

Acid-Alkaline Balance and Its Effect on Bone Health

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In our bones, most North Americans are in woeful shape. Our bone health crisis worsens each year, despite intensive public health and disease treatment efforts. In this article, we will suggest that the preoccupation with the consequences of osteoporosis, rather than a focus on its fundamental causes, underlies our inability to solve the contemporary epidemic of poor bone health.

Osteoporosis can be seen as a "hidden tax of high-tech living." We pay this tax as a consequence of chronic metabolic acidosis, which robs us of our mineral reserves and impairs efforts to rebuild the bone matrix. The basis of, support for, and a comprehensive response to this situation are detailed below.

Following are the basics of bone growth and turnover. Understanding these basics can help prevent or repair osteopenia and osteoporosis, as detailed later in this article.

Bone growth largely halts after puberty. However, "bone tissue in adults is not dormant -- our bones are continuously being remodeled through repeated cycles of destruction and rebuilding."¹ Healthy people balance osteoclastic bone destruction with osteoblastic bone rebuilding.² The normative remodeling half-life of bone is five years. This means that every 10 years a healthy person will have an entirely new skeletal structure.

While bone-remodeling processes constantly attempt to renew bone, the skeletal structure of a typical North American senior citizen is not healthy. In the United States, half of all Caucasian women aged 65 and over, and at least one in five men, will experience one or more osteoporotic fractures during their lifetime.⁴ Currently, about 10,000,000 North Americans have been diagnosed with osteoporosis. An additional 18,000,000 North Americans have osteopenia and face a high risk of bone fracture or related complications. Direct medical costs related to osteoporosis exceed \$14 billion per year. This represents about one-third of the total cost to our nation for osteoporosis-related ill health.

Furthermore, in 1999, 1,167 scientific articles on bone health and osteoporosis were added to the index Medicus database, representing about half of the peer-reviewed studies published on the subject that year. According to the National Institutes of Health (NIH) Public Information Office, \$136.7 million federal dollars were spent in 1999 on osteoporosis research. Despite these great efforts, a solution to our current bone health crisis still evades us, and fracture incidence increases, particularly in younger people.³

The Link Between Osteoporosis and Metabolic Acidosis

From a broad systemic perspective, primary osteoporosis is seen largely as secondary to metabolic acidosis. First, we will discuss the causal link between acid-alkaline balance and bone health, and then we will present options for restoration and maintenance of bone health throughout the full life span.

In contemporary Western society, diet/lifestyle-induced metabolic acidosis is more the rule than the exception. As detailed below, the excess acid load promoting metabolic acidosis is acquired by:

1. Dietary choices (excess protein, fat, phosphate/phosphoric acid, and sulfate/sulfuric acid);
2. Maladaptation to stress (distress-induced excess cortisol and adrenaline); and
3. Immune hypersensitivity (delayed allergy) reactions.

For 80 years, it has been repeatedly confirmed that bone responds to an acid load by dissolving its basic buffering mineral salts. For background, the average adult skeleton contains a large but finite amount of Ca^{2+} (50-65,000 mEq, 99% of total body stores) and Mg^{2+} (1,060-1,600 mEq, 50% to 80% of body stores). Bone minerals serve as a sizeable reservoir of buffer, usable in the control of plasma pH. Extensive research has documented the following⁸⁻¹⁸:

1. Urinary calcium excretion is associated with bone loss.
2. Urinary calcium loss in the face of an acid load strongly suggests cellular potassium and sodium deficits.
3. Bone loss is accelerated in the face of magnesium deficit.
4. Urinary calcium excretion parallels total acid excretion until substantial calcium and magnesium deficits accumulate.
5. Upon significant depletion of buffering mineral salts, compensation for acid load is reduced, intracellular and first-morning urine pH is concomitantly reduced, and the consequences of metabolic acidosis are accelerated. As an equilibrated specimen, first-morning urine pH is a useful clinical approximation of the cellular and systemic acid/alkaline state.

