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ROLE OF FGF23 IN THE PATHOGENESIS OF MINERAL AND BONE DISORDERS IN CHRONIC KIDNEY DISEASE

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ROLE OF FGF23 IN THE PATHOGENESIS OF MINERAL AND BONE DISORDERS IN CHRONIC KIDNEY DISEASE

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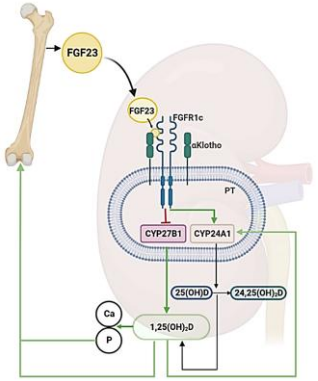
Research Relevance: Mineral and bone disorders in chronic kidney disease (CKD-MBD) are among the most common and severe complications in patients with renal insufficiency. Fibroblast Growth Factor 23 (FGF23) is an osteocytic hormone that regulates phosphate metabolism and vitamin D homeostasis. Its levels begin to rise in the early stages of CKD and have systemic effects on mineral balance. Excessive FGF23 production disrupts bone mineralization, exacerbates hypophosphatemia, reduces the synthesis of active vitamin D, and is associated with an increased risk of cardiovascular complications. Studying the role of FGF23 in the pathogenesis of CKD-MBD is of high clinical importance for diagnosis, monitoring, and therapeutic strategy selection

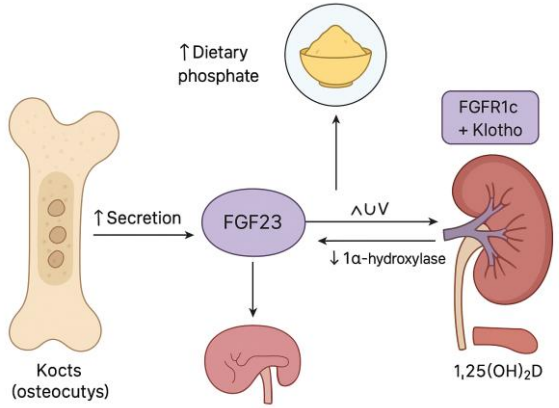
Research Goal: To assess the significance of FGF23 in the development of mineral and bone disorders in CKD, establish its clinical and laboratory correlations, and identify potential directions for targeted therapy.

Materials and Methods: Analysis of scientific publications from the past 10 years, including clinical studies, meta-analyses, and reviews on FGF23 and CKD. Evaluation of phosphate-calcium metabolism indicators, levels of $1,25(OH)_2D$, parathyroid hormone (PTH), and FGF23 in patients at various stages of CKD.



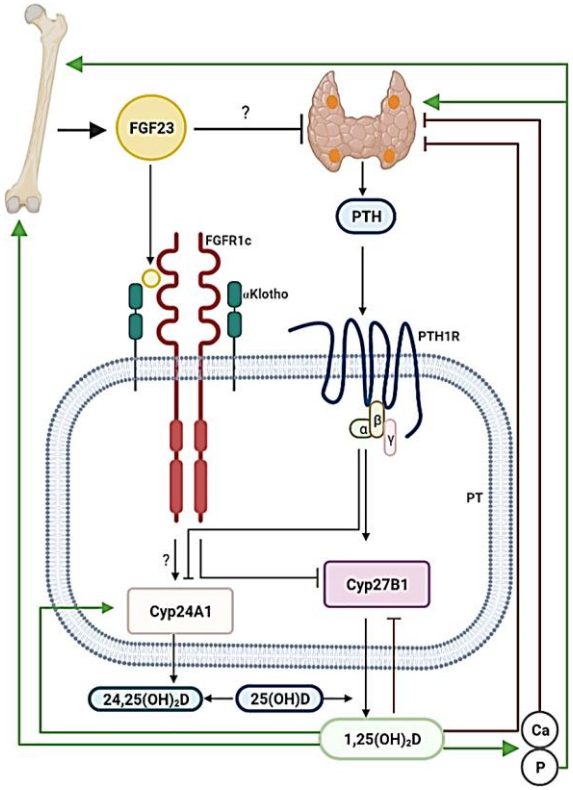
Results: The analysis revealed that FGF23 levels increase as early as CKD stages 2–3, well before changes in serum phosphorus and PTH levels. This elevation is linked to several key mechanisms.

In CKD, the kidneys lose the ability to efficiently excrete phosphates, leading to their accumulation in the blood. In response, bone tissue produces more FGF23 to stimulate phosphate excretion by the kidneys. FGF23 also suppresses the activity of 1-alpha-hydroxylase in the kidneys, reducing the production of active vitamin D ($1,25(OH)_2D$). This impairs the absorption of calcium and phosphates in the intestines, worsening hypophosphatemia and hypocalcemia. In response to decreased blood calcium levels, the body increases PTH production, which promotes phosphate excretion and disrupts phosphate metabolism, further stimulating FGF23 production. FGF23 inhibits sodium-phosphate cotransporters in the proximal renal tubules, enhancing phosphate excretion and lowering blood phosphate levels. These changes lead to impaired bone mineralization, manifesting as osteopenia, osteomalacia, and an increased risk of fractures. Elevated FGF23 levels are also associated with cardiotoxic effects, particularly left ventricular hypertrophy and increased mortality in CKD patients. In dialysis patients, higher FGF23 levels correlate with the severity of secondary hyperparathyroidism and the need for aggressive therapy. FGF23 is considered not only a biomarker but also a therapeutic target: approaches aimed at reducing its production or blocking its receptors (e.g., anti-FGF23 antibodies) are promising, especially in severe phosphate metabolism disorders.



Conclusion: FGF23 plays a key role in the pathogenesis of mineral and bone disorders in CKD, initiating a cascade of interconnected changes—hypophosphatemia, active vitamin D deficiency, secondary hyperparathyroidism, and impaired bone mineralization.

Elevated FGF23 levels serve as an early biomarker of CKD-MBD and contribute to the progression of cardiovascular and skeletal complications. Assessing and controlling FGF23 may be crucial steps in the individualized management of patients with chronic kidney disease.



Role of FGF23 in vitamin D and phosphate metabolism: implications in chronic kidney disease https://pubmed.ncbi.nlm.nih.gov/22421513/
Regulation of FGF23 production and phosphate metabolism by bone-kidney interactions https://pubmed.ncbi.nlm.nih.gov/36624273/
FGF23: a key player in mineral and bone disorder in CKD. https://scielo.isciii.es/scielo.php?script=sci_arttext&pid=S0211-69952009000500003

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