

**FGFR2 Antibody (C-term)**  
**Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP7637b****Specification**

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**FGFR2 Antibody (C-term) - Product Information**

Application	WB, IHC-P-Leica,E
Primary Accession	<a href="#">P21802</a>
Other Accession	<a href="#">P21803</a>
Reactivity	Human, Mouse
Predicted	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	92025
Antigen Region	794-821

**FGFR2 Antibody (C-term) - Additional Information****Gene ID** 2263**Other Names**

Fibroblast growth factor receptor 2, FGFR-2, K-sam, KGFR, Keratinocyte growth factor receptor, CD332, FGFR2, BEK, KGFR, KSAM

**Target/Specificity**

This FGFR2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 794-821 amino acids from the C-terminal region of human FGFR2.

**Dilution**WB~~1:1000  
IHC-P-Leica~~1:500**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

FGFR2 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**FGFR2 Antibody (C-term) - Protein Information****Name** FGFR2

**Synonyms** BEK, KGFR, KSAM

**Function** Tyrosine-protein kinase that acts as a cell-surface receptor for fibroblast growth factors and plays an essential role in the regulation of cell proliferation, differentiation, migration and apoptosis, and in the regulation of embryonic development. Required for normal embryonic patterning, trophoblast function, limb bud development, lung morphogenesis, osteogenesis and skin development. Plays an essential role in the regulation of osteoblast differentiation, proliferation and apoptosis, and is required for normal skeleton development. Promotes cell proliferation in keratinocytes and immature osteoblasts, but promotes apoptosis in differentiated osteoblasts. Phosphorylates PLCG1, FRS2 and PAK4. 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. FGFR2 signaling is down-regulated by ubiquitination, internalization and degradation. Mutations that lead to constitutive kinase activation or impair normal FGFR2 maturation, internalization and degradation lead to aberrant signaling. Over-expressed FGFR2 promotes activation of STAT1.

