

**PIM1 / Pim-1 Antibody (clone 3F5)**  
**Mouse Monoclonal Antibody**  
**Catalog # ALS14127****Specification**

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**PIM1 / Pim-1 Antibody (clone 3F5) - Product Information**

Application	IF, IHC
Primary Accession	<a href="#">P11309</a>
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Calculated MW	45kDa KDa

**PIM1 / Pim-1 Antibody (clone 3F5) - Additional Information****Gene ID** 5292**Other Names**

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

**Target/Specificity**

Human PIM1

**Reconstitution & Storage**

Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles.

**Precautions**

PIM1 / Pim-1 Antibody (clone 3F5) is for research use only and not for use in diagnostic or therapeutic procedures.

**PIM1 / Pim-1 Antibody (clone 3F5) - 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:<a href="http://www.uniprot.org/citations/15528381" target="\_blank">15528381</a>, PubMed:<a href="http://www.uniprot.org/citations/1825810" target="\_blank">1825810</a>, PubMed:<a href="http://www.uniprot.org/citations/31548394" target="\_blank">31548394</a>). 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:<a href="http://www.uniprot.org/citations/18593906" target="\_blank">18593906</a>). 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:<a href="http://www.uniprot.org/citations/19749799" target="\_blank">19749799</a>). Stimulates cell cycle progression at the G1-S and G2-M transitions by phosphorylation of CDC25A and CDC25C (PubMed:<a href="http://www.uniprot.org/citations/16356754" target="\_blank">16356754</a>). 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:<a href="http://www.uniprot.org/citations/12431783" target="\_blank">12431783</a>). 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:<a href="http://www.uniprot.org/citations/18593906" target="\_blank">18593906</a>). Phosphorylation of CDKN1B, induces 14-3-3 proteins binding, nuclear export and proteasome-dependent degradation (PubMed:<a href="http://www.uniprot.org/citations/18593906" target="\_blank">18593906</a>). May affect the structure or silencing of chromatin by phosphorylating HP1 gamma/CBX3 (PubMed:<a href="http://www.uniprot.org/citations/10664448" target="\_blank">10664448</a>). 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:<a href="http://www.uniprot.org/citations/31548394" target="\_blank">31548394</a>). 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:<a href="http://www.uniprot.org/citations/37797010" target="\_blank">37797010</a>). Also phosphorylates and activates the ATP-binding cassette transporter ABCG2, allowing resistance to drugs through their excretion from cells (PubMed:<a href="http://www.uniprot.org/citations/18056989" target="\_blank">18056989</a>). 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.

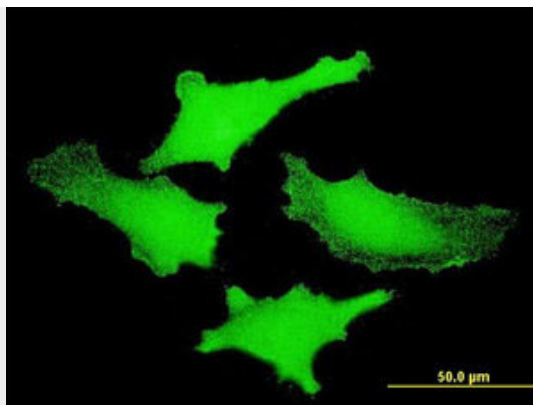
### **PIM1 / Pim-1 Antibody (clone 3F5) - Protocols**

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

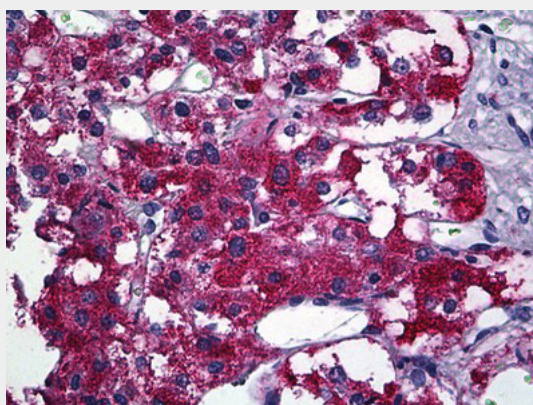
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### **PIM1 / Pim-1 Antibody (clone 3F5) - Images**





Immunofluorescence of monoclonal antibody to PIM1 on HeLa cell. [antibody concentration 10 ug/ml].



Anti-PIM1 antibody IHC of human adrenal.

### **PIM1 / Pim-1 Antibody (clone 3F5) - Background**

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### **PIM1 / Pim-1 Antibody (clone 3F5) - 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).