Dietary, Metabolic, Physiologic, and Disease-Related Aspects of Acid-Base Balance: Foreword to the Contributions of the Second International Acid-Base Symposium

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Abstract

The role of nutrition in human acid-base homeostasis has gained increasing attention in recent years. Although in healthy humans, homeostatic mechanisms and the kidneys' capacity to excrete acid equivalents can prevent strong diet-induced alterations in blood pH, even moderate increases in blood hydrogen ion levels as a result of unfavorable diet composition can have long-term consequences for the occurrence and progression of a number of diseases. The Second International Acid-Base Symposium, Nutrition–Health–Disease, provided deeper insight and updates in the scientific basis of the relation among diet, acid-base homeostasis, physiology, and pathophysiological consequences. J. Nutr. 138: 413S–414S, 2008.

Introduction

Acid-base status is becoming increasingly important in nutritional medicine. The regulation of the pH inside and outside of the cells is essential for enzyme-controlled metabolic processes of the human body. The hydrogen ion concentrations also determine the structure and function of proteins, permeability of cell membranes, distribution of electrolytes, and structure of connective tissue. By excreting the surplus of acid or base equivalents through the kidney and using the ion-binding properties of connective tissue and the minerals of bone as additional buffer systems (1), nearly stable tissue-specific hydrogen ion levels are achieved. During human evolution, the usual diets, even those including abundant animal protein, mostly contained a surplus of base equivalents (2,3). However, after the invention of agriculture and animal husbandry, particularly alkali-rich fruits and vegetables were more and more replaced by net acid-producing animal foods and cereal grains. Therefore, daily net-acid loads of current western diets average 50–100 mEq/d (4,5). As a consequence, conditions of chronic, low-grade metabolic acidosis can develop, which, in the long term, could considerably contribute to impairments of numerous body functions, the best studied of which is the maintenance of bone function (1,6,7). Because the skeleton represents a large but not endless alkaline reservoir, even mild forms of long-term low-grade metabolic acidosis can impair skeletal architecture and stability. The importance of acid-base balance for several physiological functions, the risk of osteoporosis, the aging organism, and hormonal interactions with diet have been discussed as well as epidemiological strategies to monitor acid-base status or emerging difficulties in determining dietary acidity.

The Second International Acid-Base Symposium, 2006, in Munich, Germany brought together scientists from 15 countries to discuss the recent developments in this often overlooked area of nutrition research, which, however, is increasingly being accepted as relevant to the field of preventive medicine. We are pleased to present several of the relevant contributions as a supplement to The Journal of Nutrition and hope that this information may contribute to arouse interest in others in nutrition research to get more involved in this important, directly diet-dependent field of human health.

The mechanisms behind these effects of acid on bone are evaluated in the article from Tim Arnett (8). He discusses how acid influences the activity of osteoclasts and osteoblasts. The insight into these mechanisms that has been elucidated in recent years helps us to understand how small changes in acidity determine the overall loss or gain of bone substance.

An aggravating factor with regard to acidosis induction is the intake of high amounts of sodium chloride. As argued by Lynda Frassetto et al. (9), the high salt together with low potassium intake in the typical American diet substantially contributes to acid-base imbalance.

Aside from strong catabolic effects on bone architecture and bone strength, an acute metabolic acidosis also affects important endocrine functions including functional glucocorticoid activity. Remer et al. (10) examined whether normal variation in net endogenous acid production may already show an association
with potentially bioactive free glucocorticoids in healthy adults. In their study, the authors focused on a new noninvasive marker of functional glucocorticoid activity (11) and took into account additional endocrine-metabolic determinants such as circulating leptin levels and overall daily cortisol secretion (12).

Acid-base metabolism is influenced not only by intakes of protein, alkalizing food constituents, or metabolically noncombustible dietary organic acid; drinking water must also be taken into consideration. The probable impact of differences in drinking water acidity is reviewed in the article from Ragnar Rylander (13). Not only the usual drinking water but also the choice of mineral water influences acid-base balance. Peter Burkhardt et al. (14) actually showed that in several studies in humans, alkali mineral waters decreased bone resorption markers.

Using an animal model, namely the dietary alkali-depleted herbivore rabbit, Heidrun Kiwull-Schöne et al. (13) provided evidence that the exhausted renal base-saving function is one cause for an increased susceptibility to develop chronic metabolic acidosis.

Taken together, an in-depth update on some of the nutritionally relevant areas of acid-base research is presented in the following articles.

**Literature Cited**