

Product datasheet for TL501643

Pim1 Mouse shRNA Plasmid (Locus ID 18712)

Product data:

Product Type: shRNA Plasmids

Product Name: Pim1 Mouse shRNA Plasmid (Locus ID 18712)

Locus ID: 18712 Synonyms: Pim-1

Vector:pGFP-C-shLenti (TR30023)E. coli Selection:Chloramphenicol (34 ug/ml)

Mammalian Cell Puromycin

Selection:

Format: Lentiviral plasmids

Components: Pim1 - Mouse, 4 unique 29mer shRNA constructs in lentiviral GFP vector(Gene ID = 18712).

5µg purified plasmid DNA per construct

29-mer scrambled shRNA cassette in pGFP-C-shLenti Vector, TR30021, included for free.

RefSeq: <u>BC042885</u>, <u>BC053019</u>, <u>BC055316</u>, <u>NM 008842</u>, <u>NM 008842.1</u>, <u>NM 008842.2</u>, <u>NM 008842.3</u>,

NM 001364913, NM 008842.4

UniProt ID: P06803

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Summary:

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 antiapoptotic protein Bcl-X(L)/BCL2L1. Phosphorylation of MAP3K5, an other proapoptotic protein, by PIM1, significantly decreases MAP3K5 kinase activity and inhibits MAP3K5mediated 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 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 (By similarity).[UniProtKB/Swiss-Prot Function]

shRNA Design:

Performance Guaranteed: These shRNA constructs were designed against multiple splice variants at this gene locus. To be certain that your variant of interest is targeted, please contact <u>techsupport@origene.com</u>. If you need a special design or shRNA sequence, please utilize our <u>custom shRNA service</u>.

OriGene guarantees that the sequences in the shRNA expression cassettes are verified to correspond to the target gene with 100% identity. One of the four constructs at minimum are guaranteed to produce 70% or more gene expression knock-down provided a minimum transfection efficiency of 80% is achieved. Western Blot data is recommended over qPCR to evaluate the silencing effect of the shRNA constructs 72 hrs post transfection. To properly assess knockdown, the gene expression level from the included scramble control vector must be used in comparison with the target-specific shRNA transfected samples.

For non-conforming shRNA, requests for replacement product must be made within ninety (90) days from the date of delivery of the shRNA kit. To arrange for a free replacement with newly designed constructs, please contact Technical Services at techsupport@origene.com. Please provide your data indicating the transfection efficiency and measurement of gene expression knockdown compared to the scrambled shRNA control (Western Blot data preferred).