

## Product datasheet for MR222342L1V

## OriGene Technologies, Inc.

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## Pim2 (NM 138606) Mouse Tagged ORF Clone Lentiviral Particle

**Product data:** 

**Product Type:** Lentiviral Particles

**Product Name:** Pim2 (NM\_138606) Mouse Tagged ORF Clone Lentiviral Particle

Symbol:

DXCch3: Pim-2 Synonyms:

**Mammalian Cell** 

Selection:

None

pLenti-C-Myc-DDK (PS100064) Vector:

Myc-DDK Tag: NM 138606 ACCN:

**ORF Size:** 1110 bp

**ORF Nucleotide** 

OTI Disclaimer:

Sequence:

The ORF insert of this clone is exactly the same as(MR222342).

The molecular sequence of this clone aligns with the gene accession number as a point of reference only. However, individual transcript sequences of the same gene can differ through

naturally occurring variations (e.g. polymorphisms), each with its own valid existence. This clone is substantially in agreement with the reference, but a complete review of all prevailing

variants is recommended prior to use. More info

**OTI Annotation:** This clone was engineered to express the complete ORF with an expression tag. Expression

varies depending on the nature of the gene.

RefSeq: NM 138606.2, NP 613072.1

RefSeq Size: 2054 bp RefSeq ORF: 1113 bp Locus ID: 18715

**UniProt ID:** Q62070

Cytogenetics: X 3.55 cM



## **Gene Summary:**

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 capdependent 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.[UniProtKB/Swiss-Prot Function]