

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived human FGFR2 alpha protein
Arg22-Glu377, with a C-terminal 6-His tag
Accession # P21802.1

N-terminal Sequence Analysis Arg22

Predicted Molecular Mass 40 kDa

SPECIFICATIONS

SDS-PAGE 58-72 kDa, under reducing conditions.

Activity Measured by its binding ability in a functional ELISA.
In a Human FGF acidic/FGF1 antibody (Catalog # [AF232](#)) coated plate, in the presence of 50.0 ng/mL of Recombinant Human FGF acidic/FGF1 (Catalog # [232-FA](#)), Human FGFR2 alpha (IIIc) His-tag Protein binds with an ED₅₀ of 0.400-2.40 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 500 µg/mL in PBS.

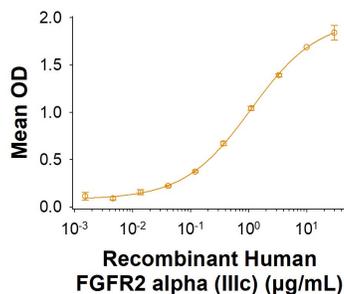
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

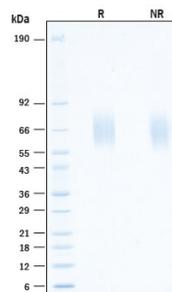
DATA

Bioactivity



Recombinant Human FGFR2 alpha (IIIc) His-tag Protein Bioactivity. In a Human FGF acidic/FGF1 antibody (Catalog # [AF232](#)) coated plate, in the presence of 50.0 ng/mL of Recombinant Human FGF acidic/FGF1 (Catalog # [232-FA](#)), Human FGFR2 alpha (IIIc) His-tag Protein (Catalog # 11119-FR) binds with an ED₅₀ of 0.400-2.40 µg/mL.

SDS-PAGE



Recombinant Human FGFR2 alpha (IIIc) His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant Human FGFR2 alpha (IIIc) His-tag Protein (Catalog # 11119-FR) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 58-72 kDa.

BACKGROUND

Fibroblast growth factor receptor 2 (FGFR2) belongs to a family of type I transmembrane tyrosine kinases which mediate the biological functions of FGFs that are involved in a multitude of physiological and pathological cellular processes (1). The FGFR family is comprised of 4 structurally conserved members (FGFR1-4) all possessing an extracellular domain (ECD) with three immunoglobulin (Ig)-like domains, an acid-box region containing a run of acidic residues between the IgI and IgII domains, a transmembrane domain and cytoplasmic split tyrosine-kinase domain (1, 2). The ECD of mature, full-length FGFR2 shares 95% amino acid sequence identity with mouse FGFR2. Alternative splicing generates multiple forms of FGFR1-3, each with unique signaling characteristics (1-3). For FGFR2, alternative splicing of the ECD, specifically the IgIII domain, results in IIIb, or IIIc isoforms (4). The FGFR splice variants also exhibit distinct and varying binding affinities for different FGF ligands (2, 4). Specifically, FGFR2A (IIIc) binds most FGF ligands but not the FGF10 subfamily, while FGFR2A (IIIb) binds only members of the FGF10 subfamily (5). FGFRs mediate the FGF signaling cascade which regulate developmental processes including cellular proliferation, differentiation, and migration, morphogenesis, and patterning (6). FGFRs transduce the signals through three dominant pathways including RAS/MAPK, PI3k/AKT, and PLC γ (7). While FGFR2 is widely expressed in many adult human tissues, isoform expression is tissue specific, with IIIb predominantly expressed in epithelial cells, while IIIc is expressed in mesenchymal cells (5). FGFR2 signaling is critical for embryonic development, tissue repair, and regulation of osteoblast function and bone growth (8). Mutations in FGFR2 or misregulation of FGFR2 mediated signaling is found in multiple skeletal dysplasias, with FGFR2A (IIIc) specifically upregulated in several cancers including prostate, breast and pancreatic and is proposed as a novel therapeutic target for colorectal carcinomas (6, 9).

References:

1. Ornitz, D.M. and Itoh, N. (2015) Wiley Interdiscip Rev Dev Biol. **4**:215.
2. Zhang, X. *et al.* (2006) J Biol Chem. **281**:15694.
3. Ferguson, H.R. *et al.* (2021) Signaling. Cells **10**:1201.
4. Holzmann, K. *et al.* (2012) J Nucleic Acids. **2012**:950508.
5. Wagner, E.J. *et al.* (2003) RNA **9**:1552.
6. Xie, Y. *et al.* (2020) Sig Transduct Target Ther **5**:181.
7. Mossahebi-Mohammadi, M. *et al.* (2020) Front Cell Dev Biol. **18**:79.
8. Teven, C.M. *et al.* (2014) Genes Dis. **1**:199.
9. Matsuda, Y. *et al.* (2012) Mol Cancer Ther. **11**:2010.