third eye, which is the seat of our cognitive vision, evokes deep cognitive memories. Through the arousal of these memories, bodily tissues are transformed and good health is restored.²⁸ It is excellent for all vata disorders and should be practiced regularly. Anuvasana basti, which is a medicated oil enema, is another deeply therapeutic treatment to be familiar with for the management of osteoporosis. Caraka suggests that sixty percent of all diseases are attributed to the vata dosha, and eighty percent of these disorders may be cured by enema therapy.²⁸ Anuvasana basti acts on the colon, the main site of vata, which we learned has a unique relationship to bone, so by loving on one, you love the other. Another incredible benefit of this practice is in maintaining and restoring equilibrium to the nervous system, also governed by vata. There is again no seasonal restriction with this therapy however care should be taken to administer properly.

The Ayurvedic lifestyle can be adopted by anyone. Its principles of healing the body, mind and Spirit through crafting a life of connection with Mother Nature and Her rhythms, is truly universal. Each day begins with ancient rituals designed to tune into one's divinity, for when we forget this, we become vulnerable to disease.¹⁹ Dinacharya and ritucharya are daily and seasonal routines that describe in detail the optimal timing for activities and foods in relation to day and night, sun and moon. Understanding the knowledge of life in this way acts as a guiding light to inspire intentional living, reminding us that we are nature and just like nature, we have the ability to heal and thrive. Rhythm and proper care for our sense organs become the medicine and peace of mind and longevity, the result. According to sage Sushutra, when a person's faculties of sense perception, mind and intellect are in harmony with the inner Self, known as atman, then svastha, or the optimal state of health, is achieved.²⁹ Acceptance of ourselves as we are is perhaps the greatest health of all³⁰ because it's from that place we recognize our uniqueness-- and that's exactly where Ayurveda will meet you.

Lokah Samastah Sukhino Bhavantu

Research Paper References by Alessandra Bizzarri Todd

1. Sözen T, Özışık L, Başaran NÇ. An overview and management of osteoporosis. *Eur J Rheumatol*. 2017 Mar;4(1):46-56. doi: 10.5152/eurjrheum.2016.048. Epub 2016 Dec 30. PMID: 28293453; PMCID: PMC5335887.
2. Rosen CJ. The Epidemiology and Pathogenesis of Osteoporosis. [Updated 2020 Jun 21]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. *Endotext* [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK279134/>
3. National Osteoporosis Foundation
<https://www.nof.org/preventing-fractures/general-facts/what-women-need-to-know/>
4. Föger-Samwald U, Dovjak P, Azizi-Semrad U, Kersch-Schindl K, Pietschmann P. Osteoporosis: Pathophysiology and therapeutic options. *EXCLI J*. 2020 Jul 20;19:1017-1037. doi: 10.17179/excli2020-2591. PMID: 32788914; PMCID: PMC7415937.
5. *Botanical Medicine for Women's Health* by Aviva Romm MD pg 147-148
6. *American Journal of Nephrology* 2018;47:373-375 DOI: 10.1159/000489672, "Cortical or Trabecular bone: What's the Difference?" by Susan M Ott
<https://www.karger.com/Article/FullText/489672>
7. Buenzli PR, Thomas CD, Clement JG, Pivonka P. Endocortical bone loss in osteoporosis: the role of bone surface availability. *Int J Numer Method Biomed Eng*. 2013 Dec;29(12):1307-22. doi: 10.1002/cnm.2567. Epub 2013 Jul 1. PMID: 23818461.
8. Zaiss MM, Jones RM, Schett G, Pacifici R. The gut-bone axis: how bacterial metabolites bridge the distance. *J Clin Invest*. 2019;129:3018–3028.
9. Cellular senescence and the senescent secretory phenotype: therapeutic opportunities. *Tchkonja T, Zhu Y, van Deursen J, Campisi J, Kirkland JL*
J Clin Invest. 2013 Mar; 123(3):966-72.
10. Identification of Senescent Cells in the Bone Microenvironment. *Farr JN, Fraser DG, Wang H, Jaehn K, Ogrodnik MB, Weivoda MM, Drake MT, Tchkonja T, LeBrasseur NK, Kirkland JL, Bonewald LF, Pignolo RJ, Monroe DG, Khosla S*
J Bone Miner Res. 2016 Nov; 31(11):1920-1929.
11. Lin JT, Lane JM. Osteoporosis: a review. *Clin Orthop Relat Res*. 2004 Aug;(425):126-34. PMID: 15292797
12. *Healing the Thyroid with Ayurveda* by Marianna Teitelbaum DC, pg 179-184
13. Loughrill E, Wray D, Christides T, Zand N. Calcium to phosphorus ratio, essential elements and vitamin D content of infant foods in the UK: Possible implications for bone health. *Matern Child Nutr*. 2017 Jul;13(3):e12368. doi: 10.1111/mcn.12368. Epub 2016 Sep 9. PMID: 27612307; PMCID: PMC6865864.
14. *Mosby's Medical Dictionary*, 10th Edition pg 1295-1296
15. *The New England Journal of Medicine*, "Postmenopausal Osteoporosis" by Dennis M Black PhD and Clifford J Rosen PhD (N ENGL J MED 374;3)
16. *Journal of Ayurvedic and Herbal Medicine*, "Ayurveda Medicinal Plants for Asthikshaya (Osteoporosis): A Review" 2016;2(6):229-235 by Dipti, Richa Khandelwal, Ankita Aggarwal, Mohan Lal Jaiswal
17. *Principles of Ayurvedic Medicine* by Dr Marc Halpern pgs 3-126
18. *The Yoga of Herbs* by Vasant Lad (1986); agni and herbs
19. *Ayurvedic Medicine, The Principles of Traditional Practice* by Sebastian Pole (2006)

