

## FGF 23 C-term Human

**Description:** FGF-23 C-term Protein is 8.67 kDa protein containing 72 amino acid residues and an additional 9 a.a. His-Tag at N-terminus.

**Catalog #:** CYP5-035

**Synonyms:** Tumor-derived hypophosphatemia-inducing factor, HYPF, ADHR, HPDR2, PHPTC, FGF23, FGF-23, Fibroblast Growth Factor-23.

For research use only.

**Source:** E. coli

**Amino Acid Sequence:** MKHHHHHHAS AED&shy;DSERDPL NVLKPRARMT PAPASCSQEL  
PSAEDNSPMA SDPLGVVRGG RVNTHAGGTG PEGCRPFAKF I.

**Purity:** Greater than 90.0% as determined by densitometric image analysis.

**Formulation:**

FGF-23 C-term was filtered (0.4

**Stability:**

Store lyophilized FGF 23 C-term at -20°C. Aliquot the product after reconstitution to avoid repeated freezing/thawing cycles. Reconstituted FGF 23 C-term can be stored at 4°C for a limited period of time; it does not show any change after two weeks at 4°C.

**Usage:**

NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drugs, agricultural or pesticidal products, food additives or household chemicals.

**Applications:**

Western blotting

**Solubility:**

It is recommended to add deionized water to prepare a working stock solution of approximately 0.5mg/ml and let the lyophilized pellet dissolve completely. Product is not sterile! Please filter the product by an appropriate sterile filter before using it on cell culture.

**Introduction:**

FGF-23 is a member of the fibroblast growth factor (FGF) family. FGF family members possess broad mitogenic and cell survival activities and are involved in a variety of biological processes including embryonic development, cell growth, morphogenesis, tissue repair, tumor growth and invasion. FGF-23 inhibits renal tubular phosphate transport. This gene was identified by its mutations associated with autosomal dominant hypophosphatemic rickets (ADHR), an inherited phosphate wasting disorder. Abnormally high level expression of FGF23 was found in oncogenic hypophosphatemic osteomalacia (OHO), a phenotypically similar disease caused by abnormal phosphate metabolism. Mutations FGF23 have also been shown to cause familial tumoral calcinosis with hyperphosphatemia.

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