Human FGFR1 alpha (IIIc) APC-conjugated Antibody

Monoclonal Mouse IgG_{2B} Clone # 1058809
Catalog Number: FAB11336A
100 Tests

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
Conjugate	Allophycocyanin Excitation Wavelength: 620-650 nm Emission Wavelength: 660-670 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

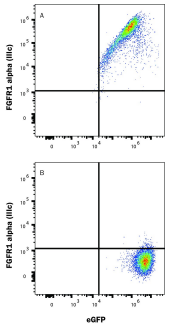
APPLICATIONS

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	10 µL/10^6 cells	HEK293 cells transfected with Human FGFR1 and eGFP vs irrelevant

DATA

Flow Cytometry



Detection of FGFR1 alpha in HEK293 cells by Flow Cytometry. HEK293 cells transfected with Human FGFR1 and eGFP (A) vs irrelevant and eGFP (B) were stained with Mouse Anti-Human FGFR1 alpha APC-conjugated Monoclonal Antibody (Catalog # FAB11336A) followed by Allophycocyanin-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # F0101B). View our protocol for Staining Membrane-associated Proteins.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Protect from light. Do not freeze.** • 12 months from date of receipt, 2 to 8 °C as supplied.

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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, PI3k/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 soluble version of the FGFR1A(IIIc) splice variant has shown anti-angiogenic and anti-proliferative properties in multiple cancer cell line models (11).

References:

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