Protein, acid-alkaline, and bone health.

Paul Bergner 3-19-19

Eur J Nutr. 2001 Oct;40(5):231-7. The acid-base hypothesis: diet and bone in the Framingham Osteoporosis Study. Tucker KL, Hannan MT, Kiel DP.

Abstract

BACKGROUND:

There continues to be considerable debate about the role of acid vs. basic components of the diet on the long-term status of bone mineral density.

AIM:

In a set of two analyses, we examined the effect of components in the diet thought to have basic effects (magnesium, potassium, fruit, vegetables) and acid effects (protein) on bone mineral density in an elderly cohort.

METHODS:

Bone mineral density of participants in the Framingham Osteoporosis Study was measured at three hip sites and one forearm site at two points in time, four years apart. At the time of baseline measurement, participants ranged in age from 69-97 years. Dietary intake was assessed at baseline by food frequency questionnaire.

RESULTS:

As hypothesized, magnesium, potassium, fruit and vegetable intakes were significantly associated with bone mineral density at baseline and among men, with lower bone loss over four years. ***In contrast to the hypothesis, higher rather than lower protein intakes were associated with lower bone loss.***

CONCLUSION:

Together these results support the role of base forming foods and nutrients in bone maintenance. The role of protein appears to be complex and is probably dependent on the presence of other nutrients available in a mixed diet. A balanced diet with ample fruit and vegetables and adequate protein appears to be important to bone mineral density.

J Am Coll Nutr. 2005 Dec;24(6 Suppl):526S-36S. Dietary protein: an essential nutrient for bone health.

Bonjour JP.

Abstract

Nutrition plays a major role in the development and maintenance of bone structures resistant to usual mechanical loadings. In addition to calcium in the presence of an adequate vitamin D supply, proteins represent a key nutrient for bone health, and thereby in the prevention of osteoporosis. ***In sharp opposition to experimental and clinical evidence,*** it has been alleged that proteins, particularly those from animal sources, might be deleterious for bone health by inducing chronic metabolic acidosis which in turn would be responsible for increased calciuria and accelerated mineral dissolution. This claim is based on an hypothesis that artificially assembles various notions, including in vitro observations on the physical-chemical property of apatite crystal, short term human studies on the calciuric response to increased protein intakes, as well as retrospective inter-ethnic comparisons on the prevalence of hip fractures. The main purpose of this review is to analyze the

evidence that refutes a relation of causality between the elements of this putative patho-physiological "cascade" that purports that animal proteins are causally associated with an increased incidence of osteoporotic fractures. In contrast, ***many experimental and clinical published data concur to indicate that low protein intake negatively affects bone health.*** Thus, selective deficiency in dietary proteins causes marked deterioration in bone mass, micro architecture and strength, the hallmark of osteoporosis. In the elderly, low protein intakes are often observed in patients with hip fracture. In these patients intervention study after orthopedic management demonstrates that protein supplementation as given in the form of casein, attenuates post-fracture bone loss, increases muscles strength, reduces medical complications and hospital stay. In agreement with both experimental and clinical intervention studies, ***large prospective epidemiologic observations indicate that relatively high protein intakes, including those from animal sources are associated with increased bone mineral mass and reduced incidence of osteoporotic fractures.*** As to the increased calciuria that can be observed in response to an augmentation in either animal or vegetal proteins it can be explained by a stimulation of the intestinal calcium absorption. Dietary proteins also enhance IGF-1, a factor that exerts positive activity on skeletal development and bone formation. Consequently, ***dietary proteins are as essential as calcium and vitamin D for bone health and osteoporosis prevention***. Furthermore, there is no consistent evidence for superiority of vegetal over animal proteins on calcium metabolism, bone loss prevention and risk reduction of fragility fractures.

BMC Musculoskelet Disord. 2010 May 10;11:88. **Low urine pH and acid excretion do not predict bone fractures or the loss of bone mineral density: a prospective cohort study.**

Fenton TR, Eliasziw M, Tough SC, Lyon AW, Brown JP, Hanley DA.

Abstract

BACKGROUND:

The acid-ash hypothesis, the alkaline diet, and related products are marketed to the general public. Websites, lay literature, and direct mail marketing encourage people to measure their urine pH to assess their health status and their risk of osteoporosis.The objectives of this study were to determine whether 1) low urine pH, or 2) acid excretion in urine [sulfate + chloride + 1.8x phosphate + organic acids] minus [sodium + potassium + 2x calcium + 2x magnesium mEq] in fasting morning urine predict: a) fragility fractures; and b) five-year change of bone mineral density (BMD) in adults.

