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FLT3 Blocking Peptide (Center)

Synthetic peptide
Catalog # BP20919a

Specification

FLT3 Blocking Peptide (Center) - Product Information

Primary Accession P36888

FLT3 Blocking Peptide (Center) - Additional Information

Gene ID 2322

Other 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, CD135, FLT3, CD135, FLK2, STK1

Target/Specificity
The synthetic peptide sequence is selected from aa 474-490 of HUMAN FLT3

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

FLT3 Blocking Peptide (Center) - Protein Information

Name FLT3

Synonyms CD135, FLK2, STK1

Function
Tyrosine-protein kinase that acts as

FLT3 Blocking Peptide (Center) - Background

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.

FLT3 Blocking Peptide (Center) - References

Small D.,et al.Proc. Natl. Acad. Sci. U.S.A. 91:459-463(1994).
Rosnet O.,et al.Blood 82:1110-1119(1993).
Dunham A.,et al.Nature 428:522-528(2004).
Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.
Rosnet O.,et al.Genomics 9:380-385(1991).

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

Tissue Location
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

FLT3 Blocking Peptide (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides