DETERMINING OSTEOPOROTIC TENDENCIES FROM TISSUE MINERAL ANALYSIS OF HUMAN HAIR TYPE I AND TYPE II

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Osteoporosis is estimated to affect approximately six million people in the U.S. alone. This condition is present in twenty five percent of women over age 65. Many are in the advanced stages, and suffer from vertebral collapse and fractures of the ribs and hips as a result of even slight trauma.

Osteoporosis is characterized by abnormal porosity and rarefaction of bone, resulting in decreased radiodensity. Albright defined osteoporosis as: "that category of decreased bone mass where the disturbance is the failure of the osteoblast to lay down bone matrix." He further stated that "Osteoporosis is a decreased production of osteoid by the osteoblasts, and is a defect in tissue metabolism." Osteoporosis differs from other bone diseases such as osteomalacia and rickets, which occur as a result of abnormal bone calcification. Osteomalacia is associated with normal osteoid formation with diminished calcium deposition, whereas rickets is a result of increased osteoplastic activity in relation to osteoblastic activity.

The factors involved in osteoporosis are numerous and usually multicausal in origin. The etiology of these factors are controversial, and are often not readily evident by abnormalities in serum calcium and phosphorus studies. In order to define the mechanisms involved in osteoporosis and to avoid confusion, osteoporosis can be categorized into two types. Each type has distinctive metabolic characteristics and nutritional requirements, and will be classified as type I or type II.

Type I. Osteoporosis-associated with negative calcium balance due to low calcium intake, decreased absorption, or increased excretion. Elevated urinary calcium excretion is usually due to resorptive hypercalcuria.

Type II. Osteoporosis-associated with adequate calcium intake, decreased or normal excretion with metastic soft tissue calcification. If urinary calcium excretion is elevated, it is often due to absorptive hypercalcuria.

Endocrine Factors and Osteoporosis Postmenopausal:

The most common type of demineralization found in women is termed postmenopausal osteoporosis, which involves a reduction in normal estrogen production. There are several theories as to the effects of estrogen on bone, which include, osteoblastic stimulatory activity, inhibition of parathyroid hormone calcium release from the bone and inhibition of bone resorption. Estrogen therapy has been shown to decrease collagen deterioration in animals, and increase anabolic collagen production.

In a 1984 study, estrogen was shown to apparently reduce the effects of parathyroid hormone on bone resorption. This was indicated by a reduction of serum alkaline phosphatase and urinary hydroxyproline excretion. The study also indicated that serum parathyroid hormone levels, which were elevated prior to estrogen therapy did not decrease significantly during therapy. Serum D3 levels were high upon entry into the study, which did not change, or further elevate, during therapy in most of the patients. This would indicate that intestinal calcium absorption was unchanged. Serum calcium levels were also reduced in the majority of the patients, while serum phosphorus levels remained low.

The effects of estrogen therapy are not well understood. However, one possible explanation of its effects may involve an increase in estrogen mediated insulin secretion. Insulin is anabolic, and the decrease in calcuria may have been due to the estrogen induced insulin secretion. Studies of individual insulin levels were not reported.

Other studies involving estrogen therapy for postmenopausal osteoporosis are somewhat conflicting, and involve certain risks. The poor response to estrogen indicates a type II osteoporotic condition. Type II osteoporosis response to estrogen would appear to have minimal results, that is, maintaining status quo calcium balance. More promise in treating postmenopausal osteoporosis has recently been reported, using both estrogen and progesterone instead of estrogen alone. This would be expected to produce greater response in type II conditions in bringing about a positive calcium balance. Type I osteoporotic conditions would probably show a greater response to estrogen therapy than would type II.

Senile Osteoporosis

Senile osteoporosis occurs in both the male and female, but the term is most often applied to the male. Senile osteoporosis is associated with adrenal insufficiency, and/or decreased androgen production in the male. A reduction in adrenal function results in a decrease in anabolic hormone production, thereby decreasing calcium deposition. Hypercalcemia is frequently found with adrenal insufficiency, which may be due to increased hemoconcentration due to sodium and water loss, or increased osteoclastic activity. Senile osteoporosis can be classified as type II.

