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Advances in therapeutics related to phosphate disorders in adults and children

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Abstract

Twenty six million Americans suffer from chronic kidney disease and the mortality rate is thirty-fold higher than in the general population. Disordered phosphorus homeostasis is a significant contributor to the morbidity and mortality of patients with chronic kidney disease. Plasma phosphorus concentration and phosphorus homeostasis are maintained by a complex interaction of hormones produced by the bone, kidney and parathyroid gland. Fibroblast growth factor 23 (FGF23), 1,25 dihydroxyvitamin D ($1,25(OH)_2D$) and parathyroid hormone (PTH) maintain normal serum phosphorus concentrations principally by regulation of intestinal and renal absorption of phosphate and by regulation of bone mineralization and turnover. Aberrant regulation of these processes occurs in acquired and inherited disorders of phosphate homeostasis resulting in adverse outcomes in affected patients. Studies of genetic and acquired diseases in humans and animal models of hypo- and hyper-phosphatemia have led to the discovery of several important molecular mechanisms by which phosphate homeostasis is maintained by the concerted interactions of circulating $1,25(OH)_2D$, FGF23 and PTH. Much has been learnt about the mechanisms of renal phosphate reabsorption and its impact on skeletal and cardiovascular health. Consequently, advances in medical therapy for disorders of phosphorus homeostasis are expected to improve the quality of life and decrease morbidity and mortality of affected patients.

Biography

Farzana Perwad received her M.D from Kasturba Medical College, India and did her Pediatrics residency training at Miami Children's Hospital, Florida. She completed her Pediatric Nephrology fellowship and is an Assistant Professor in the Department of Pediatrics, at the University of California San Francisco. Dr. Perwad's research focuses on the regulation of vitamin D and phosphorus homeostasis in health and disease. Her research projects include investigating the pathophysiology of X-Linked Hypophosphatemia in mouse models of the human disease, and to study the molecular mechanisms of action of fibroblast growth factor-23 in the kidney.