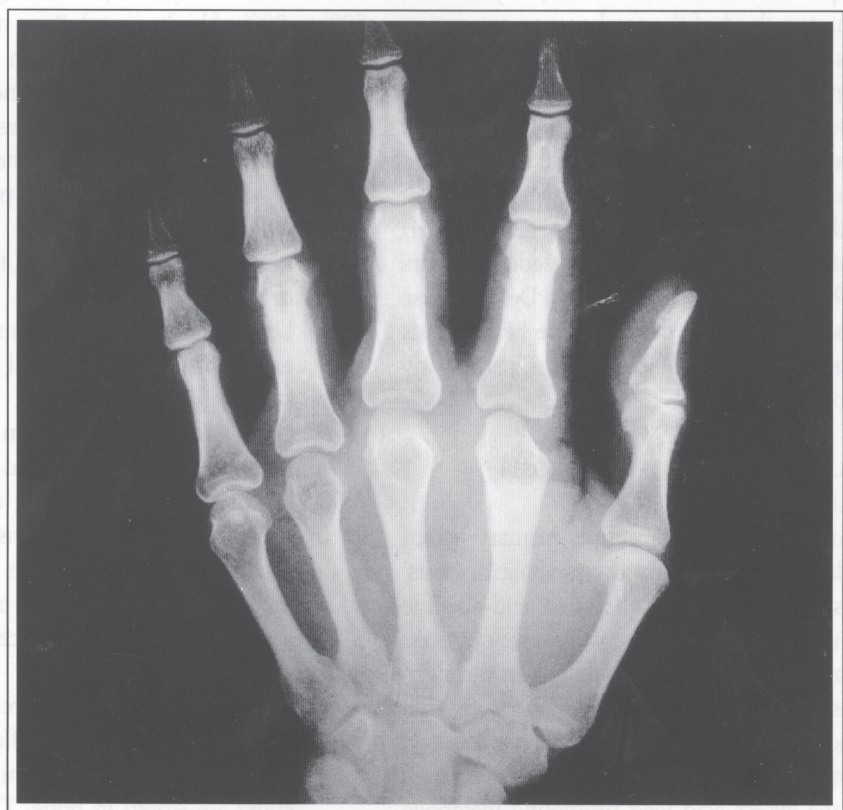


ACID-ALKALINE BALANCE AND ITS EFFECT ON BONE HEALTH

by Susan E. Brown, Ph.D., C.C.N., and Russell Jaffe, M.D., Ph.D., C.N.N.



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 this helps prevent or repair osteopenia and osteoporosis, 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 destruction² with osteoblastic bone rebuilding.³ 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.⁵

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);

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

...THE EXTENT TO WHICH CONTEMPORARY DIETARY PATTERNS PRODUCE EXCESS METABOLIC ACIDS HAS BEEN UNDERESTIMATED.

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.⁶

For background, the average adult skeleton contains a large but finite amount of Ca²⁺ (50-65,000 mEq, 99% of total body stores) and Mg²⁺ (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.⁷ Extensive research has documented the following:⁸⁻¹⁸

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.

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);³⁵ 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,

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)

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 already been a prolonged period of excess acid production and depletion of critical sodium and potassium reserves.

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

...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.

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 meta-

bolic acidosis is correspondingly increased.⁵¹ (See sidebar for the sources of net acid load in the body.)

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 a corresponding loss of calcium, magnesium, and other minerals, as available. This is the same amount of buffering capacity found in 4 Tums™ 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"⁶⁹ 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}

