

## Product datasheet for **MR226330L4V**

### Prkg1 (NM\_001013833) Mouse Tagged ORF Clone Lentiviral Particle

#### Product data:

Product Type:	Lentiviral Particles
Product Name:	Prkg1 (NM_001013833) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Prkg1
Synonyms:	AW125416; CGKI; Gm19690; Prkg1b; Prkgr1b
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_001013833
ORF Size:	2013 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR226330).
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="#">NM_001013833.3</a> , <a href="#">NP_001013855.1</a>
RefSeq Size:	3914 bp
RefSeq ORF:	2016 bp
Locus ID:	19091
UniProt ID:	<a href="#">P0C605</a>
Cytogenetics:	19 C1



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**Gene Summary:**

Serine/threonine protein kinase that acts as key mediator of the nitric oxide (NO)/cGMP signaling pathway. GMP binding activates PRKG1, which phosphorylates serines and threonines on many cellular proteins. Numerous protein targets for PRKG1 phosphorylation are implicated in modulating cellular calcium, but the contribution of each of these targets may vary substantially among cell types. Proteins that are phosphorylated by PRKG1 regulate platelet activation and adhesion, smooth muscle contraction, cardiac function, gene expression, feedback of the NO-signaling pathway, and other processes involved in several aspects of the CNS like axon guidance, hippocampal and cerebellar learning, circadian rhythm and nociception. Smooth muscle relaxation is mediated through lowering of intracellular free calcium, by desensitization of contractile proteins to calcium, and by decrease in the contractile state of smooth muscle or in platelet activation. Regulates intracellular calcium levels via several pathways: phosphorylates MRV11/IRAG and inhibits IP3-induced Ca(2+) release from intracellular stores, phosphorylation of KCNMA1 (BKCa) channels decreases intracellular Ca(2+) levels, which leads to increased opening of this channel. PRKG1 phosphorylates the canonical transient receptor potential channel (TRPC) family which inactivates the associated inward calcium current. Another mode of action of NO/cGMP/PKG1 signaling involves PKGI-mediated inactivation of the Ras homolog gene family member A (RhoA). Phosphorylation of RHOA by PRKG1 blocks the action of this protein in myriad processes: regulation of RHOA translocation; decreasing contraction; controlling vesicle trafficking, reduction of myosin light chain phosphorylation resulting in vasorelaxation. Activation of PRKG1 by NO signaling alters also gene expression in a number of tissues. In smooth muscle cells, increased cGMP and PRKG1 activity influence expression of smooth muscle-specific contractile proteins, levels of proteins in the NO/cGMP signaling pathway, down-regulation of the matrix proteins osteopontin and thrombospondin-1 to limit smooth muscle cell migration and phenotype. Regulates vasodilator-stimulated phosphoprotein (VASP) functions in platelets and smooth muscle.[UniProtKB/Swiss-Prot Function]