

PIM1 Blocking Peptide

Catalog # PBV10352b

Specification

PIM1 Blocking Peptide - Product Information

Primary Accession P26794
Gene ID 24649
Calculated MW 35631

PIM1 Blocking Peptide - Additional Information

Gene ID 24649

Application & Usage The peptide is used for blocking the

antibody activity of PIM1. It usually blocks the antibody activity completely in Western blot analysis by incubating the peptide with equal volume of antibody for

30-60 minutes at 37°C.

Other Names

Serine/threonine-protein kinase pim-1, 2.7.11.1, Pim1, Pim-1

Target/Specificity

PIM₁

Formulation

 $50~\mu g$ (0.5 mg/ml) in phosphate buffered saline (PBS), pH 7.2, containing 50% glycerol, 1% BSA and 0.02% thimerosal.

Reconstitution & Storage

-20 °C

Background Descriptions

Precautions

PIM1 Blocking Peptide is for research use only and not for use in diagnostic or therapeutic procedures.

PIM1 Blocking Peptide - Protein Information

Name Pim1

Synonyms Pim-1

Function

Proto-oncogene with serine/threonine kinase activity involved in cell survival and cell proliferation and thus providing a selective advantage in tumorigenesis. Exerts its oncogenic activity through:



the regulation of MYC transcriptional activity, the regulation of cell cycle progression and by phosphorylation and inhibition of proapoptotic proteins (BAD, MAP3K5, FOXO3) (By similarity). Phosphorylation of MYC leads to an increase of MYC protein stability and thereby an increase of transcriptional activity. The stabilization of MYC exerted by PIM1 might explain partly the strong synergism between these two oncogenes in tumorigenesis. Mediates survival signaling through phosphorylation of BAD, which induces release of the anti-apoptotic protein Bcl- X(L)/BCL2L1 (By similarity). Phosphorylation of MAP3K5, another proapoptotic protein, by PIM1, significantly decreases MAP3K5 kinase activity and inhibits MAP3K5-mediated phosphorylation of JNK and JNK/p38MAPK subsequently reducing caspase-3 activation and cell apoptosis. Stimulates cell cycle progression at the G1-S and G2-M transitions by phosphorylation of CDC25A and CDC25C. Phosphorylation of CDKN1A, a regulator of cell cycle progression at G1, results in the relocation of CDKN1A to the cytoplasm and enhanced CDKN1A protein stability. Promotes cell cycle progression and tumorigenesis by down-regulating expression of a regulator of cell cycle progression, CDKN1B, at both transcriptional and post-translational levels. Phosphorylation of CDKN1B, induces 14-3-3 proteins binding, nuclear export and proteasome-dependent degradation. May affect the structure or silencing of chromatin by phosphorylating HP1 gamma/CBX3 (By similarity). Acts also as a regulator of homing and migration of bone marrow cells involving functional interaction with the CXCL12-CXCR4 signaling axis (By similarity). Acts as a positive regulator of mTORC1 signaling by mediating phosphorylation and inhibition of DEPDC5 component of the GATOR1 complex. Acts as a negative regulator of innate immunity by mediating phosphorylation and inactivation of GBP1 in absence of infection: phosphorylation of GBP1 induces interaction with 14-3-3 protein sigma (SFN) and retention in the cytosol (By similarity). Also phosphorylates and activates the ATP-binding cassette transporter ABCG2, allowing resistance to drugs through their excretion from cells (By similarity). Promotes brown adipocyte differentiation (By similarity).

Cellular Location Cytoplasm. Nucleus. Cell membrane

PIM1 Blocking Peptide - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

PIM1 Blocking Peptide - Images