Food & Chemical Effects on Acid / Alkaline Body Chemical Balance

Most Alkaline	More Alkaline	Low Alkaline	Lowest Alkaline	Food Category	Lowest Acid	Low Acid	More Acid	Most Acid
• Baking Soda	Spices/Cinnamon Valerian Licorice • Black Cohosh	• Herbs (most): Amica, White Willow Bark Bergamot, Echinacea, Slippery Elm Chrysanthemum, Artemisia Annua Ephedra, Fevewef, Goldenseal, Lemongrass	White Willow Bark Slippery Elm Artemisia Annua	Spice/Herb	Curry	Vanilla Stevia	Nutmeg	Pudding/Jam/Jelly
Sea Salt Mineral Water	• Kambucha Molasses Soy Sauce	<i>Sulfite</i> Ginger Tea • Sucanat • Umeboshi Vinegar • Algae, Blue-Green • Ghee (Clarified Butter) Human Breast Milk		Preservative Beverage Sweetener Vinegar Therapeutic	MSG Kona Coffee Honey/Maple Syrup Rice Vinegar Cream/Butter Yogurt Goat/Sheep Cheese Chicken Egg	Benzoate Alcohol Black Tea Balsamic Vinegar Cow Milk Aged Cheese Soy Cheese Goat Milk	Aspartame Coffee Saccharin Psychotropics • Casein, Milk Protein, Cottage Cheese New Cheese Soy Milk	Table Salt (NaCl) Beer; 'Soda' Yeast/Hops/Malt Sugar/Cocoa White/Acetic Vinegar Antibiotics Processed Cheese Ice Cream
• Umeboshi Plum	• Sake	• Quail Egg	• Duck Egg	Processed Dairy Cow/Human Soy Goat/Sheep Egg				
				Meat Game Fish/Shell Fish Fowl	Gelatin/Organs • Venison Fish Wild Duck • Triticale Millet Kasha • Amaranth Brown Rice Pumpkin Seed Oil Grape Seed Oil Sunflower Oil Pine Nut Canola Oil	Lamb/Mutton Boar/Elk/•Game Meat Shell Fish/Mollusks Goose/Turkey Buckwheat Wheat • Spelt/Teff/Kamut Farina/Semolina White Rice Almond Oil Sesame Oil Safflower Oil Tapioca • Seitan or Tofu	Pork/Veal Bear Mussel/Squid Chicken Maize Barley Groat Corn Rye Oat Bran Pistachio Seed Chestnut Oil Lard Pecan Palm Kernel Oil Green Pea Peanut Snow Pea	Beef Lobster Pheasant Barley Processed Flour
				Grain Cereal Grass				
				Nut Seed/Sprout Oil				
				Bean Vegetable Legume Pulse Root	Spinach Fava Bean Kidney Bean Black-eyed Pea String/Wax Bean Zucchini Chutney Rhubarb	Split Pea Pinto Bean White Bean Navy/Red Bean Aduki Bean Lima or Mung Bean Chard	Legumes (other) Carrot Chick Pea/Garbanzo	Soybean Carob
				Citrus Fruit	Coconut Guava • Pickled Fruit Dry Fruit Fig Persimmon Juice • Cherimoya Date	Plum Prune Tomato	Cranberry Pomegranate	
				Fruit				

• **Therapeutic, gourmet, or exotic items**

Italicized items are NOT recommended.

