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## **Calcium, Phosphorus and Vitamin D Disorders in Uremia**

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### **Abstract**

**Background:** Alterations in calcium, phosphate (P) and vitamin D metabolism play a critical role in the development of secondary hyperparathyroidism (SH), parathyroid hyperplasia and soft tissue and vascular calcification. **Methodology:** Studies were performed in uremic dogs and rats fed a low and high P diet over a period of 1–4 months. In addition, in vitro studies were performed in normal parathyroid glands incubated in culture media containing 0.2 mM P (low) or 2.0 mM P (high). **Results:** Uremic rats maintained on a low P diet did not develop SH or parathyroid hyperplasia. There was an enhancement of p21, the suppressor of the cell cycle, in these parathyroid glands. Opposite results were obtained using a high P diet. There was an enhancement of transforming growth factor- $\alpha$  and epidermal growth factor receptor, known enhancers of cell proliferation. In vitro studies demonstrated the direct effect of P on parathyroid hormone secretion. **Conclusions:** Early dietary P restriction prevents the development of SH and parathyroid hyperplasia. If dietary P restriction is applied to rats with established SH, there is a significant amelioration of SH and parathyroid hyperplasia. In addition, control of serum P in uremic patients is crucial in the prevention of vascular calcification.

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### **Introduction**

Secondary hyperparathyroidism (SH) and hyperplasia of the parathyroid glands are universal complications in patients with chronic kidney disease (CKD). Abnormal calcium, phosphorus and vitamin D metabolism in chronic renal failure play a key role in the development of SH and renal osteodystrophy. In addition, hyperphosphatemia is a major factor in the pathogenesis of vascular calcification.