FGFR-3 alpha (IIIc) Protein, Human (HEK293, His-Avi)

Cat. No.:	HY-P78439
Synonyms:	ACH; CD333; CEK; CEK2; EC 2.7.10; FGF R3; FGFR3; HSFGFR3EX; JTK4
Species:	Human
Source:	HEK293
Accession:	P22607 (E23-G375)
Gene ID:	2261
Molecular Weight:	65-75 kDa

PROPERTIES

Biological Activity	Immobilized Human FGFR3 alpha (IIIc) at 0.5 μg/mL (100 μl/Well) on the plate. Dose response curve for Anti-FGFR3 Antibody, hFc Tag with the EC_{50} of ≤11.7 ng/mL determined by ELISA.
Appearance	Lyophilized powder
Formulation	Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4. Normally 5% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	FGFR-3 protein, a tyrosine-protein kinase, functions as a cell-surface receptor for fibroblast growth factors, playing a vital role in the regulation of cell proliferation, differentiation, and apoptosis. Its significance is particularly notable in the regulation of chondrocyte differentiation, proliferation, and apoptosis, contributing to normal skeleton development. Additionally, FGFR-3 plays a crucial role in both osteogenesis and postnatal bone mineralization by osteoblasts, while also promoting apoptosis in chondrocytes. Beyond its role in normal development, FGFR-3 is involved in inner ear development and has implications in the regulation of vitamin D metabolism. Upon ligand binding, FGFR-3 activates several signaling cascades, including the phosphorylation of PLCG1, CBL, and FRS2. This activation leads to the production of cellular signaling molecules such as diacylglycerol and inositol 1,4,5-trisphosphate. Furthermore, phosphorylation of FRS2 triggers the recruitment of GRB2, GAB1, PIK3R1, and SOS1, mediating the activation of RAS, MAPK1/ERK2, MAPK3/ERK1, the MAP kinase signaling pathway, and the AKT1 signaling pathway. Mutations leading to constitutive kinase activation or impairing normal FGFR3 maturation, internalization, and degradation result in aberrant signaling. Overexpression or constitutive activation of FGFR3 promotes the activation of PTPN11/SHP2, STAT1, STAT5A, and STAT5B. Additionally, the secreted

	isoform 3 retains its capacity to bind FGF1 and FGF2, potentially interfering with FGF signaling.

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