SANTA CRUZ BIOTECHNOLOGY, INC.

FGF-23 (N-20): sc-16849

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Fibroblast growth factor-1 (FGF-1), also designated acidic FGF, and fibroblast growth factor-2 (FGF-2), also designated basic FGF, are members of a family of growth factors that stimulate proliferation of cells of mesenchymal, epithelial and neuroectodermal origin. Additional members of the FGF family include the oncogenes FGF-3 (Int2) and FGF-4 (hst/Kaposi), FGF-5, FGF-6, FGF-7 (KGF), FGF-8 (AIGF), FGF-9 (GAF) and FGF-10 through FGF-23. Members of the FGF family share 30-55% amino acid sequence identity and similar gene structure, and are capable of transforming cultured cells when overexpressed in transfected cells. Cellular receptors for FGFs are members of a second multigene family, including four tyrosine kinases designated Flg (FGFR-1), Bek (FGFR-L), TKF and FGFR-3.

REFERENCES

1. Yamashita, T., et al. 2000. Identification of a novel fibroblast growth factor, FGF-23, preferentially expressed in the ventrolateral thalamic nucleus of the brain. Biochem. Biophys. Res. Commun. 277: 494-498.
2. White, K.E., et al. 2001. The autosomal dominant hypophosphatemic rickets (ADHR) gene is a secreted polypeptide overexpressed by tumors that cause phosphate wasting. J. Clin. Endocrinol. Metab. 86: 497-500.
3. Bowe, A.E., et al. 2001. FGF-23 inhibits renal tubular phosphate transport and is a PHEX substrate. Biochem. Biophys. Res. Commun. 284: 977-981.
4. Yamashita, T., et al. 2002. Fibroblast growth factor (FGF)-23 inhibits renal phosphate reabsorption by activation of the mitogen-activated protein kinase pathway. J. Biol. Chem. 277: 28265-28270.
5. Riminucci, M., et al. 2003. FGF-23 in fibrous dysplasia of bone and its relationship to renal phosphate wasting. J. Clin. Invest. 112: 683-692.

CHROMOSOMAL LOCATION

Genetic locus: FGF23 (human) mapping to 12p13.32; Fgf23 (mouse) mapping to 6 F3.

SOURCE

FGF-23 (N-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping within an internal region of FGF-23 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-16849 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

FGF-23 (N-20) is recommended for detection of precursor and mature FGF-23 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

FGF-23 (N-20) is also recommended for detection of precursor and mature FGF-23 in additional species, including equine, canine and porcine.

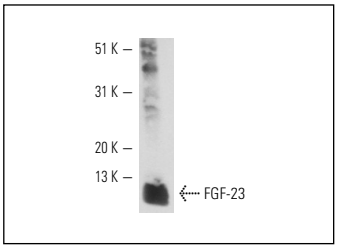
Suitable for use as control antibody for FGF-23 siRNA (h): sc-39486, FGF-23 siRNA (m): sc-39487, FGF-23 shRNA Plasmid (h): sc-39486-SH, FGF-23 shRNA Plasmid (m): sc-39487-SH, FGF-23 shRNA (h) Lentiviral Particles: sc-39486-V and FGF-23 shRNA (m) Lentiviral Particles: sc-39487-V.

Molecular Weight of mature FGF-23: 32 kDa.

Molecular Weight of FGF-23 fragment(s): 12 kDa.

Positive Controls: mouse heart extract: sc-2254.

DATA



FGF-23 (N-20): sc-16849. Western blot analysis of FGF-23 expression in mouse heart tissue extract.

SELECT PRODUCT CITATIONS

1. Koriyama, N., et al. 2006. Oncogenic osteomalacia in a case with a maxillary sinus mesenchymal tumor. Am. J. Med. Sci. 332: 142-147.
2. Martin, A., et al. 2008. Degradation of MEPE, DMP1, and release of SIBLING ASARM-peptides (minhibins): ASARM-peptide(s) are directly responsible for defective mineralization in HYP. Endocrinology 149: 1757-1772.
3. Blomberg Jensen, M., et al. 2012. Vitamin D metabolism and effects on pluripotency genes and cell differentiation in testicular germ cell tumors *in vitro* and *in vivo*. Neoplasia 14: 952-963.