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

Product datasheet for RR210840L4V

Sarm1 (NM_001105817) Rat Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	Sarm1 (NM_001105817) Rat Tagged ORF Clone Lentiviral Particle
Symbol:	Sarm1
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_001105817
ORF Size:	2172 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RR210840).
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. More info
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:	NM_001105817.1, NP_001099287.1
RefSeq Size:	4039 bp
RefSeq ORF:	2175 bp
Locus ID:	287545
UniProt ID:	D3ZUM2
Cytogenetics:	10q25

View online »

Gene Summary: NAD(+) hydrolase, which plays a key role in axonal degeneration following injury by regulating NAD(+) metabolism. Acts as a negative regulator of MYD88- and TRIF-dependent toll-like receptor signaling pathway by promoting Wallerian degeneration, an injury-induced form of programmed subcellular death which involves degeneration of an axon distal to the injury site. Wallerian degeneration is triggered by NAD(+) depletion: in response to injury, SARM1 is activated and catalyzes cleavage of NAD(+) into ADP-D-ribose (ADPR), cyclic ADPR (cADPR) and nicotinamide; NAD(+) cleavage promoting cytoskeletal degradation and axon destruction. Also able to hydrolyze NADP(+), but not other NAD(+)-related molecules. Can activate neuronal cell death in response to stress. Regulates dendritic arborization through the MAPK4-JNK pathway. Involved in innate immune response: inhibits both TICAM1/TRIF- and MYD88-dependent activation of JUN/AP-1, TRIF-dependent activation of NF-kappa-B and IRF3, and the phosphorylation of MAPK14/p38.[UniProtKB/Swiss-Prot Function]