

**FGFR-4 (phospho Tyr642) Polyclonal Antibody**  
**Catalog # AP67441****Specification**

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**FGFR-4 (phospho Tyr642) Polyclonal Antibody - Product Information**

Application	WB
Primary Accession	<a href="#">P22455</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

**FGFR-4 (phospho Tyr642) Polyclonal Antibody - Additional Information****Gene ID** 2264**Other Names**

FGFR4; JTK2; TKF; Fibroblast growth factor receptor 4; FGFR-4; CD antigen CD334

**Dilution**

WB~~Western Blot: 1/500 - 1/2000. ELISA: 1/10000. Not yet tested in other applications.

**Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

**Storage Conditions**

-20°C

**FGFR-4 (phospho Tyr642) Polyclonal Antibody - 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

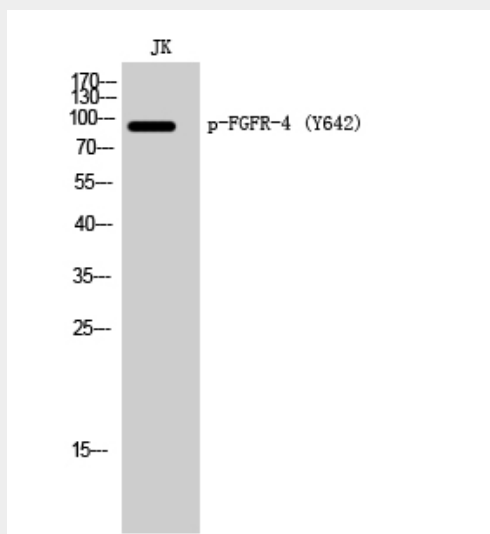
**Tissue Location**

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

**FGFR-4 (phospho Tyr642) Polyclonal Antibody - Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**FGFR-4 (phospho Tyr642) Polyclonal Antibody - Images****FGFR-4 (phospho Tyr642) Polyclonal Antibody - 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.