Less appreciated, however, are the following facts:

1. A variety of alkaline buffering salts (including those of sodium, potassium, zinc, and other minerals) are stored in bone. They are also lost from bone in the obligatory buffering of excessive metabolic acids.¹⁹
2. The contribution of contemporary dietary patterns to the induction of excess metabolic acids has been clinically underestimated. These fixed acids, which must be neutralized with alkaline buffering mineral salts, are largely the result of less healthy dietary choices.^{20,21}

3. The mineral deficits in our soil and water reduce the availability of minerals in the conventional food supply.^{22,23}
 4. Compensated chronic metabolic acidosis is more the rule than the exception. The results are depletion of bone tissue and a disposition to chronic illnesses.²⁴⁻²⁸
- Thus, although osteoporosis is a complex and often multi-faceted disorder, we propose that primary osteoporosis is largely secondary to acquired and reversible chronic metabolic acidosis.

Small Change, Big Impact

Bone is sensitive to small changes in pH. *In vitro* studies document that even one-tenth of a point drop in pH does the following.^{64,65}

1. Greatly stimulates osteoclastic activity
2. Inhibits osteoblastic action; and
3. Induces a multifold bone mineral loss.

A 500%-900% increase in osteoclast cell-mediated rat bone resorption was noted with just a 0.2 pH unit change.⁶⁶ Acidosis also induces mineral dissolution, independent of osteoclastic activity. For example, a human study on acute fasting showed a venous pH decrease from 7.37 to 7.33 (4/100th of a pH unit). This caused a significant calcium release from bone, which was independent of osteoclast or PTH activity.⁶⁷

Acid-Alkaline Bone and Body Balance

It has been said that “the body is alkaline by design, but acidic by function.”²⁹ The human body has also been described as largely dilute seawater encapsulated in a membrane skin. Our immune defense and repair mechanisms, and a host of cell and system enzyme catalysts, all do their best in an exquisitely narrow pH range. The healthy pH range of oxygenated arterial blood is 7.35 to 7.45, and that of the carbon dioxide-laden venous blood is 7.31 to 7.41. To remain viable, the body must remain slightly alkaline. The viable human arterial blood pH range is just 7.4 ± 0.5 pH units. Even minor variations from these values are biologically costly.

For intracellular cytoplasmic pH, the healthy range is 7.4 ± 0.1 . An acidic tilt to cellular pH alters cellular metabolism dramatically and adversely. This results in:

- Swelling and impaired function of mitochondrial electron transport, with reduced ATP energy production and more rapid ATP energy consumption;³⁰
- Increase in intracellular free water with less efficient metabolism, protein synthesis, and increased membrane free radical production;³¹
- Increase in interstitial "third space" water (fluid retention), particularly in any susceptible (distressed) organ;³²
- Accelerated bone resorption;³³ Reduced bone formation;³⁴
- Nitrogen wasting (accelerated catabolism); and
- Suppression of growth hormone and other pituitary hormones.³⁶

Although alkaline by design, everyday metabolic processes produce some 70,000 mmol of protons (H⁺) daily. For the most part, these H⁺ do not accumulate in the body because of the body's elegant buffering systems, and because acids are generally formed with a partner that aids in their removal. In fact, while an enormous number of H⁺ are produced daily, most of them are balanced by bicarbonate production. The amount of free H⁺ is tiny, yet significant, in terms of health maintenance and disease risk.

In most individuals, the source of net acid load is from the metabolism of protein (when its consumption exceeds 60g/day) and long-chain fatty acids (when they comprise more than 20 % of calories in the diet). A marker of net acid production is the extent of degeneration of sulfur-containing amino acids: cysteine, cystine, and methionine. More accurately, any of the seven acidic amino acids (aspartate, glutamate, cysteine, cystine, proline/hydroxyproline, serine, and threonine), plus the keto-acids produced from amino acid metabolism, contribute to the body's fixed, organic acid load.³⁷ The metabolism of these amino acids produces H⁺ without buffering partners. These H⁺ accumulate and must be neutralized by matching buffering elements from the body. The buffering elements include the organic anions (usually as K⁺ or other mineral salts) in fruits, vegetables, lentils/pulses, herbs, and spices. These include metabolically alkaline-forming citrate, malate, succinate, and fumarate.³⁸ In addition, short- and medium-chain fatty acids reduce net acid burden by "soaking up" acetate and 2-carbon acidic units in the cells.