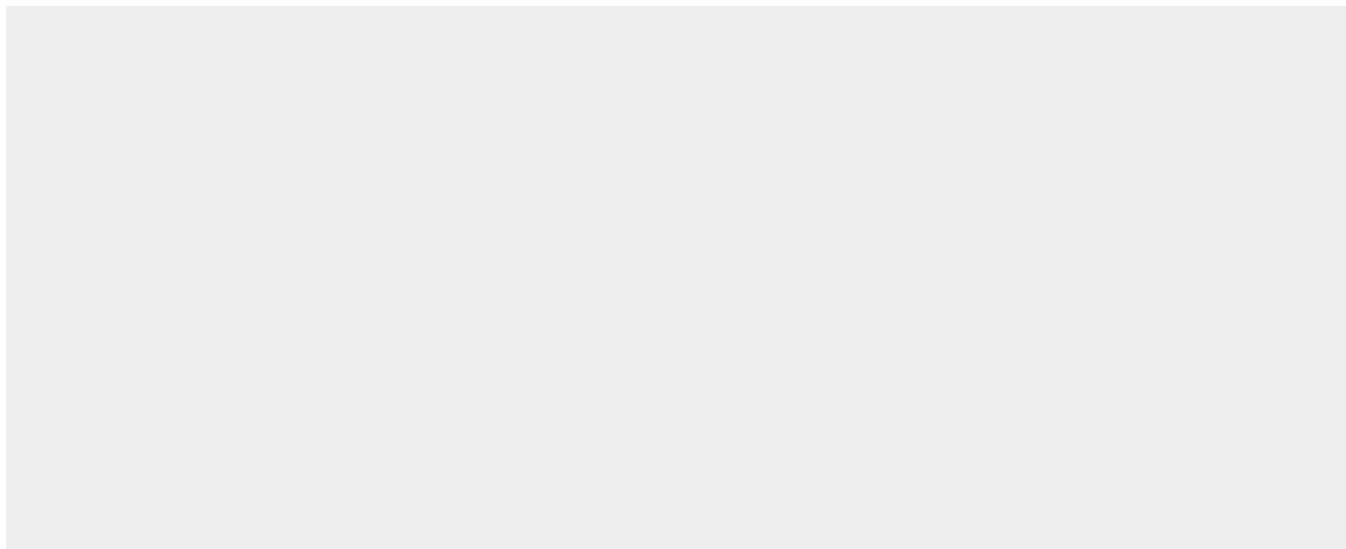
**Cellular Location**

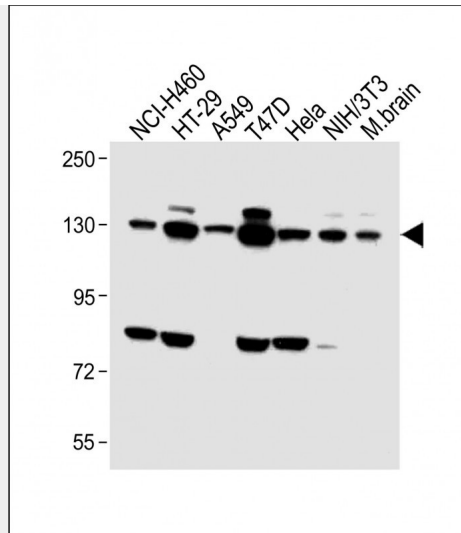
Cell membrane; Single-pass type I membrane protein. Golgi apparatus. Cytoplasmic vesicle. Note=Detected on osteoblast plasma membrane lipid rafts. After ligand binding, the activated receptor is rapidly internalized and degraded [Isoform 3]: Cell membrane; Single-pass type I membrane protein. Note=After ligand binding, the activated receptor is rapidly internalized and degraded [Isoform 13]: Secreted.

**FGFR2 Antibody (C-term) - Protocols**

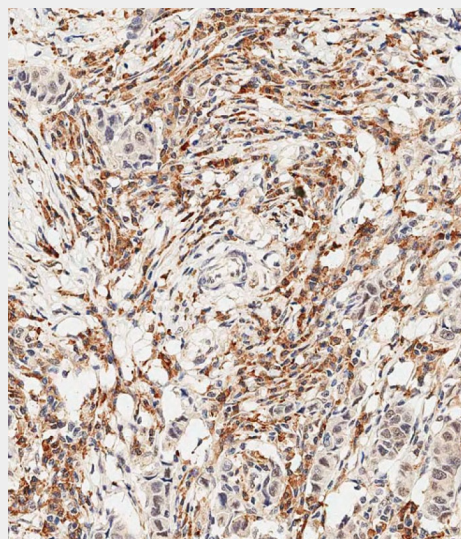
Provided below are standard protocols that you may find useful for product applications.

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

**FGFR2 Antibody (C-term) - Images**



All lanes : Anti-FGFR2 Antibody (C-term) at 1:1000 dilution Lane 1: NCI-H460 whole cell lysate Lane 2: HT-29 whole cell lysate Lane 3: A549 whole cell lysate Lane 4: T47D whole cell lysate Lane 5: HeLa whole cell lysate Lane 6: NIH/3T3 whole cell lysate Lane 7: Mouse brain lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 92 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Immunohistochemical analysis of paraffin-embedded Human breast carcinoma tissue using AP7637B performed on the Leica® BOND RXm. Tissue was fixed with formaldehyde at room temperature, antigen retrieval was by heat mediation with a EDTA buffer (pH9. 0). Samples were incubated with primary antibody(1:500) for 1 hours at room temperature. A undiluted biotinylated CRF Anti-Polyvalent HRP Polymer antibody was used as the secondary antibody.

**FGFR2 Antibody (C-term) - Background**

FGFR2 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 consists 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. This particular family member is a high-affinity receptor for acidic, basic and/or keratinocyte growth

factor, depending on the isoform. Mutations in the gene are associated with many craniosynostotic syndromes and bone malformations. The genomic organization of the gene encompasses 20 exons. Alternative splicing in multiple exons, including those encoding the Ig-like domains, the transmembrane region and the carboxyl terminus, results in varied isoforms which differ in structure and specificity. Isoform 1 has equal affinity for aFGF and bFGF but does not bind KGF.

#### **FGFR2 Antibody (C-term) - References**

- Freeman, K.W., et al., *Cancer Res.* 63(19):6237-6243 (2003).  
Goriely, A., et al., *Science* 301(5633):643-646 (2003).  
Fomenkov, A., et al., *J. Biol. Chem.* 278(26):23906-23914 (2003).  
Katoh, M., et al., *Int. J. Mol. Med.* 11(5):579-583 (2003).  
Katoh, M., et al., *Int. J. Oncol.* 22(5):1155-1159 (2003).