

Technical Information

FGF-23 Intact, Human (Kainos)

Fibroblast Growth Factor 23

Cat. No.:	CY-4000
Tests:	96
Method:	ELISA
Range:	8 – 800 pg/ml
Sensitivity:	3.0 pg/ml
Incubation time:	3.5 hours
Sample volume:	50 µl
Sample type:	Serum / Heparin- and EDTA Plasma
Sample preparation:	It is recommended to collect the sample in the morning after a 12-hour fasting period. Intact FGF-23 is very instable. Therefore, collection and testing or storage should take place promptly. Store samples at -20 °C or below. Avoid repeated freezing and thawing of specimens.

Reference values: 10 – 50 pg/ml

Species: Human, Rat, Mouse

Intended use:

FGF-23 Fibroblast Growth Factor 23

FGF-23 is produced in osteoblast precursor cells and is a potent regulator of phosphate and vitamin D metabolism.

Phosphate plays an essential role in the stability of skeletal bones and energy metabolism as well as in DNA synthesis and intracellular signal cascades.

FGF-23 inhibits in combination with cofactor Klotho phosphate reabsorption in renal proximal tubular cells via FGF-23 receptors (increased phosphate loss, reduced serum phosphate) and decreases calcitriol synthesis by suppressing alpha-1-hydroxylase.

FGF-23 in Osteology

FGF-23 is involved in a variety of diseases accompanied by hypophosphatemia caused by renal phosphate loss. Moreover, the clinical pictures show distinctly reduced calcitriol synthesis and osteomalacia or vitamin D resistant rickets.

1. Tumor-induced osteomalacia / hypophosphatemia (TIO; paraneoplastic overexpression of FGF-23)
2. Autosomal dominant hypophosphatemic rickets (ADHR; due to mutation in FGF-23 protein, FGF-23 cannot be inactivated by endopeptidases)
3. X-linked hypophosphatemia (XHL, mutation in degrading enzyme (PHEX))
4. Craniofacial dysplasia with hypophosphatemia (increased FGF-23 levels caused by mutation of FGF receptor 1)
5. Fibrous dysplasia of bone (overproduction of FGF-23 due to mutation in G-protein subunit G5a/GNAS1)

FGF-23 in Nephrology

1. Elevated FGF-23 values are seen in chronic renal insufficiency and correlate negatively with GFR.
2. Increased serum FGF-23 levels may help maintain normophosphatemia in early chronic renal insufficiency until creatinine clearance is reduced to approximately 30 mL/min and hyperphosphatemia develops due to exhausted regulatory mechanisms and concurrently decreased calcitriol and sHPT.
3. Monitoring of FGF-23 and serum phosphate in early chronic renal insufficiency allows, if necessary, to institute phosphate reduction therapy at an earlier stage.
4. Creatinine levels within the normal range do not exclude disorders of phosphate metabolism.
5. In the ArMoRR study published by Guitierrez et al. in August 2008, it was demonstrated that the FGF-23 level at the beginning of hemodialysis therapy may be seen as an independent risk marker. Patients showing FGF-23 levels within the highest range developed a 5.7fold higher risk of death within one year.

References

Guitierrez et al.: Fibroblast Growth Factor 23 and Mortality among Patients Undergoing Hemodialysis. N Eng J Med 2008; 359: 584-92

Chi-yuan Hsu: FGF-23 and Outcomes Research – When Physiology meets Epidemiology. N Engl J Med 2008; 359 6

Andreas L. Serra et al.: Phosphatemic Effect of Cinacalcet in Kidney Transplant Recipients With Persistent Hyperparathyroidism. American Journal of Kidney Diseases 2008

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