

**Phospho-KIT(Y703) Antibody Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP3570a****Specification**

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**Phospho-KIT(Y703) Antibody Blocking peptide - Product Information**Primary Accession [P10721](#)**Phospho-KIT(Y703) Antibody Blocking peptide - Additional Information****Gene ID** 3815**Other Names**

Mast/stem cell growth factor receptor Kit, SCFR, Piebald trait protein, PBT, Proto-oncogene c-Kit, Tyrosine-protein kinase Kit, p145 c-kit, v-kit Hardy-Zuckerman 4 feline sarcoma viral oncogene homolog, CD117, KIT, SCFR

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP3570a](/products/AP3570a) was selected from the region of human Phospho-KIT-pY703. 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.

**Phospho-KIT(Y703) Antibody Blocking peptide - Protein Information****Name** KIT**Synonyms** SCFR**Function**

Tyrosine-protein kinase that acts as a cell-surface receptor for the cytokine KITLG/SCF and plays an essential role in the regulation of cell survival and proliferation, hematopoiesis, stem cell maintenance, gametogenesis, mast cell development, migration and function, and in melanogenesis. In response to KITLG/SCF binding, KIT can activate several signaling pathways. Phosphorylates PIK3R1, PLCG1, SH2B2/APS and CBL. Activates the AKT1 signaling pathway by phosphorylation of PIK3R1, the regulatory subunit of phosphatidylinositol 3-kinase. Activated KIT also transmits signals via GRB2 and activation of RAS, RAF1 and the MAP kinases MAPK1/ERK2 and/or MAPK3/ERK1. Promotes activation of STAT family members STAT1, STAT3, STAT5A and

STAT5B. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5- trisphosphate. KIT signaling is modulated by protein phosphatases, and by rapid internalization and degradation of the receptor. Activated KIT promotes phosphorylation of the protein phosphatases PTPN6/SHP-1 and PTPRU, and of the transcription factors STAT1, STAT3, STAT5A and STAT5B. Promotes phosphorylation of PIK3R1, CBL, CRK (isoform Crk-II), LYN, MAPK1/ERK2 and/or MAPK3/ERK1, PLCG1, SRC and SHC1.

#### **Cellular Location**

[Isoform 1]: Cell membrane; Single-pass type I membrane protein [Isoform 3]: Cytoplasm.

Note=Detected in the cytoplasm of spermatozoa, especially in the equatorial and subacrosomal region of the sperm head.

#### **Tissue Location**

[Isoform 3]: In testis, detected in spermatogonia in the basal layer and in interstitial Leydig cells but not in Sertoli cells or spermatocytes inside the seminiferous tubules (at protein level) (PubMed:20601678). Expression is maintained in ejaculated spermatozoa (at protein level) (PubMed:20601678)

### **Phospho-KIT(Y703) Antibody Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

### **Phospho-KIT(Y703) Antibody Blocking peptide - Images**

### **Phospho-KIT(Y703) Antibody Blocking peptide - Background**

KIT is the human homolog of the proto-oncogene c-kit. C-kit was first identified as the cellular homolog of the feline sarcoma viral oncogene v-kit. KIT is a type 3 transmembrane receptor for MGF (mast cell growth factor, also known as stem cell factor). Mutations in KIT are associated with gastrointestinal stromal tumors, mast cell disease, acute myelogenous leukemia, and piebaldism.

### **Phospho-KIT(Y703) Antibody Blocking peptide - References**

Wardelmann, E., et al., Int. J. Cancer 106(6):887-895 (2003).Lennartsson, J., et al., Exp. Cell Res. 288(1):110-118 (2003).Sakuma, Y., et al., Cancer Sci 94(6):486-491 (2003).Araki, K., et al., Lung Cancer 40(2):173-180 (2003).Voytyuk, O., et al., J. Biol. Chem. 278(11):9159-9166 (2003).