

## Product datasheet for **MR202008L3V**

### **Pttg1 (BC023324) Mouse Tagged ORF Clone Lentiviral Particle**

#### **Product data:**

Product Type:	Lentiviral Particles
Product Name:	Pttg1 (BC023324) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Pttg1
Synonyms:	PTTG
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-Myc-DDK-P2A-Puro (PS100092)
Tag:	Myc-DDK
ACCN:	BC023324
ORF Size:	597 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR202008).
OTI Disclaimer:	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. <a href="#">More info</a>
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:	<a href="#">BC023324</a> , <a href="#">AAH23324</a>
RefSeq Size:	746 bp
RefSeq ORF:	599 bp
Locus ID:	30939
Cytogenetics:	11 B1.1



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**Gene Summary:**

Regulatory protein, which plays a central role in chromosome stability, in the p53/TP53 pathway, and DNA repair. Probably acts by blocking the action of key proteins. During the mitosis, it blocks Separase/ESPL1 function, preventing the proteolysis of the cohesin complex and the subsequent segregation of the chromosomes. At the onset of anaphase, it is ubiquitinated, conducting to its destruction and to the liberation of ESPL1. Its function is however not limited to a blocking activity, since it is required to activate ESPL1. Negatively regulates the transcriptional activity and related apoptosis activity of p53/TP53. The negative regulation of p53/TP53 may explain the strong transforming capability of the protein when it is overexpressed. May also play a role in DNA repair via its interaction with Ku, possibly by connecting DNA damage-response pathways with sister chromatid separation (By similarity). [UniProtKB/Swiss-Prot Function]