Adrenal Hyperactivity

The most serious pathological condition of hyperadrenalcorticism is Cushings' disease, which results in hyperplasia of the adrenal cortex. Excessive production of glucocorticoid hormones increase protein catabolism and subsequently results in decreased skeletal protein deposition and collagen deterioration. Cushings' disease will eventually cause diabetes, which will also promote an anti-anabolic effect, if insulin secretion is suppressed.

Excessive aldosterone secretion from the adrenal cortex increases urinary calcium excretion. Aldosterone increases the predominance of renal tubular sodium reabsorption over that of calcium reabsorption.

Demineralization as a result of adrenal cortical hypertrophy can be classified as type I osteoporosis. The above pathological conditions are the extreme. However a mild increase in adrenal cortical hormone production as a result of physical or emotional stress can contribute to a negative calcium balance and osteoporosis. This is especially true if the stress is sustained over prolonged periods.

Hyperthyroidism

Hyperthyroidism contributes to osteoporosis by increasing urinary calcium excretion, and/or increasing osteoclastic activity. Since hyperthyroid activity increases the metabolic rate, a concomitant elevation of protein use occurs, further contributing to a decrease in collagen

formation and demineralization. Hyperthyroidism contributing to osteoporosis can be classified as type I.

It is interesting to note that hyperthyroidism often occurs following extreme physical or emotional stress, and is found in much greater frequencies in cold climates. Also interesting is the relationship of the thyroid with adrenal cortical function. Thyroxine increases glucocorticoid secretions. Increased thyroid activity reciprocally stimulates the adrenal cortex, and the reverse is also true.

With this relationship in mind, we can see that type I osteoporosis would almost invariably be found to be related to hyperadrenia and hyperthyroidism. Further analysis of this reciprocal relationship also reveals that adrenal insufficiency and hypothyroidism would also be found concomitantly with type II osteoporosis.

Parathyroid

The parathyroid glands regulate not only the blood calcium and phosphorus levels, but also exert an effect upon their absorption. Increased parathyroid activity produces an increase in calcium absorption from the intestinal tract, as well as an increase in the renal excretion of phosphorus. Conversely, hypoparathyroidism results in increased phosphorus retention relative to calcium. Hyperparathyroidism results in soft tissue calcification, and can be classified as a type II related condition.   The thyroid and parathyroid glands appear to oppose each other's action. Hypothyroid patients have frequently been found to have elevated parathyroid hormone levels in the presence of normal serum calcium. Hyperthyroidism appears to decrease parathyroid activity.

Thyrocalcitonin antagonizes the effects of the parathyroid hormone. One would suspect that when a patient presents a clinical picture of hypothyroidism, a corresponding hyperparathyroidism, type II condition would also exist.   A type I osteoporotic condition would be seen in patients with hyperthyroidism with a co-existing hypoparathyroid condition.

Primary hyperparathyroidism, which is often asymptomatic, is becoming more widely recognized. Only mild hypercalcemia may be present. It is estimated that two-thirds of all patients found to have primary hyperparathyroidism are postmenopausal women. We can again assume that the majority of postmenopausal women who have an osteoporotic condition, are of the type II classification.

Physical Findings, Type I and Type II

There are specific clinical findings associated with type I and type II osteoporotic individuals.   Being aware of these differences will aid the clinician in determining the biochemical, physiological and endocrine involvements of the patient.

We are reminded that the majority of patients suffering from or having tendencies toward osteoporosis, are in the early or primary stages of endocrine imbalance. Evidence of these imbalances are not easily discernable, since they are not overt pathological conditions.

It is agreed by most authorities that osteoporosis can only be detected radiographically when there is at least a thirty to fifty percent reduction of bone mass. Studies have shown that resorption activity is greater in cortical bone in hyperthyroid states, as compared to trabecular bone resorption. In hyperparathyroid conditions, the trabecular bone resorption is greater. These distinguishing characteristics may be evident radiographically, which may help in identifying a type I or type II condition.

Type I patients can be identified by the following characteristics, which are associated with primary or subclinical endocrinopathies, and may include; Hyperthyroid in conjunction with hyperadrenia and hypoparathyroidism. Warm body temperature due to increased metabolic rate, increased perspiration, anxiety, noise sensitivity, hyperreflexia, and tachycardia. The patient may complain of calcium deficiency symptoms, which include muscle cramps, (especially noted at night), insomnia, nervousness and irritability. Hyperthyroidism is frequently found to produce an elevation of systolic blood pressure. Depending upon the degree of adrenal hyperactivity, a rise in diastolic blood pressure may or may not be present.

