Food composition and acid-base balance: alimentary acid load and clinical implications in neonates.

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In preterm neonates, functional limits of e.g. pulmonary and/or renal regulation processes and the considerable acid load of common formulas predispose to a great risk for the development of latent metabolic acidosis, characterized by e.g. impaired mineralization and reduced growth. To demonstrate the implications of latent acidosis in neonates, we present first results of a prospective clinical study on the development of nephrocalcinosis, second an analysis of acid-base regulation in preterm neonates under different diets, and third an algorithm to estimate the renal net acid excretion of formulas for preterm infants.

In a prospective study, very low birth weight (VLBW)- infants developing nephrocalcinosis frequently showed a tendency towards metabolic acidosis (systemic pH<7.25) on day 2-7, followed by low serum levels of phosphorus (P) and high renal calcium (Ca) and P excretion within 2 weeks. Thus, impaired acid-base homeostasis with metabolic acidosis may be a further risk factor predisposing to the development of nephrocalcinosis in preterm infants.
Moreover, to obtain fundamental data of acid-base regulation in preterm infants under different diets, we investigated 48 preterm infants fed their own mother`s milk (28 native human milk, 20 enriched with fortifier) and 34 patients on formula (23 on a standard batch, 11 on a modified batch with reduced acid load). We found no notable differences between individual data of acid-base status in blood samples, irrespective of the diet. In contrast, dietary acid-base intake was accurately reflected in the urine, pointing to effective individual compensation of alimentary acid-load. Interestingly, net acid excretion (NAE) in preterm infants on human milk fortified with protein and electrolytes was only slightly higher than on native human milk in the presence of a higher urine-pH, but low when compared to standard formula.
Based on own studies and on literature data a physiologically based and empirically adjusted calculation model is presented to estimate the impact of mineral and protein content of a formula on the urinary ionogram and thus on the average renal NAE in a regularly fed and growing preterm infant.

Preterm infants fed formulas are at a considerable risk of spontaneously developing latent, and occasionally manifest, late metabolic acidosis. Renal mechanisms are predominant and effective in compensating for minor differences in alimentary acid-base intake. Thus, in preterm infants, nutritional acid-base challenges can be judged earlier and more safely by urinary than by blood acid-base analysis. The algorithm of the proposed calculation model could prove to be a useful tool in the design of new formulas with adaequate base supply for preterm infants.