

FGF23 Recombinant Rabbit mAb

Catalog # AP98700

Product Information

Application	WB, IHC-P, IHC-F, IF, E
Primary Accession	Q9GZV9
Other Accession	Q9GZV9
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Recombinant
Calculated MW	27954
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the heparin-binding growth factors family.
SUBUNIT	Interacts with FGFR1, FGFR2, FGFR3 and FGFR4. Affinity between fibroblast growth factors (FGFs) and their receptors is increased by KL and heparan sulfate glycosaminoglycans that function as coreceptors.
Post-translational modifications	Following secretion this protein is inactivated by cleavage into a N-terminal fragment and a C-terminal fragment. The processing is effected by proprotein convertases. O-glycosylated by GALT3. Glycosylation is necessary for secretion; it blocks processing by proprotein convertases when the O-glycan is alpha 2,6-sialylated. Competition between proprotein convertase cleavage and block of cleavage by O-glycosylation determines the level of secreted active FGF23.
DISEASE	Defects in FGF23 are the cause of autosomal dominant hypophosphataemic rickets (ADHR) [MIM:193100]. ADHR is characterized by low serum phosphorus concentrations, rickets, osteomalacia, leg deformities, short stature, bone pain and dental abscesses. Defects in FGF23 are a cause of hyperphosphatemic familial tumoral calcinosis (HFTC) [MIM:211900]. HFTC is a severe autosomal recessive metabolic disorder that manifests with hyperphosphatemia and massive calcium deposits in the skin and subcutaneous tissues.
Background Descriptions	This gene encodes a member of the fibroblast growth factor family of proteins, which possess broad mitogenic and cell survival activities and are involved in a variety of biological processes. The product of this gene regulates phosphate homeostasis and transport in the kidney. The full-length, functional protein may be deactivated via cleavage into N-terminal and C-terminal chains. Mutation of this cleavage site causes autosomal dominant hypophosphatemic rickets (ADHR). Mutations in this gene are also associated with hyperphosphatemic familial tumoral calcinosis (HFTC). [provided by RefSeq, Feb 2013],

Additional Information

Gene ID 8074

Other Names	ADHR; FGFR; HPTC2; HPDR2; HYPF; PHPTC; FGF23_HUMAN; FGF23; FGF-23; Phosphatonin; Tumor-derived hypophosphatemia-inducing factor; fibroblast growth factor 23
Dilution	IHC-P=1:50-200,IHC-F=1:50-200,WB=1:500-2000,IF=1:50-200,ELISA=1:5000-10000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	FGF23
Synonyms	HYPF
Function	Regulator of phosphate homeostasis (PubMed: 11062477). Inhibits renal tubular phosphate transport by reducing SLC34A1 levels (PubMed: 11409890). Up-regulates EGR1 expression in the presence of KL (By similarity). Acts directly on the parathyroid to decrease PTH secretion (By similarity). Regulator of vitamin-D metabolism (PubMed: 15040831). Negatively regulates osteoblast differentiation and matrix mineralization (PubMed: 18282132).
Cellular Location	Secreted. Note=Secretion is dependent on O-glycosylation
Tissue Location	Expressed in osteogenic cells particularly during phases of active bone remodeling. In adult trabecular bone, expressed in osteocytes and flattened bone-lining cells (inactive osteoblasts)

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