

Product Information

Anti-Fibroblast Growth Factor Receptor-2, Cytoplasmic

produced in rabbit, affinity isolated antibody

Catalog Number **F0300**

Product Description

Anti-Fibroblast Growth Factor Receptor-2 (FGFR-2), Cytoplasmic is produced in rabbit using as immunogen a synthetic peptide (K-LPQYPHINGSVKT) corresponding to amino acid residues 809-821 of the cytoplasmic region of human FGFR-2 (or KGFR) with N-terminal added lysine. The peptide is conjugated to KLH with glutaraldehyde. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Fibroblast Growth Factor Receptor-2, Cytoplasmic reacts specifically with FGFR-2 by immunoblotting (doublet at 115-125 kDa) and immunoprecipitation, using whole cell lysates of transfected 293T cells, a human embryonic kidney cell line, expressing recombinant human FGFR-2. The antibody can also be used for the detection of FGFR-2 by immunohistochemistry using trypsin-digested, formalin-fixed, paraffin-embedded human and animal tissue sections. No reaction with human FGFR-1 and FGFR-3 is detected.

Fibroblast growth factors (FGFs) are members of a large family of structurally related heparin binding polypeptides (17-38 kDa) that are potent physiological regulators of growth and differentiation for a wide variety of cells of mesodermal, ectodermal and endodermal origin.¹⁻⁴ FGFs are substantially involved in normal development, wound healing and repair, angiogenesis, a variety of neurotrophic activities, in hematopoiesis as well as in tissue remodeling and maintenance. They have also been implicated in pathological conditions such as tumorigenesis and metastasis. The FGF family consists of at least seventeen members designated FGF-1 through FGF-17. To date, four genes encoding for high affinity cell surface FGF receptors (FGFRs) have been identified: FGFR-1 [flg, cek-1], FGFR-2 [bek, cek-3], FGFR-3 [cek-2] and FGFR-4. Multiple additional variants (isoforms) arising by alternative splicing have been reported.⁵⁻⁸ Soluble, secreted⁸ or possibly cleaved⁹ forms of FGFR-1 and FGFR-2 have also been found in body fluids or were artificially constructed.

FGFRs are members of the tyrosine kinase family of growth factor receptors. They are glycosylated 110-150 kDa proteins that are constructed of an extracellular ligand binding region with either two or typically three immunoglobulin (Ig)-like domains and an eight amino acid 'acidic box', a transmembrane region and a cytoplasmic split tyrosine kinase domain that is activated following ligand binding and receptor dimerization. The ligand binding site of all FGFRs is confined to the extracellular Ig-like domains 2 and 3.¹⁰ FGFRs exhibit overlapping recognition and redundant specificity. One receptor type may bind several of the FGFs with a similar affinity. Also one FGF type may bind similarly to several distinct receptors. This accounts for the rather identical effects of different FGF ligands on common cell types. FGFs binding to cellular FGFRs depends on, or is markedly facilitated by, the low-affinity interaction of FGFs with the polysaccharide component of cell surface or extracellular matrix heparan sulfate proteoglycans (HSPG).¹¹ For example, perlecan, a basement membrane HSPG, promotes high affinity binding of FGF2 *in vitro* and angiogenesis *in vivo*.¹² Signal transduction by FGFRs requires dimerization or oligomerization and autophosphorylation of the receptors through their tyrosine kinase domain. Subsequent association with cytoplasmic signaling molecules leads to DNA synthesis or differentiation. The signaling and biological responses elicited by distinct FGFRs substantially differ and are dictated by the intracellular domain.^{13,14} At the mRNA level, FGFR-2 is highly expressed in developing human tissues including the brain (preferentially in glial cells), choroid plexus, skin, lung, kidney and bone. It is widely expressed in many adult human and animal tissues.^{15,16} It may be found in several anchorage-dependent cells, such as normal and malignant breast cancer cells.¹⁷ Crouzon, as well as other craniosynostosis syndromes (e.g. Pfeiffer's, Apert's, Jackson-Weiss'), disorders of human skeletal development, have been shown to be the result of mutations in the extracellular domain of FGFR-2.¹⁸

Reagents

Supplied in 0.01 M phosphate buffered saline, pH 7.4, containing 1% BSA and 15 mM sodium azide as a preservative.

Protein concentration: ~1 mg/mL (A_{280})

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working antibody concentration of 0.25-0.5 µg/mL is determined using a whole extract of transfected cells expressing recombinant human FGFR-2.

Immunohistochemistry: a working antibody concentration of 2-4 µg/mL is determined using indirect immunoperoxidase staining with trypsin-digested, formalin-fixed, paraffin-embedded human and animal tissue sections.

Note: In order to obtain the best results in various techniques and preparations, we recommend determining optimal working dilutions by titration.

References:

1. Burgess, W.H., and Maciag, T., *Ann. Rev. Biochem.*, **58**, 575 (1989).
2. Klagsburn, M., *Prog. Growth Factor Res.*, **1**, 207 (1989).
3. Givol, D., and Yayon, A., *FASEB J.*, **6**, 3362 (1992).
4. Baird, A., et al., *Curr. Opin. Neurobiol.*, **4**, 78 (1994).
5. Yayon, A., et al., *EMBO J.*, **5**, 1885 (1992).
6. Miki, T., et al., *Proc. Natl. Acad. Sci. USA*, **89**, 246 (1992).
7. Johnson, D.E., et al., *Mol. Cell Biol.*, **11**, 4627 (1991).
8. Hanneken, A., et al., *Proc. Natl. Acad. Sci. USA*, **91**, 9170 (1994).
9. Levi, E., et al., *Proc. Natl. Acad. Sci. USA*, **93**, 7069 (1996).
10. Zimmer, Y., et al., *J. Biol. Chem.*, **268**, 7899 (1993).
11. Yayon, A., et al., *Cell*, **64**, 841 (1991).
12. Aviezer, D., et al., *Cell*, **79**, 1005 (1994).
13. Wang, J.K., et al., *Mol. Cell Biol.*, **14**, 181 (1994).
14. Friesel, R.E., and Maciag, T., *FASEB J.*, **9**, 919 (1995).
15. Hughes, S.E., et al., *J. Histochem. Cytochem.*, **45**, 1005 (1997).
16. Orr-Urtreger, A., et al., *Development.*, **113**, 1419 (1991).
17. Johnston, C.L., et al., *J. Biol. Chem.*, **270**, 30643 (1995).
18. Galvin, B. D., et al., *Proc. Natl. Acad. Sci. USA*, **93**, 7894 (1996).

MG,KAA,PHC 12/09-1