

## Product datasheet for **TL518383V**

### Rapgef2 Mouse shRNA Lentiviral Particle (Locus ID 76089)

#### Product data:

Product Type:	shRNA Lentiviral Particles
Product Name:	Rapgef2 Mouse shRNA Lentiviral Particle (Locus ID 76089)
Locus ID:	76089
Synonyms:	5830453M24Rik; CNRasGEF; mKIAA0313; nRapGEP; Pdzgef1; RA-GEF-1
Vector:	pGFP-C-shLenti (TR30023)
Format:	Lentiviral particles
Components:	Rapgef2 - Mouse shRNA lentiviral particles (4 unique 29mer target-specific shRNA, 1 scramble control), 0.5 ml each, >10 <sup>7</sup> TU/ml.
RefSeq:	<a href="#">NM_001099624</a> , <a href="#">NM_001310536</a> , <a href="#">NM_001099624.1</a> , <a href="#">NM_001099624.2</a> , <a href="#">NM_001099624.3</a> , <a href="#">BC067056</a>

**Summary:** Functions as a guanine nucleotide exchange factor (GEF), which activates Rap and Ras family of small GTPases by exchanging bound GDP for free GTP in a cAMP-dependent manner. Serves as a link between cell surface receptors and Rap/Ras GTPases in intracellular signaling cascades. Acts also as an effector for Rap1 by direct association with Rap1-GTP thereby leading to the amplification of Rap1-mediated signaling. Shows weak activity on HRAS. It is controversial whether RAPGEF2 binds cAMP and cGMP or not. Its binding to ligand-activated beta-1 adrenergic receptor ADRB1 leads to the Ras activation through the G(s)-alpha signaling pathway. Involved in the cAMP-induced Ras and Erk1/2 signaling pathway that leads to sustained inhibition of long term melanogenesis by reducing dendrite extension and melanin synthesis. Provides also inhibitory signals for cell proliferation of melanoma cells and promotes their apoptosis in a cAMP-independent manner. Regulates cAMP-induced neuriteogenesis by mediating the Rap1/B-Raf/ERK signaling through a pathway that is independent on both PKA and RAPGEF3/RAPGEF4. Involved in neuron migration and in the formation of the major forebrain fiber connections forming the corpus callosum, the anterior commissure and the hippocampal commissure during brain development. Involved in neuronal growth factor (NGF)-induced sustained activation of Rap1 at late endosomes and in brain-derived neurotrophic factor (BDNF)-induced axon outgrowth of hippocampal neurons. Plays a role in the regulation of embryonic blood vessel formation and in the establishment of basal junction integrity and endothelial barrier function. May be involved in the regulation of the vascular endothelial growth factor receptor KDR and cadherin CDH5 expression at allantois endothelial cell-cell junctions.[UniProtKB/Swiss-Prot Function]



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<b>shRNA Design:</b>	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="#">custom shRNA service</a> .
<b>Performance Guaranteed:</b>	<p>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.</p> <p>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 <a href="mailto:techsupport@origene.com">techsupport@origene.com</a>. Please provide your data indicating the transfection efficiency and measurement of gene expression knockdown compared to the scrambled shRNA control (Western Blot data preferred).</p>