

**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 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 Blocking Peptide (Center) - Protocols**

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

- [Blocking Peptides](#)

#### **FLT3 Blocking Peptide (Center) - Images**

#### **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).