

## Product datasheet for **TR512614**

### Cnr1 Mouse shRNA Plasmid (Locus ID 12801)

#### Product data:

Product Type:	shRNA Plasmids
Product Name:	Cnr1 Mouse shRNA Plasmid (Locus ID 12801)
Locus ID:	12801
Synonyms:	CB-R; CB1; CB1R
Vector:	pRS (TR20003)
E. coli Selection:	Ampicillin
Mammalian Cell Selection:	Puromycin
Format:	Retroviral plasmids
Components:	Cnr1 - Mouse, 4 unique 29mer shRNA constructs in retroviral untagged vector(Gene ID = 12801). 5µg purified plasmid DNA per construct 29-mer scrambled shRNA cassette in pRS Vector, TR30012, included for free.
RefSeq:	<a href="#">BC070447</a> , <a href="#">BC079564</a> , <a href="#">NM_007726</a> , <a href="#">NM_001355020</a> , <a href="#">NM_001355021</a> , <a href="#">NM_007726.1</a> , <a href="#">NM_007726.2</a> , <a href="#">NM_007726.3</a> , <a href="#">BC075644</a> , <a href="#">NM_001365881</a> , <a href="#">NM_007726.5</a>
UniProt ID:	<a href="#">P47746</a>
Summary:	G-protein coupled receptor for cannabinoids, including endocannabinoids (eCBs), such as N-arachidonylethanolamide (also called anandamide or AEA) and 2-arachidonoylglycerol (2-AG) (PubMed:9888857, PubMed:22388959). Mediates many cannabinoid-induced effects, acting, among others, on food intake, memory loss, gastrointestinal motility, catalepsy, ambulatory activity, anxiety, chronic pain (PubMed:9888857, PubMed:27828947). Signaling typically involves reduction in cyclic AMP (PubMed:8832654, PubMed:27828947). Signaling typically involves reduction in cyclic AMP (By similarity). In the hypothalamus, may have a dual effect on mitochondrial respiration depending upon the agonist dose and possibly upon the cell type. Increases respiration at low doses, while decreases respiration at high doses (PubMed:25707796, PubMed:27828947). At high doses, CNR1 signal transduction involves G-protein alpha-i protein activation and subsequent inhibition of mitochondrial soluble adenylate cyclase, decrease in cyclic AMP concentration, inhibition of protein kinase A (PKA)-dependent phosphorylation of specific subunits of the mitochondrial electron transport system, including NDUFS2 (PubMed:27828947). In the hypothalamus, inhibits leptin-induced reactive oxygen species (ROS) formation and mediates cannabinoid-induced increase in



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SREBF1 and FASN gene expression (PubMed:25869131). In response to cannabinoids, drives the release of orexigenic beta-endorphin, but not that of melanocyte-stimulating hormone alpha/alpha-MSH, from hypothalamic POMC neurons, hence promoting food intake (PubMed:25707796). In the hippocampus, regulates cellular respiration and energy production in response to cannabinoids. Involved in cannabinoid-dependent depolarization-induced suppression of inhibition (DSI), a process in which depolarization of CA1 postsynaptic pyramidal neurons mobilizes eCBs, which retrogradely activate presynaptic CB1 receptors, transiently decreasing GABAergic inhibitory neurotransmission (PubMed:22388959). Also reduces excitatory synaptic transmission (PubMed:27828947). In superior cervical ganglions and cerebral vascular smooth muscle cells, inhibits voltage-gated Ca(2+) channels in a constitutive, as well as agonist-dependent manner (By similarity). In cerebral vascular smooth muscle cells, cannabinoid-induced inhibition of voltage-gated Ca(2+) channels leads to vasodilation and decreased vascular tone (By similarity). Induces leptin production in adipocytes and reduces LRP2-mediated leptin clearance in the kidney, hence participating in hyperleptinemia (PubMed:22841573). In adipose tissue, CNR1 signaling leads to increased expression of SREBF1, ACACA and FASN genes (PubMed:15864349). In the liver, activation by endocannabinoids leads to increased de novo lipogenesis and reduced fatty acid catabolism, associated with increased expression of SREBF1/SREBP-1, GCK, ACACA, ACACB and FASN genes (PubMed:15864349, PubMed:21987372). May also affect de novo cholesterol synthesis and HDL-cholesteryl ether uptake (PubMed:21987372). Peripherally modulates energy metabolism. In high carbohydrate diet-induced obesity, may decrease the expression of mitochondrial dihydrolipoyl dehydrogenase/DLD in striated muscles, as well as that of selected glucose/ pyruvate metabolic enzymes, hence affecting energy expenditure through mitochondrial metabolism (PubMed:26671069). In response to cannabinoid anandamide, elicits a proinflammatory response in macrophages, which involves NLRP3 inflammasome activation and IL1B and IL18 secretion. In macrophages infiltrating pancreatic islets, this process may participate in the progression of type-2 diabetes and associated loss of pancreatic beta-cells (PubMed:23955712).[UniProtKB/Swiss-Prot Function]

**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).