Type II conditions include hyperparathyroidism, in conjunction with hypothyroidism and adrenal insufficiency, and may show the following signs depending upon the degree of endocrine involvement: Fatigue, cold body temperature (especially noted in the hands and feet), due to the reduction in metabolic rate, hypotension (especially postural), hyporeflexia, and bradycardia. Diastolic blood pressure may rise and remain elevated depending upon the degree of hypothyroidism. Soft tissue calcification is often seen in the lymph nodes, and gall bladder.

The degree of these physical manifestations, and the extent of osteoporosis development would depend upon the chronicity of endocrine activity and their ratio dominance to each other, which include the adrenal/thyroid, parathyroid/thyroid, adrenal/parathyroid, pancreas/thyroid relationship, rather than the degree of endocrine function alone. Pathological manifestations of severe endocrine imbalances associated with osteopathologies are readily recognized and include, rickets, Paget's disease and osteomalacia.

Nutritional Factors Involved in Osteoporosis

There are several nutritional factors required for the normal mineralization and demineralization process of osseous structures. A disturbance of any one, or a combination of these factors can contribute to osteoporosis. The reverse is also true, in that an improvement in nutrition can be of benefit in the treatment or prevention of this condition.

Protein
Protein will be discussed ahead of calcium, phosphorus and vitamin D, due to its importance in making up the organic matrix of bone. Protein comprises approximately thirty percent of bone and usually receives little attention in relationship to osteoporosis. The organic matrix of osseous tissue is made up largely of collagen fibers in which mineral salts are deposited. Any factor interfering with normal protein metabolism can contribute to demineralization. These conditions were discussed previously, which include, increased catabolism of proteins, increased utilization, and decreased anabolic activity. Adequate protein in the diet as well as its digestion, absorption and metabolic utilization is very important in the treatment of osteoporosis.

It is known that inadequate protein intake and utilization can adversely affect normal endocrine function. Protein deficiency as a result of inadequate intake or synthesis can contribute to metastatic calcification, which is most frequently found in type II conditions.

Calcium and Phosphorus
It has been estimated that a negative calcium balance of as little as 40 milligrams per day can result in the bone loss of calcium at the rate of 1.5 percent per year. This could result in a substantial net loss over a 20 to 30 year period.

Calcium metabolism is controlled by dietary availability, the absorption of adequate calcium and phosphorus from the diet, and the level of calcium and phosphorus in the blood. Plasma levels are controlled by renal and parathyroid function, vitamin D and its metabolites, as well as the availability of stored calcium and phosphate in the bone. Calcium intake and availability from the diet does not insure absorption or deposition into the bone. Achlorhydria decreases calcium absorption and if chronic, can contribute to osteoporosis.

Foods that may impede calcium absorption include spinach, rhubarb, cocoa, chards and beet greens. The oxalic acid content of these foods can bind with calcium in the intestine, producing insoluble salts, which cannot be absorbed. Grains and cereals containing phytic acid may also hinder absorption. High fiber intake can reduce absorption not only of calcium, but also magnesium, phosphorus and zinc. Casual consumption of these foods should present no serious problem, however, over-indulgence may significantly reduce absorption. Alcohol ingestion as well as high sodium intake increases urinary calcium excretion and should be limited in the treatment of osteoporosis, especially type I conditions.

Calcium supplementation is generally accepted as a course of therapy for osteoporosis. However, studies have shown that calcium supplementation alone over extended periods has resulted in decreased calcium retention. This is probably due to not taking into consideration the other mechanisms involved in a particular osteoporotic individual.

Mineral salts are deposited into the bone as hydroxyapetites, principally a calcium-phosphorus complex. Other minerals are also present, including magnesium, sodium and potassium. An imbalance in the intake of any of these minerals can adversely affect calcium and phosphorus absorption, deposition, resorption, and excretion.

Urinary calcium excretion can be affected by mechanical factors. Immobilization or prolonged bed rest increases urinary calcium excretion. Exercise decreases calcium excretion. At least a moderate exercise regime should be included in the treatment of osteoporosis.

