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Mouse Pim2 Antibody (N-term) Blocking Peptide

Synthetic peptide
Catalog # BP14455a

Specification

Mouse Pim2 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q62070

Mouse Pim2 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 18715

Other Names
Serine/threonine-protein kinase pim-2, Pim2, Pim-2

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.

Mouse Pim2 Antibody (N-term) Blocking Peptide - Protein Information

Name Pim2

Synonyms Pim-2

Function
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 of 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

Widely expressed, with highest expression in spleen, thymus and brain. Expressed in epiphyseal chondrocytes

Mouse Pim2 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

Mouse Pim2 Antibody (N-term) Blocking Peptide - Images

Mouse Pim2 Antibody (N-term) Blocking Peptide - Background

Promotes cell survival in response to a variety of proliferative signals via positive regulation of the I-kappaB kinase/NF-kappaB cascade; this process requires phosphorylation of MAP3K8/COT. Prevents apoptosis induced by growth factor withdrawal via inhibition of caspase-3 activation, and via phosphorylation of pro-apoptotic proteins. Inhibits BAD-induced cell death via phosphorylation of BAD; isoform 1 is less active in this respect. PIM2-mediated cell survival is glucose-dependent but independent of several AKT regulators such as PI3K, HSP-90 and TOR, indicating that PIM2 and PI3K/AKT/TOR function via distinct pathways. Involved in the positive regulation of chondrocyte survival and autophagy in the epiphyseal growth plate.

Mouse Pim2 Antibody (N-term) Blocking Peptide - References

Agrawal-Singh, S., et al. Blood 115(22):4507-4516(2010)Lin, Y.W., et al. Blood 115(4):824-833(2010)Narayana, Y., et al. Mol. Immunol. 46(15):2947-2954(2009)Grundler, R., et al. J. Exp. Med. 206(9):1957-1970(2009)Derudder, E., et al. Nat. Immunol. 10(6):647-654(2009)