CHRONIC LOW-GRADE METABOLIC ACIDOSIS IN NORMAL ADULT HUMANS: PATHOPHYSIOLOGY AND CONSEQUENCES

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Introduction

Normal adult humans eating modern-day diets have a chronic low-grade metabolic acidosis whose severity is determined in part by the net rate of endogenous acid production (NEAP). NEAP varies mainly with diet composition. The greater the quantity of organic and sulfuric acids produced from metabolism of animal foods, and the lower the amounts of potassium salts metabolizable to bicarbonate, which come mainly from fruits and vegetables, the greater the production rate of acid. It had previously been thought that "healthy" kidneys were capable of excreting any excess acid produced by the body's metabolism [1]; the authors' research suggests that the normally occurring slow decline in renal function with age allows the kidneys to merely mitigate the degree of severity of the acidosis, and with increasing age, the steady-state levels of acidity in the body slowly rise [2-3].

This rise in blood acidity levels may be pathogenic in the age-related decline in bone mass that leads to osteoporosis and increased bone fractures. Epidemiologic studies of hip fracture incidence worldwide show an increasing incidence of hip fractures in populations with higher intakes of animal protein, a surrogate marker for dietary acid [4]. In short-term General Clinical Research Center (GCRC) studies of postmenopausal women (weeks to months), neutralizing the acid in the diet with dietary supplements of potassium bicarbonate improves calcium and phosphate balance and improves biochemical markers of bone breakdown [5]. It increases the average 24-hour blood concentration of growth hormone, another factor that declines with age. And, it decreases the amount of nitrogen excreted in the urine, which may reflect a decrease in muscle breakdown. Decreased muscle mass and strength with increasing age are risk factors for falls, and therefore for increased fractures. A simple method for predicting diet acid load from diet protein and potassium content developed by the authors is described. Since future studies will be necessary to show whether long-term neutralization of dietary acid reduces fracture incidence; a simple method to evaluate diet acid load may make this type of study more widely usable to the nonacid-base physiologist.

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