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FLT3 (CD135) Antibody (N-term) Blocking peptide

Synthetic peptide
Catalog # BP7644a

Specification

FLT3 (CD135) Antibody (N-term) Blocking peptide - Product Information

Primary Accession P36888

FLT3 (CD135) Antibody (N-term) Blocking peptide - 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 used to generate the antibody AP7644a was selected from the N-term region of human FLT3 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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 (CD135) Antibody (N-term) Blocking peptide - Protein Information

Name FLT3

Synonyms CD135, FLK2, STK1

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

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 (CD135) Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

FLT3 (CD135) Antibody (N-term) Blocking peptide - Images

FLT3 (CD135) Antibody (N-term) Blocking peptide - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the g phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains. The tyrosine kinase (TK) group is mainly involved in the regulation of cell-cell interactions such as differentiation, adhesion, motility and death. There are currently about 90 TK genes sequenced, 58 are of receptor protein TK (e.g. EGFR, EPH, FGFR, PDGFR, TRK, and VEGFR families), and 32 of cytosolic TK (e.g. ABL, FAK, JAK, and SRC families).

FLT3 (CD135) Antibody (N-term) Blocking peptide - References

Minami, Y., et al., Blood 102(8):2969-2975 (2003).Zwaan, C.M., et al., Blood 102(7):2387-2394 (2003).Sitnicka, E., et al., Blood 102(3):881-886 (2003).Galimberti, S., et al., Anticancer Res. 23(4):3419-3426 (2003).Kottaridis, P.D., et al., Leuk. Lymphoma 44(6):905-913 (2003).