

Human FGFR1 alpha (IIIc) Antibody

Monoclonal Mouse IgG_{2B} Clone # 1058809 Catalog Number: MAB11336

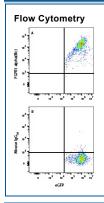
DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human FGFR1 alpha (IIIc) in direct ELISA.
Source	Monoclonal Mouse IgG _{2B} Clone # 1058809
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Human embryonic kidney cell, HEK293-derived human FGFR1 alpha Arg22-Glu376 Accession # P11362.3
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Flow Cytometry	0.25 μg/10 ⁶ cells	HEK293 cells transfected with hFGFR1 and eGFP

DATA



Detection of FGFR1 alpha in HEK293 cells transfected with hFGFR1 and eGFP cells by Flow Cytometry. HEK293 cells transfected with hFGFR1 and eGFP were stained with either (A) Mouse Anti-Human FGFR1 alpha (IIIc) Monoclonal Antibody (Catalog # MAB11336) or (B) Mouse IgG2_B Isotype Control (Catalog # MAB004). View our protocol for Staining Membrane-associated Proteins.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in steme PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
	*Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C

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.
- 6 months, -20 to -70 °C under sterile conditions after reconstitution.





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BACKGROUND

Fibroblast growth factor receptor 1 (FGFR1) 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 FGFR1 shares 98% amino acid sequence identity with mouse FGFR1. Alternative splicing generates multiple forms of FGFR1-3, each with unique signaling characteristics (1-3). For FGFR1, alternative splicing of the ECD generates FGFR1A, FGFR1B, and FGFR1G isoforms of the receptor with the A isoform containing three Ig domains, while the B and G isoforms lack the N-terminal IgI domain (3). Additional splicing of the IgIII domain, results in IIIa, IIIb, or IIIc isoforms (3). Only the alpha isoform has been identified for FGFR3 and FGFR4 and FGFR4 also lacks the IIIb and IIIc splicing events (4). The FGFR splice variants also exhibit distinct and varying binding affinities for different FGF ligands (2). FGFRs mediate the FGF signaling cascade which regulate developmental processes including cellular proliferation, differentiation, and migration, morphogenesis, and patterning (5). FGFRs transduce the signals through three dominant pathways including RAS/MAPK, Pl3k/AKT, and PLCγ (6). FGFR1 the most abundant FGFR and is widely expressed in many adult human tissues, but the splice variants display distinct tissue-specific differences with IIIc splice variants expressed in mesenchymal tissue (4, 7, 8). Mutations in FGFR1 or misregulation of FGFR1 mediated signaling is found in multiple diseases, with FGFR1A(IIIc) specifically upregulated, from breast and pancreatic cancer to Pfeiffer syndrome and osteoarthritis (4, 9-11). A solub

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. Xie, Y. et al. (2020) Sig. Transduct. Target Ther. 5:181.
- 6. Mossahebi-Mohammadi, M. et al. (2020) Front Cell Dev. Biol. 18:79.
- 7. Hughes, S.E. (1997) J. Histochem. Cytochem. 45:1005.
- 8. Delezoide, A.L. et al. (1998) Mech. Dev. 77:19.
- 9. Yamashita-Sugahara, Y. et al. (2016) Sci. Rep. 6:35908.
- 10. Teven, C.M. et al. (2014) Genes Dis. 1:199.
- 11. Babina, I. and Turner, N. (2017) Nat. Rev Cancer 17:318