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FGFR4 Antibody Blocking Peptide

Synthetic peptide
Catalog # BP7639d

Specification

FGFR4 Antibody Blocking Peptide - Product Information

Primary Accession P22455

FGFR4 Antibody Blocking Peptide - Additional Information

Gene ID 2264

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

Target/Specificity
The synthetic peptide sequence used to generate the antibody <a >AP7639d was selected from the region of human FGFR4. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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 Antibody Blocking Peptide - 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 Antibody Blocking Peptide - Protocols

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

- Blocking Peptides

FGFR4 Antibody Blocking Peptide - Images

FGFR4 Antibody Blocking Peptide - Background

FGFR4 is a member of the fibroblast growth factor receptor family, where amino acid sequence is highly conserved between members and throughout evolution. FGFR family members differ from one another in their ligand affinities and tissue distribution. A full-length representative protein would consist of an extracellular region, composed of three immunoglobulin-like domains, a single hydrophobic membrane-spanning segment and a cytoplasmic tyrosine kinase domain. The extracellular portion of the protein interacts with fibroblast growth factors, setting in motion a cascade of downstream signals, ultimately influencing mitogenesis and differentiation. Although alternative splicing has been observed, there is no evidence that the C-terminal half of the IgIII domain of this protein varies between three alternate forms, as indicated for family members 1-3. This particular family member preferentially binds acidic fibroblast growth factor and, although its specific function is unknown, it is overexpressed in gynecological tumor samples, suggesting a role in breast and ovarian tumorigenesis.

FGFR4 Antibody Blocking Peptide - References

Jezequel, P., et al., Br. J. Cancer 90(1):189-193 (2004).Qian, Z.R., et al., J. Clin. Endocrinol. Metab. 89(4):1904-1911 (2004).Yu, S., et al., J. Biol. Chem. 278(22):19597-19602 (2003).Becker, N., et al., Cancer Epidemiol. Biomarkers Prev. 12(6):582-583 (2003).Takenaka, H., et al., Arch. Dermatol. Res. 294(7):331-338 (2002).