There are over thirty factors that can contribute to hypercalcuria that are not always related to osteoporosis directly. Increased urinary calcium excretion should be distinguished from absorptive or resorptive hypercalcuria. It has been shown that urinary hydroxyproline/creatinine, calcium/creatinine and serum hydroxproline/creatinine ratios indicate bone resorption. This may be a better indication than hypercalcuria alone, since the majority of osteoporotic patients do not manifest hypercalcuria.

Keeping in mind the endocrine effects on calcium and magnesium absorption and ex-cretion, one can determine calcium needs depending upon the determination of a type I or type II condition.

Hyperthyroidism and increased adrenal activity leads to decreased intestinal calcium absorption with increased phosphorus reabsorption and hypercalcuria, resulting in a negative calcium balance, with an increase in hydroxyproline/creatinine ratio. This would indicate a type I condition, requiring calcium supplementation.

Primary hyperparathyroidism with hypothyroid activity, increases calcium retention and decreases phosphorus reabsorption by the kidneys. Hypercalcuria may or may not be present, with the other urinary indicators. This would be considered type II osteoporosis, which would not respond well to calcium supplementation.

Vitamin D
Vitamin D3 has long been known to increase calcium absorption. Although the exact mechanism is not clearly understood, it is thought that vitamin D acts by increasing cellular permeability or is involved in the production of specific calcium binding proteins.

Parathyroid hormone is required for the conversion of vitamin D into its active form, which is the mechanism involved in increasing intestinal absorption by the parathyroid secretions. It has been found that plasma vitamin D3 metabolites are elevated in hyperparathyroid states. Vitamin D activity is similar to PTH activity, in that given in large amounts can cause bone resorption. Hypervitaminosis D also increases soft tissue deposition of calcium. It has also been found that plasma vitamin D3 metabolite levels are reduced in hyperthyroid patients, and increased in hypothyroid patients.

This further implicates that hyperthyroidism frequently occurs simultaneously with hypoparathyroidism, and that hyperparathyroidism frequently occurs simultaneously with hypothyroidism.

Vitamin D3 requirements can be said to be increased in type I osteoporotic conditions, involving hypoparathyroidism, hyperthyroidism and excessive adrenal cortical activity. Vitamin D3 requirements are decreased in type II conditions involving hyperparathyroidism in conjunction with hypothyroidism.

Magnesium
The similarities that exist between calcium and phosphorus also exist between calcium and magnesium. Calcium and magnesium are synergistic in their metabolic function, however, they are antagonistic to each other in intestinal absorption. An excess in calcium intake can lead to a magnesium deficiency, and visa-versa.

Magnesium, calcium and phosphorus are a triad, which should always be considered together. An imbalance of any one of these minerals will have adverse effects upon the other two.

Magnesium, as with most other minerals, responds readily to hormonal regulation. Hypomagnesemia is associated with hyperthyroidism. Increased parathyroid activity producing hypercalcemia results in a relative magnesium deficiency, even though magnesium absorption is also increased. Magnesium has been used to decrease the effects of hyperparathyroid activity. Excessive adrenal cortical activity produces a magnesium loss.

Magnesium balance can be greatly affected by dietary factors. Excessive protein intake increases magnesium requirements, and alcohol causes extensive magnesium loss through the urine.

Magnesium supplementation is indicted in both the type I and type II forms of osteoporosis. In type I, a frank magnesium deficiency is present, along with calcium. Therefore, calcium and magnesium should be supplemented together. Type II conditions are associated with a relative magnesium deficiency and supplementation can aid in decreasing parathyroid-induced calcium resorption from the bone, as well as hypercalcemia.

Vitamin C and Copper
The effects of vitamin C deficiency have been well documented in producing adverse effects on collagen synthesis as seen in studies involving scurvy. A deficiency of vitamin C results in decreased bone matrix production, thereby contributing to osteoporosis. Proline hydroxylase is an enzyme that is necessary for collagen synthesis and is dependent upon adequate amounts of vitamin C.

The mineral copper is closely associated with vitamin C, both synergistically and antagonistically. One of the earliest signs of copper deficiency includes osteoporosis. The cross linking of collagen is found to be reduced in a copper deficiency state. This cross-linking is essential for normal collagen connective tissues of the bone matrix.

A number of enzymes involved in collagen synthesis require copper. Many of the conditions associated with copper deficiency, including bone abnormalities, are seen in Menkes disease, which is an inherited, inborn error of copper metabolism.

