

## **Product datasheet for TL507143**

## OriGene Technologies, Inc.

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## Parp3 Mouse shRNA Plasmid (Locus ID 235587)

**Product data:** 

**Product Type:** shRNA Plasmids

**Product Name:** Parp3 Mouse shRNA Plasmid (Locus ID 235587)

**Locus ID:** 235587

Synonyms: A930002C11Rik; Adprt3; Adprtl3; AW990611; pADPRT-3; PARP-3

Vector: pGFP-C-shLenti (TR30023)

E. coli Selection: Chloramphenicol (34 ug/ml)

Mammalian Cell Pur

Selection:

Puromycin

Format: Lentiviral plasmids

**Components:** Parp3 - Mouse, 4 unique 29mer shRNA constructs in lentiviral GFP vector(Gene ID = 235587).

5µg purified plasmid DNA per construct

29-mer scrambled shRNA cassette in pGFP-C-shLenti Vector, TR30021, included for free.

RefSeq: <u>BC014870</u>, <u>BC058754</u>, <u>NM 001311150</u>, <u>NM 145619</u>, <u>NM 145619.1</u>, <u>NM 145619.2</u>,

NM 145619.3, BC062204

UniProt ID: Q8CFB8



## Summary:

Mono-ADP-ribosyltransferase that mediates mono-ADP-ribosylation of target proteins and plays a key role in the response to DNA damage (PubMed:21270334, PubMed:24598253). Mediates mono-ADP-ribosylation of glutamate, aspartate or lysine residues on target proteins (By similarity). In contrast to PARP1 and PARP2, it is not able to mediate poly-ADPribosylation (By similarity). Associates with a number of DNA repair factors and is involved in the response to exogenous and endogenous DNA strand breaks (PubMed:21270334). Together with APLF, promotes the retention of the LIG4-XRCC4 complex on chromatin and accelerate DNA ligation during non-homologous end-joining (NHEJ) (By similarity). Cooperates with the XRRC6-XRCC5 (Ku70-Ku80) heterodimer to limit end-resection thereby promoting accurate NHEJ (PubMed:24598253). Involved in DNA repair by mediating mono-ADPribosylation of a limited number of acceptor proteins involved in chromatin architecture and in DNA metabolism, such as XRRC5 and XRCC6 (By similarity). ADP-ribosylation follows DNA damage and appears as an obligatory step in a detection/signaling pathway leading to the reparation of DNA strand breaks (By similarity). May link the DNA damage surveillance network to the mitotic fidelity checkpoint (By similarity). In addition to proteins, also able to ADP-ribosylate DNA: mediates DNA mono-ADP-ribosylation of DNA strand break termini via covalent addition of a single ADP-ribose moiety to a 5'- or 3'-terminal phosphate residues in DNA containing multiple strand breaks (By similarity). Acts as a negative regulator of immunoglobulin class switch recombination, probably by controlling the level of AICDA /AID on the chromatin (PubMed:26000965).[UniProtKB/Swiss-Prot Function]

shRNA Design:

Performance Guaranteed: 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 <a href="mailto:techsupport@origene.com">techsupport@origene.com</a>. If you need a special design or shRNA sequence, please utilize our <a href="mailto:custom shRNA service">custom shRNA service</a>.

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).