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FLT3 Rabbit pAb

Catalog # AP96563

Product Information

Application	WB, IHC-P, IHC-F, IF
Primary Accession	Q00342
Other Accession	Q00342
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	113496
Physical State	Liquid
Immunogen	Recombinant mouse Flt3 protein
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the protein kinase superfamily. Tyr protein kinase family. CSF-1/PDGF receptor subfamily.Contains 1 Ig-like C2-type (immunoglobulin-like) domain.Contains 1 protein kinase domain.
SUBUNIT	Monomer in the absence of bound FLT3LG. Homodimer in the presence of bound FLT3LG. One homodimer interacts with one FLT3LG molecule. Interacts with FIZ1 following ligand activation (By similarity). Interacts with FES, FER and GRB2. Interacts with PTPRJ/DEP-1 and PTPN11/SHP2.
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.
DISEASE	Defects in FLT3 are a cause of acute myelogenous leukemia (AML) [MIM:601626]. AML is a malignant disease in which hematopoietic precursors are arrested in an early stage of development. Note=Somatic mutations that lead to constitutive activation of FLT3 are frequent in AML patients. These mutations fall into two classes, the most common being in-frame internal tandem duplications of variable length in the juxtamembrane region that disrupt the normal regulation of the kinase activity. Likewise, point mutations in the activation loop of the kinase domain can result in a constitutively activated kinase.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	CD135 is a tyrosine kinase receptor expressed on normal cells including CD34+ hematopoietic stem cells, myelomonocytic progenitors, primitive B cell progenitors, and thymocytes. CD135 is also expressed on malignant hematopoietic cells including AML, ALL and CML BC. CD135, also known as FMS-like tyrosine kinase 3, FLT3, STK1, and Flk2, is a growth factor receptor that binds the FLT3 ligand to promote the growth and differentiation of primitive hematopoietic cells. The intracytoplasmic domain of CD135 is modified by phosphorylation and has been shown to interact with Grb2,

SOCS1, VAV1, and Shc. In humans, expression of Flt3 is restricted to subsets of CD34 positive as well as CD34 negative normal bone marrow cells. In these cells, the level of expression of Flt3 is rather low. Most of the CD34 bright Flt3+ cells co-express CD117 at high levels. They may represent early cycling, but not quiescent stem cells. Flt3+ cells in the CD34lo and CD34- populations do not co-express CD117 molecule and may represent B lymphoid precursors.

Additional Information

Gene ID	14255
Other Names	CD135; FLK-2; FLK2; STK1; B230315G04; Flt-3; Ly72; wmfl; FLT3_HUMAN; FLT3; FL cytokine receptor; Fetal liver kinase-2 (FLK-2); Fms-like tyrosine kinase 3 (FLT-3); Stem cell tyrosine kinase 1 (STK-1); 2.7.10.1; FLT3_MOUSE; Fetal liver kinase 2 (FLK-2); Tyrosine-protein kinase receptor flk-2; fms related receptor tyrosine kinase 3; fms-related tyrosine kinase 3; fms related tyrosine kinase 3
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	Flt3
Synonyms	Flk-2, Flt-3
Function	Tyrosine-protein kinase that acts as a 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.
Cellular Location	Membrane; Single-pass type I membrane protein. Endoplasmic reticulum lumen. Note=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 (By similarity).
Tissue Location	Hematopoietic stem and progenitor cell-enriched populations. Found in brain, placenta and testis

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