

FGFR2 Antibody (N-term R22)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7637D

Specification

FGFR2 Antibody (N-term R22) - Product Information

Application	IF, WB, FC,E
Primary Accession	P21802
Other Accession	P21803
Reactivity	Human
Predicted	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	92025
Antigen Region	7-37

FGFR2 Antibody (N-term R22) - 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 7-37 amino acids from the N-terminal region of human FGFR2.

Dilution

IF~~1:10~50
WB~~1:1000
FC~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

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 (N-term R22) is for research use only and not for use in diagnostic or therapeutic procedures.

FGFR2 Antibody (N-term R22) - 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.

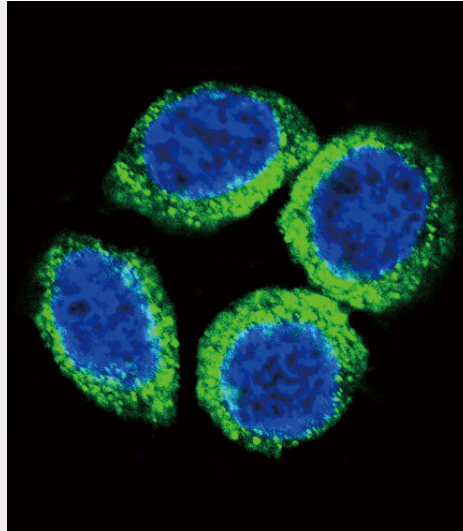
FGFR2 Antibody (N-term R22) - Protocols

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

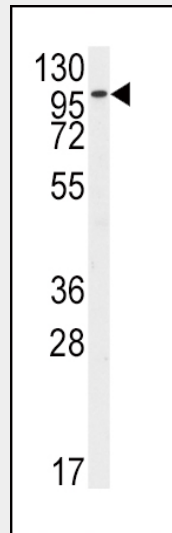
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

FGFR2 Antibody (N-term R22) - Images

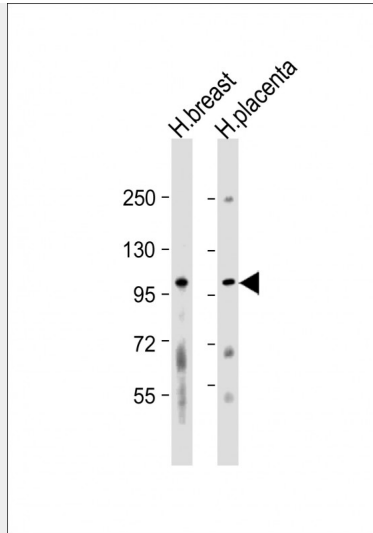




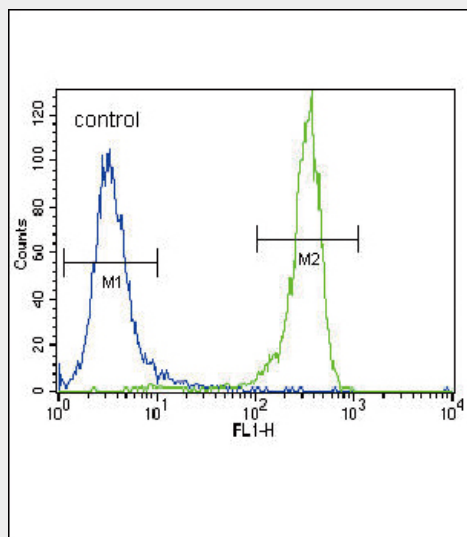
Confocal immunofluorescent analysis of FGFR2 Antibody (N-term R22)(Cat#AP7637d) with HeLa cell followed by Alexa Fluor 488-conjugated goat anti-rabbit IgG (green).DAPI was used to stain the cell nuclear (blue).



Western blot analysis of anti-FGFR2 Antibody (N-term R22) (Cat.#AP7637d) in HeLa cell line lysates (35ug/lane). FGFR2(arrow) was detected using the purified Pab.



All lanes : Anti-FGFR2 Antibody (N-term R22) at 1:1000 dilution Lane 1: human breast lysate Lane 2: human placenta lysate Lysates/proteins at 20 μ g per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 92 kDa Blocking/Dilution buffer: 5% NFDN/TBST.



FGFR2 Antibody (N-term R22) (Cat. #AP7637d) flow cytometric analysis of NCI-H460 cells (right histogram) compared to a negative control cell (left histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

FGFR2 Antibody (N-term R22) - 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 for FGFR2 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 (N-term R22) - 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).