10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

FGFR4 Blocking Peptide (N-term)

Synthetic peptide
Catalog # BP20781a

Specification

FGFR4 Blocking Peptide (N-term) - Product Information

Primary Accession	P22455
Other Accession	Q03142

FGFR4 Blocking Peptide (N-term) - Additional Information

Gene ID 2264

Other Names
Fibroblast growth factor receptor 4, FGFR-4, CD334, FGFR4, JTK2, TKF

Target/Specificity
The synthetic peptide sequence is selected from aa 143-158 of HUMAN FGFR4

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

FGFR4 Blocking Peptide (N-term) - Protein Information

Name FGFR4

Synonyms JTK2, TKF

Function
Tyrosine-protein kinase that acts as a cell-surface receptor for fibroblast growth factors and plays a role in the regulation of cell proliferation, differentiation and migration, and in regulation of lipid metabolism, bile acid biosynthesis, glucose uptake, vitamin D metabolism and phosphate homeostasis. Required for normal down- regulation of the expression of CYP7A1, the rate-limiting enzyme in bile acid synthesis, in response to FGF19. Phosphorylates PLCG1 and FRS2. Ligand binding leads to the activation of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate. Phosphorylation of FRS2 triggers recruitment of GRB2, GAB1, PIK3R1 and SOS1, and mediates activation of RAS, MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. Promotes SRC-dependent phosphorylation of the matrix protease MMP14 and its lysosomal degradation. FGFR4 signaling is down-regulated by receptor internalization and degradation; MMP14 promotes internalization and degradation of FGFR4.

Mutations that lead to constitutive kinase activation or impair normal FGFR4 inactivation lead to aberrant signaling.

Cellular Location
Cell membrane; Single-pass type I membrane protein. Endosome. Endoplasmic reticulum. Note=Internalized from the cell membrane to recycling endosomes, and from there back to the cell membrane

Tissue Location
Expressed in gastrointestinal epithelial cells, pancreas, and gastric and pancreatic cancer cell lines

FGFR4 Blocking Peptide (N-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

FGFR4 Blocking Peptide (N-term) - Images

FGFR4 Blocking Peptide (N-term) - Background

Tyrosine-protein kinase that acts as cell-surface receptor for fibroblast growth factors and plays a role in the regulation of cell proliferation, differentiation and migration, and in regulation of lipid metabolism, bile acid biosynthesis, glucose uptake, vitamin D metabolism and phosphate homeostasis. Required for normal down-regulation of the expression of CYP7A1, the rate-limiting enzyme in bile acid synthesis, in response to FGF19. Phosphorylates PLCG1 and FRS2. Ligand binding leads to the activation of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate. Phosphorylation of FRS2 triggers recruitment of GRB2, GAB1, PIK3R1 and SOS1, and mediates activation of RAS, MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. Promotes SRC-dependent phosphorylation of the matrix protease MMP14 and its lysosomal degradation. FGFR4 signaling is down-regulated by receptor internalization and degradation; MMP14 promotes internalization and degradation of FGFR4. Mutations that lead to constitutive kinase activation or impair normal FGFR4 inactivation lead to aberrant signaling.

FGFR4 Blocking Peptide (N-term) - References

Partanen J.M.,et al.EMBO J. 10:1347-1354(1991).
Ron D.,et al.J. Biol. Chem. 268:5388-5394(1993).
Takaishi S.,et al.Biochem. Biophys. Res. Commun. 267:658-662(2000).
Kostrzewa M.,et al.Mamm. Genome 9:131-135(1998).
Ezzat S.,et al.J. Clin. Invest. 109:69-78(2002).