

# Product datasheet for TL500327V

## Cdk1 Mouse shRNA Lentiviral Particle (Locus ID 12534)

### **Product data:**

Product Type:	shRNA Lentiviral Particles
Product Name:	Cdk1 Mouse shRNA Lentiviral Particle (Locus ID 12534)
Locus ID:	12534
Synonyms:	Cdc2; Cdc2a; p34
Vector:	pGFP-C-shLenti (TR30023)
Format:	Lentiviral particles
Components:	Cdk1 - Mouse shRNA lentiviral particles (4 unique 29mer target-specific shRNA, 1 scramble control), 0.5 ml each, >10^7 TU/ml.
RefSeq:	BC024396, NM 007659, NM 007659.1, NM 007659.2, NM 007659.3, BC005614, NM 007659.4
UniProt ID:	<u>P11440</u>

### OriGene Technologies, Inc.

9620 Medical Center Drive, Ste 200 Rockville, MD 20850, US Phone: +1-888-267-4436 https://www.origene.com techsupport@origene.com EU: info-de@origene.com CN: techsupport@origene.cn



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Summary:

Plays a key role in the control of the eukaryotic cell cycle by modulating the centrosome cycle as well as mitotic onset; promotes G2-M transition, and regulates G1 progress and G1-S transition via association with multiple interphase cyclins. Required in higher cells for entry into S-phase and mitosis. Phosphorylates PARVA/actopaxin, APC, AMPH, APC, BARD1, BclxL/BCL2L1, BRCA2, CALD1, CASP8, CDC7, CDC20, CDC25A, CDC25C, CC2D1A, CENPA, CSNK2 proteins/CKII, FZR1/CDH1, CDK7, CEBPB, CHAMP1, DMD/dystrophin, EEF1 proteins/EF-1, EZH2, KIF11/EG5, EGFR, FANCG, FOS, GFAP, GOLGA2/GM130, GRASP1, UBE2A/hHR6A, HIST1H1 proteins/histone H1, HMGA1, HIVEP3/KRC, LMNA, LMNB, LMNC, LBR, LATS1, MAP1B, MAP4, MARCKS, MCM2, MCM4, MKLP1, MYB, NEFH, NFIC, NPC/nuclear pore complex, PITPNM1/NIR2, NPM1, NCL, NUCKS1, NPM1/numatrin, ORC1, PRKAR2A, EEF1E1/p18, EIF3F/p47, p53/TP53, NONO/p54NRB, PAPOLA, PLEC/plectin, RB1, UL40/R2, RAB4A, RAP1GAP, RCC1, RPS6KB1/S6K1, KHDRBS1/SAM68, ESPL1, SKI, BIRC5/survivin, STIP1, TEX14, betatubulins, MAPT/TAU, NEDD1, VIM/vimentin, TK1, FOXO1, RUNX1/AML1, SAMHD1, SIRT2 and RUNX2. CDK1/CDC2-cyclin-B controls pronuclear union in interphase fertilized eggs. Essential for early stages of embryonic development. During G2 and early mitosis, CDC25A/B/Cmediated dephosphorylation activates CDK1/cyclin complexes which phosphorylate several substrates that trigger at least centrosome separation, Golgi dynamics, nuclear envelope breakdown and chromosome condensation. Once chromosomes are condensed and aligned at the metaphase plate, CDK1 activity is switched off by WEE1- and PKMYT1-mediated phosphorylation to allow sister chromatid separation, chromosome decondensation, reformation of the nuclear envelope and cytokinesis. Inactivated by PKR/EIF2AK2- and WEE1mediated phosphorylation upon DNA damage to stop cell cycle and genome replication at the G2 checkpoint thus facilitating DNA repair. Reactivated after successful DNA repair through WIP1-dependent signaling leading to CDC25A/B/C-mediated dephosphorylation and restoring cell cycle progression. In proliferating cells, CDK1-mediated FOXO1 phosphorylation at the G2-M phase represses FOXO1 interaction with 14-3-3 proteins and thereby promotes FOXO1 nuclear accumulation and transcription factor activity, leading to cell death of postmitotic neurons. The phosphorylation of beta-tubulins regulates microtubule dynamics during mitosis. NEDD1 phosphorylation promotes PLK1-mediated NEDD1 phosphorylation and subsequent targeting of the gamma-tubulin ring complex (gTuRC) to the centrosome, an important step for spindle formation. In addition, CC2D1A phosphorylation regulates CC2D1A spindle pole localization and association with SCC1/RAD21 and centriole cohesion during mitosis. The phosphorylation of Bcl-xL/BCL2L1 after prolongated G2 arrest upon DNA damage triggers apoptosis. In contrast, CASP8 phosphorylation during mitosis prevents its activation by proteolysis and subsequent apoptosis. This phosphorylation occurs in cancer cell lines, as well as in primary breast tissues and lymphocytes. EZH2 phosphorylation promotes H3K27me3 maintenance and epigenetic gene silencing. CALD1 phosphorylation promotes Schwann cell migration during peripheral nerve regeneration. CDK1-cyclin-B complex phosphorylates NCKAP5L and mediates its dissociation from centrosomes during mitosis. Regulates the amplitude of the cyclic expression of the core clock gene ARNTL/BMAL1 by phosphorylating its transcriptional repressor NR1D1, and this phosphorylation is necessary for SCF(FBXW7)-mediated ubiguitination and proteasomal degradation of NR1D1 (By similarity).[UniProtKB/Swiss-Prot Function]

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shRNA Design:	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> .
Performance Guaranteed:	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).

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