

Product name:	PIM2 (1717) Rabbit Monoclonal Antibody
Cat number:	MABN16151
Conjugate:	Unconjugated
Size:	100µL
Clone:	Monoclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	A synthetic peptide of human PIM2
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:1000-1:5000,IP 1:10-1:100
Molecular Weight:	34kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% New type preservative N and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

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. 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.