The Role of Bone In Systemic Acid-Alkaline Balance

It is well-known that the skeleton contains 99% of the body's calcium. However, bone also contains substantial amounts of sodium, potassium, magnesium, citrate, and carbonate. This means that the bone of a typical, healthy, 70 kg (154 lbs) adult contains.³⁹⁻⁴⁴

1. 1,065-1,400 mmol of sodium = 1,065-1,400 meq sodium (37%-49% of the body's sodium)
2. 22-62 mmol of potassium = 22-62 meq potassium (0.1%-0.2% of the body's potassium)
3. 530-800 mmol of magnesium = 1,060-1,600 meq magnesium (53%-80% of the body's magnesium)
4. 3,500-5,000 mmol of carbonate = 7,000 to 10,000 meq carbonate (59%-83% of the body's carbonate)

Half of these are located on the bone crystalloid surface and in the hydration shell of bone. These buffering minerals are available for rapid exchange with the general extracellular fluid (ECF). The ECF of bone also contains a potassium concentration 25 times that of general ECF, and thus is a major source from which the body can draw potassium. This potassium is neither incorporated into the bone mineral phase, nor bound to collagen. Therefore, it is completely exchangeable with the potassium of systemic ECF. Potassium accumulates both in the bone ECF and in the bone hydration shell and, overall, is about 60% available for immediate systemic mobilization.^{45,46} Thus, a wide range of buffering substances are stored in and around the bone. These are

available to neutralize excess acid products, unless (or until) they become depleted through lack of “alkaline way” replenishment.

Initially, the acid load involves significant changes in the bone content of carbonate, sodium, and potassium, but not calcium. In early phase (fully compensated) acidosis, protons exchange with sodium and potassium, providing a “first line” of buffering defense. Chronic overproduction of acid (chronic metabolic acidosis) leads to depletion of the sodium and potassium buffers. When this occurs, calcium and magnesium cations, along with carbonate, become the major source of buffers.⁴⁷ This means that when we see accelerated calcium and magnesium loss, there has been a prolonged period of excess acid production and depletion of critical sodium and potassium reserves.

Sources of Acidic Load

The major recognized sources of net acid load in the body are:

- 1) Diet
 - a) Protein consumption above 60g/day
 - b) Dietary phosphate/phosphoric acid
 - c) Dietary sulfate
 - d) Long-chain fatty acids in excess of 15%-20% of total dietary calories
- 2) Distress (excess cortisol and adrenaline)
- 3) Delayed immune system reactions (from delayed immune sensitivities/reactions)

Net Acid Excess (NAE) from the North American Diet

Our contemporary diet commonly produces an excess load of fixed acids of 100 to 200 mEq per day.⁴⁸⁻⁵⁰ For example, analysis done by Remer and Manz found that a diet containing 120 grams of protein yielded a net acid excretion of 135.5 mEq/day. Two “moderate” protein diets (95g/day protein) yielded an NAE of 69 to 112 mEq/day. A lactovegetarian “low” protein diet (49g/day protein) yielded an NAE of 24 mEq/day. Thus, dietary choice influences net acid production. High-protein diets produce a six-fold (600%) increase in NAE. This results in low first-morning urine pH, indicating that buffering functional reserve is deficient, and that the risk of metabolic acidosis is correspondingly increased.⁵¹

For example, as Barzel and Massey⁵² calculate, the pH of colas with phosphoric acid is 2.8 to 3.2. However, the kidney cannot excrete urine with a pH much lower than 5, without significantly damaging the genitourinary tract. To achieve a urinary pH of 5, a 12 oz. (330mL) can of cola would have to be diluted 100-fold, requiring an additional 33 liters of urine. Otherwise, a corresponding amount of buffer must be drawn from the body to neutralize the excess acid.