20. Astanga Hridayam The Essence of Ayurveda by Dr Sanjay Pisharodi; (twenty basic gunas, AH su 1.18); (six tastes AH su 1.15-16); (qualities of the doshas 11-12 AH su.1.11)
21. Clinical Ayurvedic Medicine by Dr Marc Halpern pg 5-98
22. Ayurvedic Herbology East & West by Vishnu Dass (2013)
23. A Path of Practice by Bri Maya Tiwari (2000)
24. Caraka Samhita, translated to English by R.K. Sharma Bhagwandash, volume 1, wholesome food pg 109 (8)
25. Living Herbs document, "The Herbal Flavor Wheel" by Rosalee de la Foret, and her book Alchemy of Herbs (2017)
26. Dravya Guna Vijnana Volume III pg145
27. Herbal Healing for Women by Rosemary Gladstar (1993)
28. Secrets of Healing by Bri Maya Tiwari (1995)
29. Ayurveda Lifestyle Wisdom by Acharya Shunya; Sushruta Samhita, sutrasthanam-15, 41
30. A Life of Balance by Bri Maya Tiwari (1995)

Abstracts for Research Paper by Alessandra Bizzarri Todd

1) Resource: NCBI, PMID: 28293453; Published online Dec 30, 2016

Title: An Overview and Management of Osteoporosis

Author: Tumay Sozen, Lale Ozisik and Nursel Calik Basaran

Abstract:

Osteoporosis -related to various factors including menopause and aging- is the most common chronic metabolic bone disease, which is characterized by increased bone fragility. Although it is seen in all age groups, gender, and races, it is more common in Caucasians (white race), older people, and women. With an aging population and longer life span, osteoporosis is increasingly becoming a global epidemic. Currently, it has been estimated that more than 200 million people are suffering from osteoporosis. According to recent statistics from the International Osteoporosis Foundation, worldwide, 1 in 3 women over the age of 50 years and 1 in 5 men will experience osteoporotic fractures in their lifetime. Every fracture is a sign of another impending one. Osteoporosis has no clinical manifestations until there is a fracture. Fractures cause important morbidity; in men, in particular, they can cause mortality. Moreover, osteoporosis results in a decreased quality of life, increased disability-adjusted life span, and big financial burden to health insurance systems of countries that are responsible for the care of such patients. With an early diagnosis of this disease before fractures occur and by assessing the bone mineral density and with early treatment, osteoporosis can be prevented. Therefore, increasing awareness among doctors, which, in turn, facilitates increase awareness of the normal populace, will be effective in preventing this epidemic.

2) Resource: PubMed/NCBI, PMID: 25905357; Published online June 21 2020

Title: The Epidemiology and Pathogenesis of Osteoporosis

Author: Clifford J Rosen and more

Abstract:

Osteoporosis is a multifactorial disorder associated with low bone mass and enhanced skeletal fragility. Although most prevalent in older females, some men are also at high risk. Risk factors in men and women include smoking, family history of fracture, age greater than 65 years, and low but also high BMI particularly in men. Secondary causes of osteoporosis include chronic treatment with glucocorticoids, gastrointestinal disorders, diabetes mellitus (T1D, T2D), rheumatoid arthritis, liver disease, gluten enteropathy, multiple myeloma and other hematologic disorders. However, primary osteoporosis is most often related to either postmenopausal estrogen loss or age-related deterioration of skeletal microarchitecture; both are due to uncoupling in the bone remodeling unit. Reduced bone formation with age is almost certainly a function of impaired stem cell differentiation into the osteoblast lineage with a resultant increase in marrow adipogenesis. Increased bone resorption also characterizes most forms of osteoporosis but the etiology is multifactorial. Changes in local and systemic growth factors are often responsible for uncoupling between resorption and formation. However, alterations in peak bone acquisition contribute years later to low bone mass and enhanced skeletal fragility. Fracture risk assessment tools (e.g. FRAX) in handheld apps and computers which combine bone density score and risk factors, have provided rapid assessments of future osteoporotic fractures and can be performed at the bedside. Newer methods of measuring bone quality have led to insights into micro-architectural deterioration that contributes to skeletal fragility. Notwithstanding, low areal bone mineral density by DEXA remains the strongest predictor of subsequent fracture beyond age, and this is potentially measurable in everyone after age 65.

