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Produktinformation



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PRODUCT INFORMATION



FGFR1 Extracellular Domain (human, recombinant)

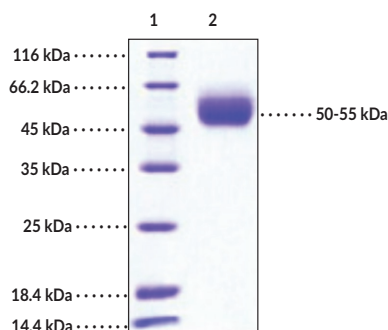
Item No. 33735

Overview and Properties

Synonyms:	Basic Fibroblast Growth Factor Receptor 1, bFGF-R-1, BFGFR, CD331, Fibroblast Growth Factor Receptor 1, FGFBR, FLT2, Fms-like Tyrosine Kinase 2
Source:	Active recombinant human C-terminal His-tagged FGFR1 expressed in HEK293 cells
Amino Acids:	22-285
Uniprot No.:	P11362
Molecular Weight:	31 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 years
Purity:	≥98% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/μg, determined by the LAL endotoxin assay
Bioactivity:	See figures for details

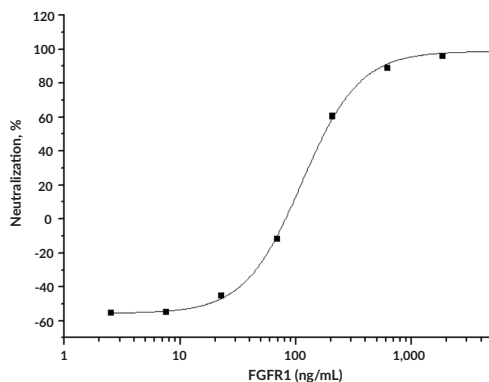
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: FGFR1 Extracellular Domain

SDS-PAGE Analysis of FGFR1 Extracellular Domain. This protein has a calculated molecular weight of 31 kDa. It has an apparent molecular weight of approximately 50-55 kDa by SDS-PAGE under reducing conditions due to glycosylation.



FGFR1 in a Cell Proliferation Assay. Measured by its ability to inhibit FGF acidic dependent proliferation of Balb/C 3T3 mouse fibroblasts. The EC₅₀ for this effect is typically 0.1-0.6 μg/ml.

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

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PRODUCT INFORMATION



Description

Fibroblast growth factor receptor 1 (FGFR1) is a growth factor receptor with roles in cell migration, differentiation, and survival, as well as apoptosis, chemotaxis, and embryonic development.^{1,2} It is composed of an N-terminal extracellular domain, which contains three immunoglobulin-like (Ig-like) domains, including the FGF ligand-binding domain, an acidic box, and a CAM-homology domain, a transmembrane domain, and a C-terminal tyrosine kinase domain. FGFR1 is expressed throughout the pre-differentiated mesenchyme of the cranium, in midfacial mesenchyme, and the endochondral skull base during early embryonic development, and broadly expressed in epithelial, osteogenic, and chondrogenic cell lineages by 10 to 13 weeks gestation.³ Upon ligand binding, FGFR1 dimerizes, resulting in autophosphorylation of the tyrosine kinase domain and activation of various intracellular signaling pathways, including the ERK/MAPK pathway.¹ Mutations in *FGFR1* induce various craniofacial dysostosis syndromes, such as Crouzon, Pfeiffer, and Jackson-Weiss syndromes that feature basicranial and midfacial deformities.³ *FGFR1* amplifications, translocations, and fusions are associated with lung cancer, 8p11 myeloproliferative syndrome, and glioblastoma multiforme, respectively.⁴ Cayman's FGFR1 Extracellular Domain (human, recombinant) can be used for enzyme activity assays. This protein consists of 275 amino acids, has a calculated molecular weight of 31 kDa, and a predicted N-terminus of Arg22 after signal peptide cleavage. By SDS-PAGE, under reducing conditions, the apparent molecular mass of the protein is 50-55 kDa due to glycosylation.

References

1. Böttcher, R.T. and Niehrs, C. Fibroblast growth factor signaling during early vertebrate development. *Endocr. Rev.* **26**(1), 63-77 (2005)
2. Villanueva, C. and de Roux, N. FGFR1 mutations in Kallmann syndrome. *Front. Horm. Res.* **39**, 51-61 (2010).
3. Britto, J.A., Evans, R.D., Hayward, R.D., *et al.* From genotype to phenotype: The differential expression of FGF, FGFR, and TGF β genes characterizes human cranioskeletal development and reflects clinical presentation in FGFR syndromes. *Plast. Reconstr. Surg.* **108**(7), 2026-2039 (2001).
4. Katoh, M. and Nakagama, H. FGF receptors: Cancer biology and therapeutics. *Med. Res. Rev.* **34**(2), 280-300 (2014).