The body routinely buffers the acidic beverage with sodium and potassium if reserves permit, then with a corresponding loss of calcium, magnesium, and other minerals, as available. The buffering needed for one can of cola is the same amount of buffering capacity found in 4 TumsTM tablets. Fruit “spritizers” and naturally carbonated mineral waters, by contrast, do not add this acid burden to the body.

Finally, in addition to the recognized acid-producing precursors, we extend the metabolic balance equation to include the additional acid produced by excess immune (delayed hypersensitivity) reactions, and the effects of distress (excess cortisol and adrenaline). In some individuals, these also add significantly to the total net acid production-excretion. Since calcium is activating (sympathomimetic), supplementation primarily with calcium is clinically both unwise and unproductive. It could accelerate acid production and buffering mineral loss.

Net Acid Excess (NAE) The Body Can Buffer

For their excretion, NAE must be buffered with alkaline agents derived from the diet. Thus, our NAE buffering capacity is diet-dependent. Classic studies show that the body can neutralize about 50 mEq of these fixed metabolic acids per day from an assumed “ideal” North American intake of fruits and vegetables.⁵³ When fruit and vegetable consumption is reduced, less than the 50 mEq of fixed acids can be buffered without going into tissue alkaline reserves. Equally, when protein intake is more than 60g/day, more acid is produced. Today, our daily NAE is commonly two to four times higher than this standard 50 mEq buffering potential. Essentially, all excess acids must be buffered at the expense of bone-buffering reserves. If not replenished, the loss of buffering causes a slow, persistent loss of bone mineral matrix. This accelerates osteopenia and osteoporosis complication risks.

Just how low is our intake of alkaline precursors? Only 15% of the total United States population meets the fruit (2-4 servings) and vegetable (3-5 servings) recommended intakes on a daily basis.⁵⁴ Among children, only 7% consume two fruit servings and three vegetables per day, with french fries accounting for nearly 25% of vegetables, in diets of the children surveyed.⁵⁵

Furthermore, an average North American teenager consumes three to six “cola” beverages a day. This additional daily consumption of 192-384 mEq of phosphoric acid (64 mEq per can of soda, times three to six per day) further accelerates bone loss in the young. It takes years for the cumulative load to present with clinically significant complications. However, it is likely that in coming decades we will see an acceleration of bone loss, and loss of body buffering competencies, in younger and younger people.

Acidosis and Osteoporosis

Support for the hypothesis that compensated metabolic acidosis is a foundational cause of osteoporosis comes from many sources. For some time, epidemiological studies have suggested the link between osteoporosis and animal protein intake.⁶⁸ More recently, analyses of cross-cultural fracture rates document the link between the consumption of animal protein and the incidence of hip fracture worldwide.⁶⁹ In addition, new studies report that those who consume more fruits and vegetables have higher bone mineral density than those consuming fewer of these “Alkaline Way®” foods.^{70,71} Also of note

are new studies showing a three- to five-fold increase in fractures among teenage girls who regularly consume acidic soft drink beverages.^{72,73}

Bone Loss Through Buffering Excess Metabolic Acids

In a study of vegetarian and animal protein diets, it was found that urinary pH was more acidic (6.17 vs. 6.55), net acid excretion was 27 mEq/day higher, and daily urinary calcium excretion was 47mg higher in those consuming animal protein. This was in spite of the fact that the diets contained the same amounts of total protein, phosphorus, sodium, potassium, and calcium. The animal protein diet, however, contained 6.8 mmol more sulfate.⁵⁶ In another study among the elderly, calcium balance was positive (+40mg/day) on a low-protein diet of 0.8 grams of protein per kilogram of body weight (56g/day for a 70 kg adult). In contrast, calcium balance was negative (-64mg/day) on a high-protein diet of 1.2 grams of protein per kilogram of body weight (84g/day for a 70 kg adult).⁵⁷ Higher protein intake would lead to even greater losses of calcium, magnesium, and other minerals.

