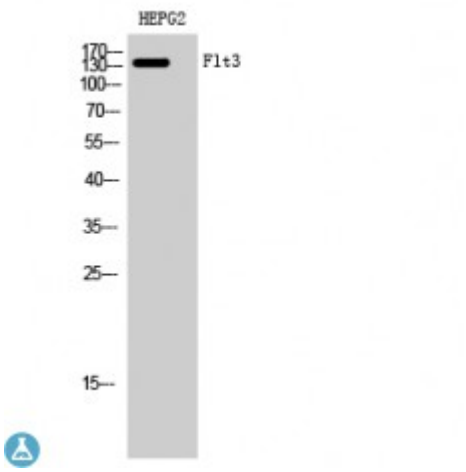
Anti-Flt3 antibody



Description	Rabbit polyclonal to Flt3.

Model	STJ93095
Host	Rabbit
Reactivity	Human, Mouse
Applications	ELISA, WB
Immunogen	Synthesized peptide derived from human Flt3 around the non-phosphorylation site of Y599.
Immunogen Region	540-620 aa
Gene ID	2322
Gene Symbol	FLT3
Dilution range	WB 1:500-1:2000ELISA 1:20000
Specificity	Flt3 Polyclonal Antibody detects endogenous levels of Flt3 protein.
Tissue Specificity	Detected in bone marrow, in hematopoietic stem cells, in myeloid progenitor cells and in granulocyte/macrophage progenitor cells (at protein level). Detected in bone marrow, liver, thymus, spleen and lymph node, and at low levels in kidney and pancreas. Highly expressed in T-cell leukemia.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Receptor-type tyrosine-protein kinase FLT3 FL cytokine receptor Fetal liver kinase-2 FLK-2 Fms-like tyrosine kinase 3 FLT-3 Stem cell tyrosine kinase 1

	STK-1 CD antigen CD135
Molecular Weight	117 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:3765OMIM:136351
Alternative Names	Receptor-type tyrosine-protein kinase FLT3 FL cytokine receptor Fetal liver kinase-2 FLK-2 Fms-like tyrosine kinase 3 FLT-3 Stem cell tyrosine kinase 1 STK-1 CD antigen CD135
Function	Tyrosine-protein kinase that acts as cell-surface receptor for the cytokine FLT3LG and regulates differentiation, proliferation and survival of hematopoietic progenitor cells and of dendritic cells. Promotes phosphorylation of SHC1 and AKT1, and activation of the downstream effector MTOR. Promotes activation of RAS signaling and phosphorylation of downstream kinases, including MAPK1/ERK2 and/or MAPK3/ERK1. Promotes phosphorylation of FES, FER, PTPN6/SHP, PTPN11/SHP-2, PLCG1, and STAT5A and/or STAT5B. Activation of wild-type FLT3 causes only marginal activation of STAT5A or STAT5B. Mutations that cause constitutive kinase activity promote cell proliferation and resistance to apoptosis via the activation of multiple signaling pathways.
Sequence and Domain Family	The juxtamembrane autoregulatory region is important for normal regulation of the kinase activity and for maintaining the kinase in an inactive state in the absence of bound ligand. Upon tyrosine phosphorylation, it mediates interaction with the SH2 domains of numerous signaling partners. In-frame internal tandem duplications (ITDs) result in constitutive activation of the kinase. The activity of the mutant kinase can be stimulated further by FLT3LG binding.
Cellular Localization	Membrane. Single-pass type I membrane protein. Endoplasmic reticulum lumen. Constitutively activated mutant forms with internal tandem duplications are less efficiently transported to the cell surface and a significant proportion is retained in an immature form in the endoplasmic reticulum lumen. The activated kinase is rapidly targeted for degradation.
Post-translational Modifications	N-glycosylated, contains complex N-glycans with sialic acid. Autophosphorylated on several tyrosine residues in response to FLT3LG binding. FLT3LG binding also increases phosphorylation of mutant kinases that are constitutively activated. Dephosphorylated by PTPRJ/DEP-1, PTPN1, PTPN6/SHP-1, and to a lesser degree by PTPN12. Dephosphorylation is important for export from the endoplasmic reticulum and location at the cell membrane.; Rapidly ubiquitinated by UBE2L6 and the E3 ubiquitin-protein ligase SIAH1 after autophosphorylation, leading to its proteasomal degradation.

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