

**PIM2 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7933a****Specification****PIM2 Antibody (C-term) Blocking Peptide -  
Product Information**Primary Accession [Q9P1W9](#)**PIM2 Antibody (C-term) Blocking Peptide -  
Additional Information****Gene ID** 11040**Other Names**Serine/threonine-protein kinase pim-2,  
Pim-2h, PIM2**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [<a href=/product/products/AP7933a>AP7933a</a>](#) was selected from the C-term region of human PIM2 . 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.

**PIM2 Antibody (C-term) Blocking Peptide -  
Protein Information****Name** PIM2**Function****PIM2 Antibody (C-term) Blocking Peptide -  
Background**

Pim proteins (Pim-1, Pim-2 and Pim-3) are oncogene-encoded serine/threonine kinases. Pim-2 is highly homologous to Pim-1 with similar oncogenic functions. Pim-2 overexpression promotes resistance to a host of apoptotic stimuli; its expression is negatively regulated by growth factor depletion. Increased levels of Pim-2 has also been observed in certain cancers.

**PIM2 Antibody (C-term) Blocking Peptide -  
References**

Yan, B., et al., J. Biol. Chem. 278(46):45358-45367 (2003). Baytel, D., et al., Biochim. Biophys. Acta 1442 (2-3), 274-285 (1998).

Proto-oncogene with serine/threonine kinase activity involved in cell survival and cell proliferation. Exerts its oncogenic activity through: the regulation of MYC transcriptional activity, the regulation of cell cycle progression, the regulation of cap-dependent protein translation and through survival signaling by phosphorylation of a pro-apoptotic protein, BAD. Phosphorylation of MYC leads to an increase of MYC protein stability and thereby an increase transcriptional activity. The stabilization of MYC exerted by PIM2 might explain partly the strong synergism between these 2 oncogenes in tumorigenesis. Regulates cap-dependent protein translation in a mammalian target of rapamycin complex 1 (mTORC1)-independent manner and in parallel to the PI3K-Akt pathway. Mediates survival signaling through phosphorylation of BAD, which induces release of the anti-apoptotic protein Bcl-X(L)/BCL2L1. Promotes cell survival in response to a variety of proliferative signals via positive regulation of the I-kappa-B kinase/NF-kappa-B cascade; this process requires phosphorylation of MAP3K8/COT. Promotes growth factor-independent proliferation by phosphorylation of cell cycle factors such as CDKN1A and CDKN1B. Involved in the positive regulation of chondrocyte survival and autophagy in the epiphyseal growth plate.

**Tissue Location**

Highly expressed in hematopoietic tissues, in leukemic and lymphoma cell lines, testis, small intestine, colon and colorectal adenocarcinoma cells. Weakly expressed in normal liver, but highly expressed in hepatocellular carcinoma tissues

**PIM2 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)