7. Lemann J Jr, Lennon EJ: Role of diet, gastrointestinal tract, and bone in acid-base homeostasis. *Kidney Int* 1:275-279, 1972.
8. Barzel US, Massey LK: Excess dietary protein can adversely affect bone. *J Nutr* 128:1051-1053, 1998.
9. Anand C, Linkswiler H: Effect of protein intake on calcium balance of young men given 500mg of calcium daily. *J Nutr* 104:695-700, 1974.
10. Chu J, et al: Studies in calcium metabolism II. Effects of low calcium and variable protein intake on human calcium metabolism. *Am J Clin Nutr* 28:1028-1035, 1975.
11. Johnson NE, et al: Effect of protein intake on urinary and fecal calcium and calcium retention of young adult males. *J Nutr* 100:1425-1430, 1970.
12. Margen S, et al: The calciuretic effect of dietary protein. *Am J Clin Nutr* 27:548-558, 1974.
13. Schwartz R, et al: Effect of magnesium and protein level on calcium balance. *Am J Clin Nutr* 26:519-523, 1973.
14. Walker RM, Linkswiler HM: Calcium retention in the adult human male as affected by protein intake. *J Nutr* 102:1297-1302, 1972.
15. Heaney RP, Recker RR: Effects of nitrogen, phosphorus, and caffeine on calcium balance in women. *J Lab Clin Med* 99:46-55, 1982.
16. Bushinsky DA: Metabolic alkalosis decreases bone calcium efflux by suppressing osteoclasts and stimulating osteoblasts. *Am J Physiol* 271:F216-F222, 1996.
17. Heaney RP: Excess dietary protein may not adversely affect bone. *J Nutr* 128:1054-1057, 1998.
18. Kleinman JG, Lemann J Jr: Acid production. In: Maxwell MH, et al (eds): *Clinical Disorders of Fluid and Electrolyte Metabolism, 4th Edition*. New York: McGraw Hill, 1987, pp. 159-173.
19. Green J, Kleeman CR: The role of bone in the regulation of systemic acid-base balance. *Contrib Nephrol* 91:61-76, 1991.
20. Ibid.
21. Remer T, Manz F: Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. *Am J Clin Nutr* 59:1356-1361, 1994.
22. Green, op. cit.
23. Remer, op. cit.
24. Ibid.
25. Frassetto L, Sebastian A: Age and systemic acid-base equilibrium: analysis of published data. *J Gerontol* 51:B91-B99, 1996.
26. Lennon EJ, et al: The effect of diet and stool composition on the net external acid balance of normal subjects. *J Clin Invest* 45:1601-1607, 1966.
27. Kurtz I, et al: Effect of diet on plasma acid-base composition in normal humans. *Kidney Int* 24:670-680, 1983.
28. Schwarz WB, Relman AS: A critique of the parameters used in the evaluation of acid-base disorders. *New Engl J Med* 268:1382, 1963.
29. Szent-Gyorgy A: *Quantum Biochemistry*. New York: Blackwell, 1957.
30. Jaffe R: Autoimmunity: clinical relevance of biological response modifiers in diagnosis, treatment, and testing. *Int J Integr Med* 2(2):7-14, March/April 2000.
31. Ibid.
32. Ibid.
33. Sebastian A: Improved mineral balance and skeletal metabolism in postmenopausal women treated with potassium bicarbonate. *N Engl J Med* 330:1776-1781, 1994.
34. Ibid.
35. Frassetto L, et al: Potassium bicarbonate reduces urinary nitrogen excretion in postmenopausal women. *J Clin Endocrinol Metab* 82:254-259, 1997.
36. Ibid.
37. Halperin ML, Goldstein MB: *Fluid, Electrolyte, and Acid-Base Physiology: A Problem-Based Approach, 3rd Edition*. Philadelphia: WB Saunders Company, 1999.
38. Ibid.
39. Green, op. cit.
40. Cohn SH, et al: Body elemental composition: comparison between black and white adults. *Am J Physiol* 232:E419-E422, 1977.
41. Armstrong WD, Singer L: Composition and constitution of the mineral phase of bone. *Clin Orthop* 38:179-190, 1965.
42. Flynn MA, et al: Total body potassium in aging humans: a longitudinal study. *Am J Clin Nutr* 50:713-717, 1989.
43. Poyart CF, et al: The bone CO₂ compartment: evidence for a bicarbonate pool. *Respir Physiol* 25:89-99, 1975.
44. Vatasery GT, et al: Determination of hydroxyl content of calcified tissue mineral. *Calcif Tissue Res* 5:183-188, 1970.
45. Green, op. cit.
46. Triffitt JT, et al: A comparative study of the exchange in vivo of major constituents of bone mineral. *Calcif Tissue Res* 2:165-176, 1968.
47. Green, op. cit.
48. Schwarz, 1963, op. cit.
