Food composition and acid-base balance: experimental observations on alimentary alkali depletion in herbivores.

H. KIWULL-SCHÖNE[1], P. KIWULL[1], F. MANZ[3], H. KALHOFF[2]
[1]Department of Physiology, Faculty of Medicine, Ruhr-University, 44780 Bochum; [2]Pediatric Clinic, 44137 Dortmund; [3]Research Institute of Child Nutrition, 44225 Dortmund, Germany
email: heidrun.kiwull-schoene@ruhr-uni-bochum.de

Alkali enriched diets are recommended for humans to diminish their normal nutritional net acid load. As typical herbivores, rabbits on the other hand have to deal with the impact of very high dietary alkali intake on control systems involved in acid-base balance. Here it is attempted to explore the role of a high versus low alkali nutritional background for the development of chronic systemic metabolic acidosis. This may be of general interest for respiratory and renal physiology, in which research fields the rabbit serves as a common animal model in vivo and in vitro.

Data were collected from healthy male adult rabbits kept in metabolism cages, to obtain the 24h urine and blood samples from the central ear artery. Different randomized groups of animals were fed rabbit chow ad libitum providing sufficient energy but variable alkali load. One subgroup was fed high-alkali standard food and additionally received NH_4Cl with the drinking water for up to nine consecutive days. Another group was fed either low-alkali food alone for five days or was given additionally NH_4Cl during the last two days on low-alkali diet.

In spite of a wide range of alimentary acid-base load, normal acid-base conditions were maintained in the arterial blood [1]. In contrast, the dietary alkali load was significantly reflected by renal acid-base excretion. On high-alkali chow, an alkaline urine (pHu >8.0) was excreted, typically containing a large amount of precipitated carbonate. The average fractional renal base re-absorption was thus incomplete, in line with negative net acid excretion (NAE). On low-alkali diet, the mean pHu decreased along with a strong reduction of total base excretion. Thereby, the fractional base re-absorption was nearly complete, and NAE rose from negative values to zero level. During high-alkali feeding, a relatively large amount of added NH_4Cl changed NAE to the zero level as well, but systemic BE was still maintained. During low-alkali feeding, a comparably small amount of added NH_4Cl exhausted renal base re-absorption completely and gave rise to NAE above zero. This was followed by a manifest systemic metabolic acidosis, indicated by distinct reduction of base excess.

It is obvious that dietary acid-base variations are more accurately reflected in the urine than in the blood. Moreover, the urinary tract of the herbivore rabbit appears to be kept without damage despite large amounts of precipitate under nutritional conditions, which would bear growing danger of nephrolithiasis in carnivore species (e.g. cats occasionally fed too alkaline food or vegetarian humans confined to bed or meeting space-lab conditions). In the herbivore rabbit, metabolic acidosis can neither be achieved by NH_4Cl against the background of normal high alkali food, nor by feeding low alkali diet alone. However, challenging renal base saving and/or net acid excretion by low alkali diet is prerequisite for growing susceptibility to NH_4Cl-induced chronic metabolic acidosis in this species.

References:

[1] Kiwull-Schöne H, Kalhoff H, Manz F, Kiwull P. Food mineral composition and acid-base balance in rabbits. Eur. J. Nutr. 44, 499-508, 2005.