

## Product datasheet for **MR227529L4V**

### **Cdkn1a (NM\_007669) Mouse Tagged ORF Clone Lentiviral Particle**

#### **Product data:**

Product Type:	Lentiviral Particles
Product Name:	Cdkn1a (NM_007669) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Cdkn1a
Synonyms:	CAP; CAP20; CDK; CDKI; Cdkn; Cdkn1; CI; CIP1; mda; mda6; P2; P21; p21C; p21Cip1; p21W; p21WAF; SD; SDI1; Waf; Waf1
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_007669
ORF Size:	480 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR227529).
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_007669.4</a>
RefSeq Size:	1910 bp
RefSeq ORF:	480 bp
Locus ID:	12575
UniProt ID:	<a href="#">P39689</a>
Cytogenetics:	17 15.12 cM



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**Gene Summary:**

This gene encodes a potent cyclin-dependent kinase inhibitor. The encoded protein binds to and inhibits the activity of cyclin-cyclin-dependent kinase2 or cyclin-dependent kinase4 complexes, and thus functions as a regulator of cell cycle progression at the G1 phase. The expression of this gene is tightly controlled by the tumor suppressor protein p53, through which this protein mediates the p53-dependent cell cycle G1 phase arrest in response to a variety of stress stimuli. This protein can interact with proliferating cell nuclear antigen, a DNA polymerase accessory factor, and plays a regulatory role in S phase DNA replication and DNA damage repair. This protein was reported to be specifically cleaved by CASP3-like caspases, which thus leads to a dramatic activation of cyclin-dependent kinase2, and may be instrumental in the execution of apoptosis following caspase activation. Mice that lack this gene have the ability to regenerate damaged or missing tissue. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Sep 2015]