

FGF23 Human

Fibroblast Growth Factor-23 Human Recombinant

GRF0100

Product Overview

Name	FGF23 Human
Accession(Primary)	Q9GZV9
Description	Fibroblast Growth Factor-23 Human Recombinant

Target information (Q9GZV9)

Gene Name

About Gene Symbol

Background

Before it was discovered in 2000, there was a hypothesis that a similar type of protein existed that performed many of the functions we see in FGF23. This was originally referred to as phosphatonin. Various effects were described and noted by researchers including inhibition of production and inhibition of secretion of parathyroid hormone. Derived from the bone Fibroblast Growth Factor 23 is a phosphaturic hormone. It increases phosphate excretion when acting on the kidney and also suppresses the biosynthesis of 1,25(OH)₂D₃. Mechanism Despite various research studies into the topic, many of the mechanisms for the regulations of FGF23 production remain a mystery to the scientific community. While we know that mutations in PHEX, ENPP1 and DMP1 result in the increased expression of FGF23, it is unclear why this occurs. This also means that currently, it is not possible to regulate the production of FGF23 either. We also do not know how signals from FGF23 regulate vitamin D metabolism. However, understanding these types of mechanisms could offer information needed to provide better treatment for deranged bone and mineral metabolism. Interactions Molecular interactions involving FGF-23, vitamin D and klotho do provide the solution needed to regulate phosphate levels within the body. Furthermore, an interaction between Vitamin D and FGF3 can have an impact on renal phosphate

balance. As well as this, when in the presence of klotho, FGF3 does actually increase bioactivity and begins to change systemic phosphate homeostasis. Function Based on research it seems that the main function for FGF23 is the regulation of phosphate concentration in plasma. It seems to be secreted from the osteocytes due to elevated levels. When acting upon the kidneys, the hormone reduces the expression of NPT2. This is a sodium-phosphate cotransporter found in the proximal tube. As such, it appears as though FGF23 is able to reduce the reabsorption, all the while maxing the excretion of phosphate. It has also been suggested that the hormone is able to suppress 1-alpha-hydroxylase. If this is the case, it can limit its potential to activate vitamin D and thus impair the ability for calcium absorption. Structure FGF23 is located on the chromosome 12. It is composed of three exons. The crystal structure of FGF23 is completely different from the common conformation typically adopted by paracrine-acting FGFs. Instead, there is a conformation of the HPR region between beta strands 10 and 12. As well as this, there is a cleft between the other HPR-binding region, the beta1-beta12 loop and this one. This comes before a direct interaction between HPR sulfate and FGF23s backbone atoms. Due to this, endocrine function is benefitted and HPR-binding affinity is reduced for the lipangs. Certain mutations that cause the protein to be completely resistant to proteolytic cleavage does trigger a surge in activity of the protein and the renal phosphate loss typically found in certain human diseases including hypophosphatemic rickets. Studies have also revealed that FGF23 is overproduced in certain tumors including phosphaturic mesenchymal tumors. Furthermore a reduced level of activity for this protein is believed to lead to higher phosphate levels and familial tumor calcinosis clinical syndrome.

References for protein:

Title: Leptin stimulates fibroblast growth factor 23 expression in bone and suppresses renal 1 α ,25-dihydroxyvitamin D3 synthesis in leptin-deficient ob/ob Mice Publication: Journal of bone and mineral research 25.8 (2010): 1711-1723. Link: FGF23 prospec publication