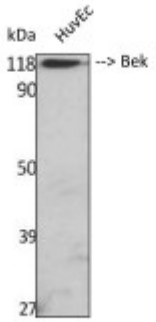
Anti-Bek antibody



Description	Rabbit polyclonal to Bek.

Model	STJ91850
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human Bek
Immunogen Region	440-520 aa, Internal
Gene ID	2263
Gene Symbol	FGFR2
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:10000
Specificity	Bek Polyclonal Antibody detects endogenous levels of Bek protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Fibroblast growth factor receptor 2 FGFR-2 K-sam KGFR Keratinocyte growth factor receptor CD antigen CD332
Molecular Weight	92 kDa
Clonality	Polyclonal
Conjugation	Unconjugated

Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:3689OMIM:101200
Alternative Names	Fibroblast growth factor receptor 2 FGFR-2 K-sam KGFR Keratinocyte growth factor receptor CD antigen CD332
Function	Tyrosine-protein kinase that acts as cell-surface receptor for fibroblast growth factors and plays an essential role in the regulation of cell proliferation, differentiation, migration and apoptosis, and in the regulation of embryonic development. Required for normal embryonic patterning, trophoblast function, limb bud development, lung morphogenesis, osteogenesis and skin development. Plays an essential role in the regulation of osteoblast differentiation, proliferation and apoptosis, and is required for normal skeleton development. Promotes cell proliferation in keratinocytes and immature osteoblasts, but promotes apoptosis in differentiated osteoblasts. Phosphorylates PLCG1, FRS2 and PAK4. Ligand binding leads to the activation of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate. Phosphorylation of FRS2 triggers recruitment of GRB2, GAB1, PIK3R1 and SOS1, and mediates activation of RAS, MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. FGFR2 signaling is down-regulated by ubiquitination, internalization and degradation. Mutations that lead to constitutive kinase activation or impair normal FGFR2 maturation, internalization and degradation lead to aberrant signaling. Over-expressed FGFR2 promotes activation of STAT1.
Sequence and Domain Family	The second and third Ig-like domains directly interact with fibroblast growth factors (FGF) and heparan sulfate proteoglycans. Alternative splicing events affecting the third Ig-like domain are crucial for ligand selectivity.
Cellular Localization	Cell membrane. Single-pass type I membrane protein. Golgi apparatus. Cytoplasmic vesicle. Detected on osteoblast plasma membrane lipid rafts. After ligand binding, the activated receptor is rapidly internalized and degraded.. Isoform 1: Cell membrane. Single-pass type I membrane protein. After ligand binding, the activated receptor is rapidly internalized and degraded.. Isoform 3: Cell membrane. Single-pass type I membrane protein. After ligand binding, the activated receptor is rapidly internalized and degraded.. Isoform 14: Secreted.. Isoform 19: Secreted.
Post-translational Modifications	Autophosphorylated. Binding of FGF family members together with heparan sulfate proteoglycan or heparin promotes receptor dimerization and autophosphorylation on several tyrosine residues. Autophosphorylation occurs in trans between the two FGFR molecules present in the dimer. Phosphorylation at Tyr-769 is essential for interaction with PLCG1. N-glycosylated in the endoplasmic reticulum. The N-glycan chains undergo further maturation to an Endo H-resistant form in the Golgi apparatus. Ubiquitinated. FGFR2 is rapidly ubiquitinated after autophosphorylation, leading to internalization and degradation. Subject to degradation both in lysosomes and by the proteasome.

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