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Product datasheet for MR207569L2V

Cnr1 (NM_007726) Mouse Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	Cnr1 (NM_007726) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Cnr1
Synonyms:	CB-R; CB1; CB1R
Mammalian Cell Selection:	None
Vector:	pLenti-C-mGFP (PS100071)
Tag:	mGFP
ACCN:	NM_007726
ORF Size:	1422 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR207569).
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_007726.3
RefSeq Size:	5807 bp
RefSeq ORF:	1422 bp
Locus ID:	12801
UniProt ID:	P47746
Cytogenetics:	4 16.28 cM

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Gene Summary:

G-protein coupled receptor for cannabinoids, including endocannabinoids (eCBs), such as N-arachidonoylethanolamide (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 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]