A 50 or 60mg daily loss of calcium might not seem like much. However, over 20 years, a daily 50mg loss of calcium would translate into depletion of 365 grams of calcium, which is one-half of the average female skeletal calcium and one-third of the male's.⁵⁸ Indeed, it is not uncommon for women to lose half of their bone mass, and men, a third of their bone mass, during their lifetimes. Thus, it is possible to explain the induction of osteoporosis in our population from the causes cited above.

What is clinically underappreciated is that such loss is avoidable, witnessed by the fact that osteoporosis is uncommon in many cultures. For example, even after careful study, no sign of bone loss can be found among the Maya Indians, who eat an alkaline-rich diet.⁵⁹ Africans have been classified as “almost immune” to osteoporotic fracture.^{60,61} The Chinese were found to have only one-fifth the U.S. fracture rate, despite eating as much, or nearly as much protein (but predominantly from plant sources).⁶² Societies with the lowest osteoporosis rates follow the suggestions for risk reduction contained in this article. All in all, the osteoporotic fracture rate varies some 30-fold around the world.⁶³ This is almost entirely explainable by diet and lifestyle choices.

Clinical Implications

The contemporary Western diet leads to chronic, low-level acidosis, to the detriment of basic health and well-being. Of particular interest here is that this acidosis first forces the loss of alkalizing sodium and potassium, and then carbonate, calcium, magnesium, and other minerals from bone stores. These losses lead to excessive bone weakness, osteopenia, and osteoporosis. The solution to this problem lies in a return to a sustainable diet, rich in alkaline precursors. Consistent, weight-bearing exercise is also essential. Guidelines for development of such an “Alkaline Way® diet” are as follows:

1. The bulk of the diet should be alkalizing vegetables, fruits, lentil/pulses, nuts, seeds and spices. The chart on p.12 illustrates the effects of foods on internal

acid/alkaline balance. Sixty to 80% of foods eaten should be from the alkaline side.

2. Limit animal flesh to four ounces per day, and restrict total protein intake to 50 to 60 grams per day.
3. Maintain a fat intake of no more than 15 to 20% of total calorie intake.
4. Drink 64 ounces of high mineral (highly dissolved solids) spring water daily.
5. Fresh vegetable juice is an exceptionally good source of buffering minerals. Those with persistent low-grade acidosis might drink 2-3 eight-ounce glasses a day. We calculate that 16 to 24 ounces of juice from organic vegetables would be sufficient to correct for 40 to 50 mEq of excess organic acid.
6. Use alkalizing nutritional supplements, such as bioavailable, ionized minerals and a high quality, antigen-free ascorbate buffered with calcium, magnesium, zinc, and potassium. Add L-glutamine with pyridoxal alphaketoglutarate (PAK), Krebs' salts, cesium, rhubidium, and sesame/flax seeds as needed to keep a healthy first-morning urine pH.
7. Modify the diet and supplement sufficiently to obtain and maintain a first-morning urine pH of 6.5 to 7.5, which may reflect the existence of adequate buffering mineral reserves.
8. Reverse learned patterns of distress (sympathetic hyperactivity) by practicing relaxation responses, enjoyable activities, and weight-bearing exercise.
9. Restore tolerance to the immune defense and repair systems.

Final Thoughts

Maintenance of healthy bone and cellular pH over a lifetime is a matter of small, yet deeply important choices. Half of all older women and 20% or more of older men will suffer fractures that can, in large measure, be avoided by following the suggestions in this article. Substantial improvement in quality of life, and reduced treatment costs of \$14 to \$60 billion per year, are some of the benefits to be harvested.

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