

## Product datasheet for **TR305469**

### **p21 (CDKN1A) Human shRNA Plasmid Kit (Locus ID 1026)**

#### **Product data:**

<b>Product Type:</b>	shRNA Plasmids
<b>Product Name:</b>	p21 (CDKN1A) Human shRNA Plasmid Kit (Locus ID 1026)
<b>Locus ID:</b>	1026
<b>Synonyms:</b>	CAP20; CDKN1; CIP1; MDA-6; P21; p21CIP1; SDI1; WAF1
<b>Vector:</b>	pRS (TR20003)
<b>E. coli Selection:</b>	Ampicillin
<b>Mammalian Cell Selection:</b>	Puromycin
<b>Format:</b>	Retroviral plasmids
<b>Components:</b>	CDKN1A - Human, 4 unique 29mer shRNA constructs in retroviral untagged vector(Gene ID = 1026). 5µg purified plasmid DNA per construct 29-mer scrambled shRNA cassette in pRS Vector, TR30012, included for free.
<b>RefSeq:</b>	<a href="#">NM_000389</a> , <a href="#">NM_001220777</a> , <a href="#">NM_001220778</a> , <a href="#">NM_001291549</a> , <a href="#">NM_078467</a> , <a href="#">NR_037150</a> , <a href="#">NR_037151</a> , <a href="#">NR_037152</a> , <a href="#">NM_078467.1</a> , <a href="#">NM_078467.2</a> , <a href="#">NM_000389.1</a> , <a href="#">NM_000389.2</a> , <a href="#">NM_000389.4</a> , <a href="#">NM_001220777.1</a> , <a href="#">NM_001220778.1</a> , <a href="#">NM_001291549.1</a> , <a href="#">BC000312</a> , <a href="#">BC000312.2</a> , <a href="#">BC013967</a> , <a href="#">BC013967.2</a> , <a href="#">BC000275</a> , <a href="#">BC001935</a> , <a href="#">NM_001220777.2</a> , <a href="#">NM_078467.3</a> , <a href="#">NM_001220778.2</a> , <a href="#">NM_001291549.3</a> , <a href="#">NM_000389.5</a>
<b>UniProt ID:</b>	<a href="#">P38936</a>
<b>Summary:</b>	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 G1. 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. Multiple alternatively spliced variants have been found for this gene. [provided by RefSeq, Sep 2015]



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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 [techsupport@origene.com](mailto:techsupport@origene.com). If you need a special design or shRNA sequence, please utilize our [custom shRNA service](#).

**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](mailto: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).