3) Resource: NCBI, PMID: 32788914; Published online July 20, 2020

Title: Osteoporosis: Pathophysiology and Therapeutic Options

Author: Ursula Foger-Samwald, Peter Dovjak and Peter Pietschmann

Abstract:

Osteoporosis is a metabolic bone disease that, on a cellular level, results from osteoclastic bone resorption not compensated by osteoblastic bone formation. This causes bones to become weak and fragile, thus increasing the risk of fractures. Traditional pathophysiological concepts of osteoporosis focused on endocrine mechanisms such as estrogen or vitamin D deficiency as well as secondary hyperparathyroidism. However, research over the last decades provided exiting new insights into mechanisms contributing to the onset of osteoporosis, which go far beyond this. Selected mechanisms such as interactions between bone and the immune system, the gut microbiome, and cellular senescence are reviewed in this article. Furthermore, an overview on currently available osteoporosis medications including antiresorptive and bone forming drugs is provided and an outlook on potential future treatment options is given.

4) Resource: PubMed/NCBI, PMID: 23818461; Published Dec 2013

Title: Endocortical Bone Loss in Osteoporosis: The Role of Bone Surface Availability

Author: Pascal Buenzli, David L Thomas, John G Clement, Peter Pivonka

Abstract:

Age-related bone loss and postmenopausal osteoporosis are due to a dysregulation of bone remodelling in which less bone is reformed than resorbed. This dysregulation of bone remodelling does not occur with equal strength in all bone regions. Loss of bone is more pronounced near the endocortical surface. This leads to thinning of the cortical wall proceeding from the endosteum, a process sometimes called 'trabecularisation'. In this paper, we investigate the influence of the nonuniform distribution of bone surface within bone tissue for osteoporotic bone losses. We use a spatio-temporal computational model of bone remodelling in which microstructural changes of bone tissue are represented by a phenomenological relationship between bone specific surface and bone porosity. The simulation of an osteoporotic condition by our model shows that the evolution of bone porosity within a bone cross section is significantly influenced by the nonuniform availability of bone surface. Greater bone loss occurs near the endocortical wall, leading to cortical wall thinning and to an expansion of the medullary cavity similar to cross-sectional observations from human femur midshafts. Our model suggests that the rate of cortical wall thinning is fast/slow in the presence/absence of an adjacent trabecular or trabecularised bone compartment.

5) Resource: PubMed/NCBI, PMID: 27341653; Published online Oct 24, 2016

Title: Identification of Senescent Cells in the Bone Microenvironment

Author: Joshua N Farr, Daniel G Fraser, Haitai Wang, Katharina Jaehn and more

Abstract:

Cellular senescence is a fundamental mechanism by which cells remain metabolically active yet cease dividing and undergo distinct phenotypic alterations, including upregulation of p16^{Ink4a}, profound

secretome changes, telomere shortening, and decondensation of pericentromeric satellite DNA. Because senescent cells accumulate in multiple tissues with aging, these cells and the dysfunctional factors they secrete, termed the senescence-associated secretory phenotype (SASP), are increasingly recognized as promising therapeutic targets to prevent age-related degenerative pathologies, including osteoporosis. However, the cell type(s) within the bone microenvironment that undergoes senescence with aging in vivo has remained poorly understood, largely because previous studies have focused on senescence in cultured cells. Thus in young (age 6 months) and old (age 24 months) mice, we measured senescence and SASP markers in vivo in highly enriched cell populations, all rapidly isolated from bone/marrow without in vitro culture. In both females and males, p16^{Ink4a} expression by real-time quantitative polymerase chain reaction (rt-qPCR) was significantly higher with aging in B cells, T cells, myeloid cells, osteoblast progenitors, osteoblasts, and osteocytes. Further, in vivo quantification of senescence-associated distension of satellites (SADS), ie, large-scale unraveling of pericentromeric satellite DNA, revealed significantly more senescent osteocytes in old compared with young bone cortices (11% versus 2%, $p < 0.001$). In addition, primary osteocytes from old mice had sixfold more ($p < 0.001$) telomere dysfunction-induced foci (TIFs) than osteocytes from young mice. Corresponding with the age-associated accumulation of senescent osteocytes was significantly higher expression of multiple SASP markers in osteocytes from old versus young mice, several of which also showed dramatic age-associated upregulation in myeloid cells. These data show that with aging, a subset of cells of various lineages within the bone microenvironment become senescent, although senescent myeloid cells and senescent osteocytes predominantly develop the SASP. Given the critical roles of osteocytes in orchestrating bone remodeling, our findings suggest that senescent osteocytes and their SASP may contribute to age-related bone loss.