SunLong Biotech Co.,LTD
Tel: 0086-571- 56623320 Fax:0086-571- 56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

Rabbit Anti-FLT-3 antibody

SL2767R

Product Name:	FLT-3
Chinese Name:	FMS样酪氨酸激酶3
Alias:	CD135 antigen; Fetal liver kinase 2; FL cytokine receptor; Flk 2; Flk2; Flt 3; Flt3; FMS like tyrosine kinase 3; Fms related tyrosine kinase 3; Growth factor receptor tyrosine kinase type III; Stem cell tyrosine kinase 1; Stk 1; Stk1; Tyrosine protein kinase receptor FLT3; FLT3_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Dog,Cow,Horse,Rabbit,
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	109kDa
Cellular localization:	cytoplasmicThe cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human FLT-3:551-650/993<Cytoplasmic>
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. 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.
PubMed:	PubMed
Product Detail:	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. **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. **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. **Subcellular Location:** Membrane; Single-pass type I membrane protein. Endoplasmic reticulum lumen. **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. **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.

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.

SWISS:
P36888

Gene ID:
2322

Database links:

Entrez Gene: 2322 Human

Entrez Gene: 14255 Mouse

Entrez Gene: 140635 Rat

Omim: 136351 Human

SwissProt: P36888 Human

SwissProt: Q00342 Mouse

Unigene: 507590 Human

Unigene: 194 Mouse

Unigene: 6774 Rat

Important Note:
This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.