磷酸化 FMS 样酪氨酸激酶 3

产品货号： mlR3147

英文名称： Phospho-FLT3 (Tyr591)

中文名称： 磷酸化 FMS 样酪氨酸激酶 3

别　　名： Flt3 / CD135 (phospho Y591); p-CD135 (phospho Y591); Flt3 / CD135 (phospho Y591); p-Flt3 (phospho Y591); ; 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.

产品类型： 磷酸化抗体

研究领域： 信号转导　激酶和磷酸酶

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应　： Human, Mouse,

产品应用： WB=1:500-2000 ELISA=1:500-1000 IHC-P=1:400-800 IHC-F=1:400-800 Flow-Cyt=3ug/Test IF=1:100-500 （石蜡切片需做抗原修复）
not yet tested in other applications.
optimal dilutions/concentrations should be determined by the end user.

分 子 量： 109kDa

细胞定位： 细胞浆 细胞膜

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原 ： KLH conjugated Synthesised phosphopeptide derived from human FLT3 around the phosphorylation site of Tyr591:YF(p-Y)VD

亚 型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件 ： 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

产品介绍 ： 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.

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

Important Note:

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