DIETARY INTAKE OF HIGH CALORIES AND HIGH PHOSPHATE PROMOTES RENAL LESIONS IN RATS

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INTRODUCTION AND AIMS

The relationship of obesity and metabolic syndrome (OB/MS) with renal disease can be explained by a variety of mechanisms, including hypertension and type II diabetes. One of the major factors related to the development of OB/MS is the intake of energy-rich foods which, in addition to promote obesity, may have other deleterious effects on renal function. Moreover, in the context of renal disease, it is important to note that many energy-dense foods tend to have a high phosphate content due to the presence of phosphate-containing additives. In this work, we evaluated the effect on kidney histology of feeding diets with high caloric and/or high phosphate content to healthy rats.

METHODS

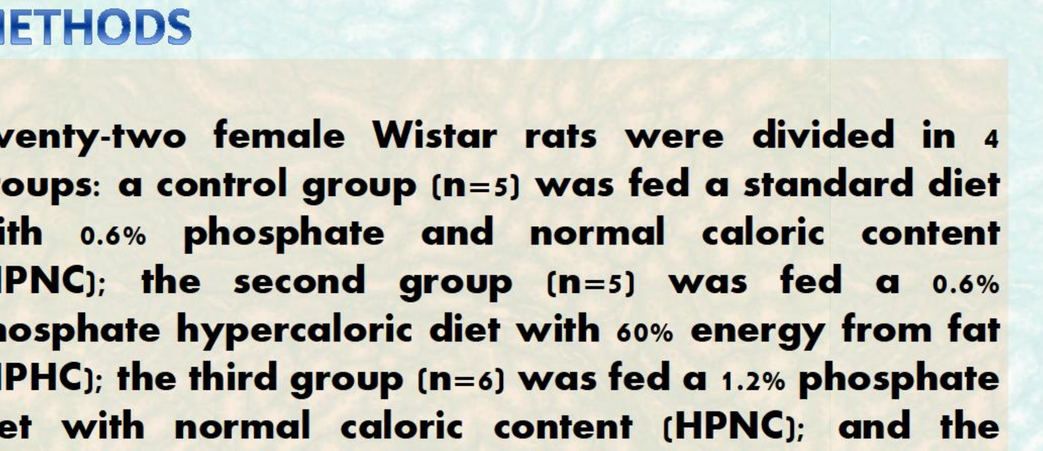
Twenty-two female Wistar rats were divided in 4 groups: a control group (n=5) was fed a standard diet with 0.6% phosphate and normal caloric content (NPNC); the second group (n=5) was fed a 0.6% phosphate hypercaloric diet with 60% energy from fat (NPHC); the third group (n=6) was fed a 1.2% phosphate diet with normal caloric content (HPNC); and the fourth group (n=6) was fed a 1.2% phosphate hypercaloric diet (HPHC). All diets were fed for 30 days and then rats were sacrificed to obtain renal tissue. Tissue samples were fixed in 10% buffered formalin, embedded in paraffin, sectioned and processed for staining with hematoxylin and eosin, periodic acid-Schiff, Masson's Trichrome and Von Kossa stains. Lesions were scored using a semi quantitative scale graded from 0-3: 0 (absent), 1 (slight), 2 (moderate) or 3 (severe). Analyses were performed in a blind manner.

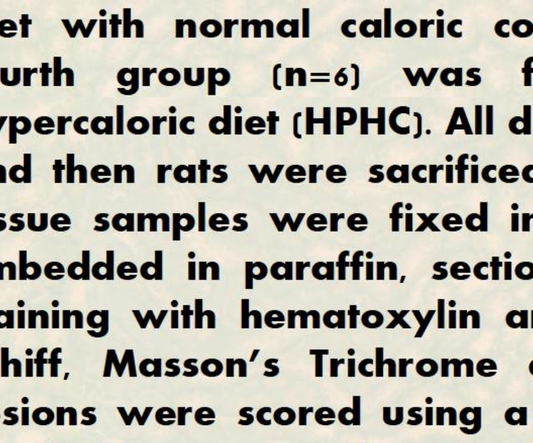
RESULTS

No lesions were observed in control animals (Figure 1). Minor lesions were observed after feeding both NPHC and HPNC diets. All rats fed HPHC diet showed moderate to severe (grades 2-3) tubular hyperplasia, tubular dilation, interstitial infiltrate and nephrocalcinosis (Table 1). Tubular hyperplasia with focal crowding and increased nuclear to cytoplasmatic ratio was accompanied by a loss of brush border in proximal tubules and marked thickening of the tubule basement membrane. Dilation of tubular lumens, which were lined by a flattened epithelium, was also noted. Interstitial infiltrate was composed by mononuclear cells which were surrounding hyperplastic tubules (Figures 2-4). Calcifications were mainly located in the juxtamedullary cortex, with some scattered small areas in outer and inner medulla (Figure 5-6).