49. Remer, op. cit.
50. Frassetto, 1996, op. cit.
51. Remer, op. cit.
52. Barzel, op. cit.
53. Halperin, op. cit.
54. Schweitzer C, et al: Dietary intake of carotenoids, fruits, and vegetables in the US: CSFII 1994-1996, a national survey. Proceedings from the 12th International Carotenoid Symposium, July 18-23, 1999, Cairns, Australia.
55. Krebs-Smith SM, et al: Fruit and vegetable intakes of children and adolescents in the United States. *Arch Pediatr Adolesc Med* 150:81-86, 1996.
56. Breslau NA, et al: Relationship of animal protein-rich diet to kidney stone formation and calcium metabolism. *J Clin Endocrinol Metab* 66:140-146, 1988.
57. Licata AA, et al: Acute effects of dietary protein on calcium metabolism in patients with osteoporosis. *J Gerontol* 36:14-19, 1981.
58. Barzel, op. cit.
59. Beyene Y: Cultural significance and physiological manifestation of menopause: a biocultural analysis. *Cult Med Psychiatry* 10:47-71, 1986.
60. Luyken R, Luyken-Koning R: Studies on the physiology of nutrition in Surinam VIII. Metabolism of calcium. *Trop Geogr Med* 13:46-54, 1961.
61. Chalmers J, Ho K: Geographical variations in senile osteoporosis. *J Bone and Joint Surgery* 52B:667-675, 1970.
62. Campbell TC, Cox C: *The China Project*. Ithaca: New Century Nutrition, 1996.
63. Melton L, Riggs B: Epidemiology of age-related fractures. In: *The Osteoporotic Syndrome: Detection, Prevention and Treatment*. New York: Grune and Stratton, 1983.
64. Krieger NS, et al: Acidosis inhibits osteoblastic and stimulates osteoclastic activity in vitro. *Am J Physiol* 31:F442-F448, 1992.
65. Arnett TR, Spowage M: Modulation of the resorptive activity of rat osteoclasts by small changes in extracellular pH near the physiological range. *Bone* 18:277-279, 1996.
66. Arnett TR, Dempster DW: Effect of pH on bone resorption by rat osteoclasts in vitro. *Endocrinology* 119:119-124, 1986.
67. Grinspoon SK, et al: Decreased bone formation and increased mineral dissolution during acute fasting in young women. *J Clin Endocrinol Metab* 80:3628-3633, 1995.
68. Marsh A, et al: Cortical bone density of adult lactovegetarian women and omnivorous women. *J Amer Diet Assoc* 76:148-151, 1980.
69. Abelow BJ, et al: Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. *Calcif Tissue Int* 50:14-18, 1992.
70. Tucker, et al: Potassium, magnesium, and fruit and vegetable intakes are associated with greater bone mineral density in elderly men and women. *Am J Clin Nutr* 69:727-736, 1999.
71. New SA, et al: Dietary influences on bone mass and bone metabolism: further evidence of a positive link between fruit and vegetable consumption and bone health? *Am J Clin Nutr* 71:142-151, 2000.
72. Wyshak G, Frisch RE: Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in girls and boys. *J Adolesc Health* 15:210-215, 1994.
73. Wyshak G: Teenaged girls, carbonated beverage consumption, and bone fractures. *Arch Pediatr Adolesc Med* 154:610-613, 2000.



Susan E. Brown, Ph.D., C.C.N., is a medical anthropologist and certified clinical nutritionist. She directs the Osteoporosis Education Project (OEP) in East Syracuse, N.Y. Dr. Brown conducts primary research, and lectures widely on osteoporosis, teaching a natural, holistic program for the regeneration of bone health. Dr. Brown's publications include *Better Bones, Better Body: Beyond Estrogen and Calcium: A Comprehensive Self-Help Program for Preventing, Halting & Overcoming Osteoporosis* (New Canaan: Keats, 2000) and *The Mend Clinic Book of Natural Remedies for Menopause and Beyond* (Dell, 1997), co-authored with Dr. Paula Maas.



Russell Jaffe, M.D., Ph.D., is an internist. He is board-certified in clinical pathology, with subspecialty board certification in chemical pathology. He is a Fellow of the following medical societies: ASCP, ACAAI, ACN, AMLI, and CCN (for whom he was national program director for nine years). Dr. Jaffe is director of ELISA/ACT Biotechnologies, Inc., and of Seraphim, Inc. He is also a Fellow of the Health Studies Collegium. He works in Sterling, VA.