Effect of Dietary Phosphorus on the Expression of NaPi-IIb, MEPE and Apoptosis Intestinal in Uremic Rats

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Introduction: Hyperphosphatemia is a frequent and serious complication in patients with Chronic Kidney Disease (CKD). Cotransporters are involved in intestinal absorption of phosphorus (Pi). Faced with declining renal function, the small intestine may be an important therapeutic target for the control of Pi. **Objective:** To investigate the effect of diets with different concentrations of Pi in the regulation of MEPE (matrix extracellular phosphoglycoprotein), apoptosis rate and of cotransporter NaPi-IIb in enterocytes of uremic rats. **Methods:** Male Wistar rats, were divided into two groups: nephrectomized rats (Nx=39) and normal renal function control rats (C=37) were maintained on standard diet for 30 days. After this period changed diets varying concentrations of Pi: 0.2 (low), 0.54 (standard) and 0.9% (high) for 48 hours. Blood from the aorta was collected for biochemical analysis and intestinal tissue of duodenum, jejunum, and ileum were obtained to evaluate protein and gene expression. **Results:** The Nx animals evolved with increasing concentrations of creatinine, Pi, FGF-23, parathyroid hormone (PTH) serum and urinary albumin. Dietary intake was not different between groups. There was no difference in serum P in C groups, demonstrating the ability of these animals to maintain homeostasis with a growing increase in fractional excretion of Pi. The administration of Pi high diet for 48 hours was sufficient to triple the levels of FGF-23 and duplicate PTH levels in Nx animals compared to C on a standard diet. The expression of mRNA for NaPi-IIb was lower in the Nx animals in Pi low diet compared to their respective control. The MEPE expression and the apoptosis rate were higher in the Nx rats with overload phosphorus. **Conclusions:** The concentration of Pi in the diet and the delivery time may interfere with the expression of NaPi-IIb, especially in Nx animals. The MEPE protein might act as first barrier transcellular in phosphorus absorption.

Keywords: Renal failure, Phosphorus, enterocytes

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