	Tubular hyperplasia	Tubular dilatation	Interstitial infiltrate	Nephrocalcinosis
0.6% Phosphate Normocaloric	0.00±0.00 [a]	0.00±0.00 [a]	0.00±0.00 [a]	0.00±0.00 [a]
0.6% Phosphate Hypercaloric	0.60±0.24 [a]	0.40±0.24 [a]	0.40±0.40 [a]	0.40±0.24 [a]
1.2% Phosphate Normocaloric	0.33±0.21[a]	0.17±0.17 [a]	0.50±0.34 [a]	0.33±0.21 [a]
1.2% Phosphate Hypercaloric	2.50±0.34	2.00±0.45	2.00±0.26	2.67±0.21

Table 1. Values are mean±SE; [a] P<0.05 vs HPHC





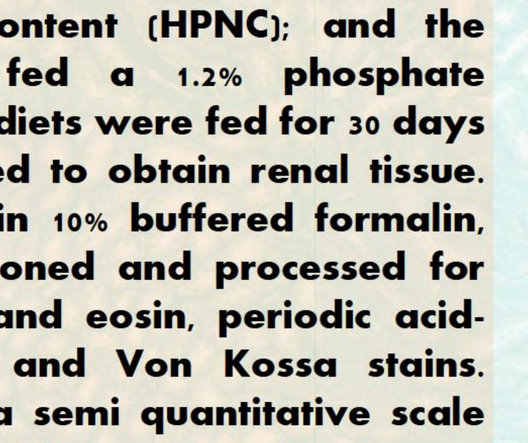
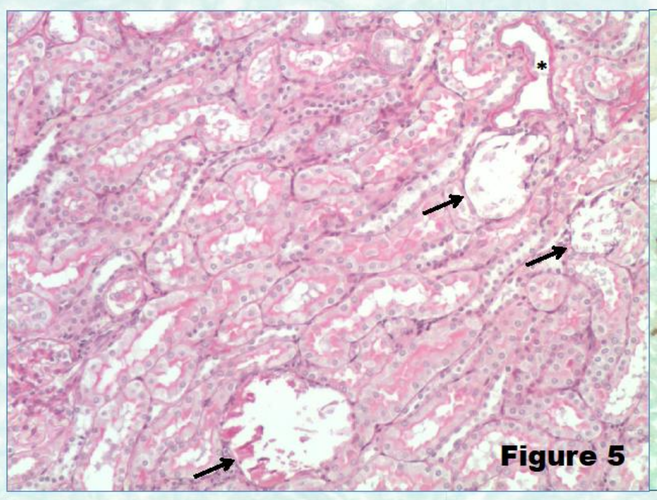


Figure 1. PAS stain.. No lesions were detected in NPNH group. Figure 2. PAS stain. HPHC group. Hyperplastic tubules with thickening of the basal membrane, crowding of epithelial cells, increase in nuclear to cytoplasmic ratio and loss of the brush border. An interstitial infiltrate composed of mononuclear cells was detected surrounding hyperplastic tubules. Figure 3. PAS stain. HPHC group. Hyperplastic tubules surrounded by interstitial infiltrate and tubular dilatation (asterisk). Figure 4. H&E stain. HPHC group. In some rats, mononuclear cells were detected also in a perivascular location.



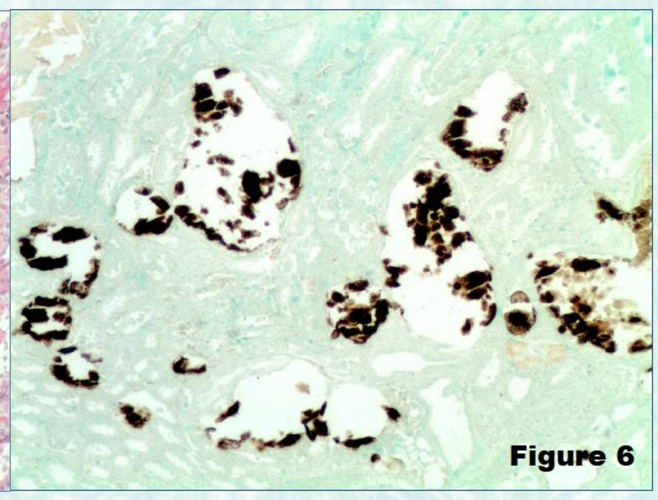


Figure 5. H&E stain. HCHP group. Foci of nephrocalcinosis (arrow) and tubular dilatation (asterisk). Figure 6. HCHP group. Staining of foci of nephrocalcinosis with Von Kossa.

CONCLUSIONS

These data show that feeding high-phosphate or energy-rich diets has the potential of inducing minor lesions in the kidneys of rats, but when both factors (high phosphate and high calories) are combined the deleterious effect is potentiated resulting in significant renal damage.

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