Ascorbic acid oxidase is a copper dependent enzyme. Increased tissue copper levels would result in an increased oxidation of vitamin C, resulting in a vitamin C deficiency, even with adequate vitamin C intake. Conversely, excessive amounts of vitamin C can deplete copper levels, resulting in scorbutic symptoms.

Type II conditions should respond well to Vitamin C, but not to copper. A type I condition should respond to copper, but not to high amounts of Vitamin C.

Zinc

Zinc is required for protein synthesis, and collagen abnormalities have been found in zinc deficiency states. An antagonistic relationship also exists between zinc and copper. The result of excess intake or a deficiency of one mineral over the other should be obvious. Determining the zinc/copper status is highly recommended in the treatment of osteoporosis.

Lead

The toxic metal lead is known to interfere with collagen synthesis. Exposure to excess lead from occupational or environmental sources can lead to a negative calcium balance, especially if calcium intake is marginal.

Cadmium

Cadmium has been shown to decrease the mineral content of bone, and thereby contribute to osteoporosis. Osteomalacia and pseudofractures have been found in workers who developed cadmium toxicity as a result of battery manufacturing. Further evidence of the adverse effects of cadmium on bone has been seen in people living near the cadmium polluted Jintsu river, in Japan. Cadmium, being antagonistic to zinc would also interfere with normal protein synthesis.

Tissue Mineral Analysis as a Model for Determining Osteoporotic Tendencies

A plausible model for determining osteoporotic tendencies and typing can be presented through macro and micro mineral patterns found in biopsied human tissue. Hair is the tissue of choice, for obvious reasons. It is easier to obtain than other tissues, such as skin, organ, or bone. Laboratory testing is economically feasible and it is easily sampled and transported. Tissue mineral studies are more advantageous than blood mineral determinations for several reasons. Blood serum levels fluctuate from moment to moment due to normal diurnal rhythms, sampling techniques. Exercise, acute or chronic conditions, such as inflammation, infections and malignancies. Serum minerals are maintained at the expense of tissue levels and only reflect extracellular activity. Tissue mineral analysis is not without its disadvantages as well. These include, improperly obtained samples, as hair obtained from different areas of the body will give conflicting results. External contamination can occur from dyes, occupational exposure and sampling tools, improper laboratory procedures and the inability to properly interpret laboratory results.

These problems should be given serious consideration when testing hair samples, in order to receive the most accurate results. Obtaining the samples from the same location will

give the most consistent results, which is especially important when making follow-up comparisons. Samples should not be submitted without noting any hair preparations being used by the patient. Only stainless steel instruments should be used in obtaining the sample. Specimens should only be sent to a federally licensed clinical laboratory. Clinically experienced consultations should also be provided to the doctor to aid in interpretation.

Over the past fifteen years, hair mineral testing has been extensive. Conservative estimation of how many multi-elemental tests have been performed during this time would probably exceed one million. Laboratory techniques are probably at their best with this extensive experience. Therefore, sufficient data is available for the acceptance of reference ranges established by the major laboratories in this country.

Considerable evidence has been presented which supports the fact that tissue mineral concentrations found in hair reflects intake, and that testing hair mineral concentrations is applicable for evaluating body stores of minerals.

As with any diagnostic test, there are limitations and laboratory results have to be carefully interpreted. Low levels of a mineral found in the hair do indicate a deficiency, but a normal level does not necessarily rule out a deficiency. This is similar to blood serum and plasma mineral tests results, in which a low, high, or normal value does not necessarily indicate a deficiency, normal or excess respectively. These studies indicate that the only reliable way to confirm an absolute mineral deficiency, is through response to therapy.

The value of hair mineral assays is not to establish a diagnosis of absolute deficiencies, but to reveal relative deficiencies and imbalances. Mineral ratio determinations are of greater importance than individual levels alone. Since minerals are synergistic and antagonistic, relative excesses and deficiencies can readily be determined from tissue mineral studies, in conjunction with the patient's history and other clinical data, as well as response to therapy. Tissue mineral analysis can be one of the most valuable tools in recognizing mineral nutritional requirements.

Due to the fact that the endocrine glands govern mineral metabolism and the minerals affect endocrine function, tissue mineral patterns found in the hair can serve as an acceptable model in determining body mineral ratio stores and endocrine effects.