DESIGN:

Cohort study: the prospective population-based Canadian Multicentre Osteoporosis Study. Multiple logistic regression was used to examine associations between acid excretion (urine pH and urine acid excretion) in fasting morning with the incidence of fractures (6804 person years). Multiple linear regression was used to examine associations between acid excretion with changes in BMD over 5-years at three sites: lumbar spine, femoral neck, and total hip (n = 651). Potential confounders controlled included: age, gender, family history of osteoporosis, physical activity, smoking, calcium intake, vitamin D status, estrogen status, medications, renal function, urine creatinine, body mass index, and change of body mass index.

RESULTS:

There were ***no associations between either urine pH or acid excretion and either the incidence of fractures or change of BMD*** after adjustment for confounders.

CONCLUSION:

Urine pH and urine acid excretion do not predict osteoporosis risk.

Nutr J. 2011 Apr 30;10:41. Causal assessment of dietary acid load and bone disease: a systematic review & meta-analysis applying Hill's epidemiologic criteria for causality. Fenton TR, Tough SC, Lyon AW, Eliasziw M, Hanley DA.

Abstract

BACKGROUND:

Modern diets have been suggested to increase systemic acid load and net acid excretion. In response, alkaline diets and products are marketed to avoid or counteract this acid, help the body regulate its pH to prevent and cure disease. The objective of this systematic review was to evaluate causal relationships between dietary acid load and osteoporosis using Hill's criteria.

METHODS:

Systematic review and meta-analysis. We systematically searched published literature for randomized intervention trials, prospective cohort studies, and meta-analyses of the acid-ash or acid-base diet hypothesis with bone-related outcomes, in which the diet acid load was altered, or an alkaline diet or alkaline salts were provided, to healthy human adults. Cellular mechanism studies were also systematically examined.

RESULTS:

Fifty-five of 238 studies met the inclusion criteria: 22 randomized interventions, 2 meta-analyses, and 11 prospective observational studies of bone health outcomes including: urine calcium excretion, calcium balance or retention, changes of bone mineral density, or fractures, among healthy adults in which acid and/or alkaline intakes were manipulated or observed through foods or supplements; and 19 in vitro cell studies which examined the hypothesized mechanism. Urine calcium excretion rates were consistent with osteoporosis development; however calcium balance studies did not demonstrate loss of whole body calcium with higher net acid excretion. Several weaknesses regarding the acid-ash hypothesis were uncovered: No intervention studies provided direct evidence of osteoporosis progression (fragility fractures, or bone strength as measured using biopsy). The supporting prospective cohort studies were not controlled regarding important osteoporosis risk factors including: weight loss during follow-up, family history of osteoporosis, baseline bone mineral density, and estrogen status. No study revealed a biologic mechanism functioning at physiological pH. Finally, randomized studies did not provide evidence for an adverse role of phosphate, milk, and grain foods in osteoporosis.

CONCLUSIONS:

A causal association between dietary acid load and osteoporotic bone disease is not supported by evidence and there is no evidence that an alkaline diet is protective of bone health.

And on the other side . . . This article argues the opposite of the above. It summarizes all the arguments and evidence for the acid-alkaline theory. Frasetto and colleagues have published a series of articles arguing for this theory. However, they do not make the classical argument that induced dietary acidosis causes damage to the bones throughout the life, only that in elder years, the aging kidney loses its ability to neutralize acid in the blood. My criticism of this article is 1) it does no more than demonstrate that consuming more vegetables is healthy for your bones and then interprets, according to the authors pre-

existing bias, that this must be due to the alkalinity of the vegetables, rather than other factors such as micronutrition, psitive effects of fiber on the microbiome, etc. And 2) despite purporting to be a review article, it ignores virtually all of the evidence cited above. It is a prime example of selective citation of evidence to support one theory while ignoring evidence to the contrary. I include it here because I do not want to do the same thing these authors do.
Here it is:

Br J Nutr. 2010 Apr;103(8):1185-94. Diet-induced acidosis: is it real and clinically relevant?
Pizzorno J, Frassetto LA, Katzinger J.
Abstract
The concept of diet-induced 'acidosis' as a cause of disease has been a subject of interest for more than a century. The present article reviews the history of our evolving understanding of physiological pH, the physiological support for the concept of 'acidosis', the causes of acidosis, how it is recognised, its short-term effects as well as the long-term clinical relevance of preventative measures, and the research support for normalisation of pH. Further, we suggest differentiation of the terms 'acidosis' and 'acidaemia' as a way to resolve the conflation of these topics which has led to confusion and controversy. The available research makes a compelling case that diet-induced acidosis, not diet-induced acidaemia, is a real phenomenon, and has a significant, clinical, long-term pathophysiological effect that should be recognised and potentially counterbalanced by dietary means.