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PIM1 / Pim-1 Antibody (clone 2C9)

Mouse Monoclonal Antibody
Catalog # ALS14123

Specification

PIM1 / Pim-1 Antibody (clone 2C9) - Product Information

Application	**IHC**
Primary Accession	P11309
Reactivity	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Calculated MW	**45kDa KDa**

PIM1 / Pim-1 Antibody (clone 2C9) - Additional Information

Gene ID 5292

Other Names
Serine/threonine-protein kinase pim-1, 2.7.11.1, PIM1

Target/Specificity
Human PIM1

Reconstitution & Storage
Store at -20°C or lower. Aliquot to avoid repeated freezing and thawing.

Precautions
PIM1 / Pim-1 Antibody (clone 2C9) is for research use only and not for use in diagnostic or therapeutic procedures.

PIM1 / Pim-1 Antibody (clone 2C9) - Protein Information

Name PIM1

Function
Proto-oncogene with serine/threonine kinase activity involved in cell survival and cell proliferation and thus providing a selective advantage in tumorigenesis (PubMed:15528381, PubMed:1825810, PubMed:31548394). 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) (PubMed:18593906). Phosphorylation of MYC leads to an increase of MYC protein stability and thereby an increase of transcriptional activity (By similarity). The stabilization of MYC exerted by PIM1 might explain partly the strong synergism between these two oncogenes in tumorigenesis (By similarity). 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 (PubMed:19749799). Stimulates cell cycle progression at the G1-S and G2-M transitions by phosphorylation of CDC25A and CDC25C (PubMed:16356754). 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 (PubMed:12431783). 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 (PubMed:18593906). Phosphorylation of CDKN1B, induces 14-3-3 proteins binding, nuclear export and proteasome-dependent degradation (PubMed:18593906). May affect the structure or silencing of chromatin by phosphorylating HP1 gamma/CBX3 (PubMed:10664448). 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 (PubMed:31548394). 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 (PubMed:37797010). Also phosphorylates and activates the ATP-binding cassette transporter ABCG2, allowing resistance to drugs through their excretion from cells (PubMed:18056989). Promotes brown adipocyte differentiation (By similarity).

Cellular Location
[Isoform 1]: Cytoplasm. Nucleus.

Tissue Location
Expressed primarily in cells of the hematopoietic and germline lineages. Isoform 1 and isoform 2 are both expressed in prostate cancer cell lines.

Volume
50 µl

PIM1 / Pim-1 Antibody (clone 2C9) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

PIM1 / Pim-1 Antibody (clone 2C9) - Images

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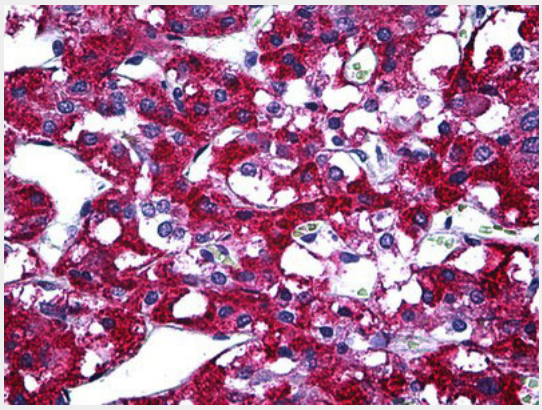

Anti-PIM1 antibody IHC of human adrenal.

PIM1 / Pim-1 Antibody (clone 2C9) - Background

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). 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. Phosphorylation of MAP3K5, an other 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. Promote 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. Acts also as a regulator of homing and migration of bone marrow cells involving functional interaction with the CXCL12-CXCR4 signaling axis.

PIM1 / Pim-1 Antibody (clone 2C9) - References

Reeves R.,et al.Gene 90:303-307(1990).
Zakut-Houri R.,et al.Gene 54:105-111(1987).
Domen J.,et al.Oncogene Res. 1:103-112(1987).
Meeker T.C.,et al.J. Cell. Biochem. 35:105-112(1987).
Xie Y.,et al.Oncogene 25:70-78(2006).