Tissue Mineral Patterns of Type I and Type II Conditions

In order to distinguish mineral patterns associated with either type I or type II conditions, ideal tissue mineral levels should be recognized. The ideal level is arrived at by determining the mean of the reference range. Since reference ranges are established by each individual laboratory, the ideal or mean may vary slightly from one laboratory to another. The ideal or mean level will be used in order to more clearly recognize mineral ratio determinations.

Ideal ratios* are:

Calcium/Phosphorus (Ca/P)	2.63
Calcium/Potassium (Ca/K)	4.2
Sodium/Magnesium (Na/Mg)	4.0
Calcium/Magnesium (Ca/Mg)	7.1
Zinc/Copper (Zn/Cu)	8.0
Sodium/Potassium (Na/K)	2.4
Calcium/Lead (Ca/Pb)	84
Zinc/Cadmium (Zn/Cd)	500

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*Ideal ratios obtained from research conducted by Trace Elements, Inc.

Type I

The tissue mineral pattern associated with type I conditions involves a lowered Ca/P ratio, with a low Ca/Pb and low Zn/Cd. Calcium and magnesium levels are usually found below the ideal, while sodium and potassium levels are usually above ideal levels. Phosphorus may or may not be elevated above the mean. This pattern would suggest the following endocrine influence. Increased adrenal and thyroid activity, with decreased parathyroid activity, and lowered insulin levels.

Increased adrenal activity is suggested by a number of indicators in this pattern. First the elevated sodium and potassium relative to the low calcium and magnesium (low Ca/K and high Na/Mg), suggest increased cellular retention of sodium and potassium, as a result of increased adrenal function. Increased epinephrine levels will produce potassium retention within the cells, which is mediated by Na-K ATPase. Sodium retention occurs as a result of an increase in the adrenal cortical production of aldosterone, due in response to potassium retention. Excess glucocorticoids and aldosterone both increase calcium and magnesium excretion. It is known that excessive aldosterone secretion induces magnesium loss. It is also possible that a magnesium deficiency can promote excess aldosterone secretion.

Corticosteroids also interfere with vitamin D metabolism, which could further account for the low tissue calcium levels.

Increased thyroid activity promotes magnesium loss, probably due to the reciprocal relationship between the thyroid and adrenal glands. The effect of hyperthyroidism on calcium has been described previously, as well as the opposing thyroid/parathyroid relationship.

This mineral pattern also suggests hypoparathyroidism. Adrenal steroids, in particular, glucocorticoids, antagonize the effects of parathyroid hormone. Copper deficiency is frequently seen due to increased adrenal stimulation.

Type II

The tissue mineral pattern model for type II osteoporosis reveals an elevated Ca/P, Ca/Mg, and Ca/K ratios, with a low Na/Mg ratio. Calcium and magnesium are usually elevated above the mean, while sodium, potassium and phosphorus are below the mean range.

This pattern suggests increased parathyroid activity, hypothyroidism, adrenal insufficiency and increased insulin secretion.

Parathyroid hormone activity influences this pattern due to its effects of increasing calcium and magnesium reabsorption with decreased renal reabsorption of sodium, potassium and phosphorus. The parathyroid exerts a greater influence on calcium than magnesium. Therefore a relative magnesium deficiency usually exists in this pattern. As a result of magnesium deficiency, parathyroid hormone activity is increased.

Decreased adrenal activity is indicated by the elevated tissue magnesium, with corresponding low levels of both sodium and potassium. Excess magnesium is known to decrease adrenal function.

In the hypothyroid state, intestinal calcium absorption is increased, while renal phosphorus reabsorption is decreased.

Insulin secretion is affected by relative calcium to magnesium levels. A high Ca/Mg ratio indicates increased insulin secretion, while a low Ca/Mg ratio indicates reduced insulin secretion.

Mild endocrine disturbances are often impossible to detect, especially if the patient is asymptomatic. Tissue mineral analysis can serve as an economical screening tool in assessing endocrine influence on mineral metabolism. When the results are properly interpreted and applied to the circumstances of the individual patient, hair tissue mineral analysis can indicate a more precise, and conservative nutritional approach in the treatment of osteoporosis.

With continuing research and incorporation of tissue mineral tests as a routine part of the patient exam, further substantiation and usefulness of hair tissue mineral analysis will be realized, thereby improving its reliability and understanding as a primary screening tool for metabolic and endocrine assessment.