10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

CNR1 Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP21304b

Specification

CNR1 Blocking Peptide (C-term) - Product Information

Primary Accession P21554

CNR1 Blocking Peptide (C-term) - Additional Information

Gene ID 1268

Other Names
Cannabinoid receptor 1, CB-R, CB1, CANN6, CNR1, CNR

Target/Specificity
The synthetic peptide sequence is selected from aa 433-448 of HUMAN CNR1

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

CNR1 Blocking Peptide (C-term) - Protein Information

Name CNR1

Synonyms CNR

Function
G-protein coupled receptor for endogenous cannabinoids (eCBs), including N-arachidonoylethanolamide (also called anandamide or AEA) and 2-arachidonoylglycerol (2-AG), as well as phytocannabinoids, such as delta(9)-tetrahydrocannabinol (THC) (PubMed:15620723, PubMed:27768894, PubMed:27851727). Mediates many cannabinoid-induced effects, acting, among others, on food intake, memory loss, gastrointestinal motility, catalepsy, ambulatory activity, anxiety, chronic pain. Signaling typically involves reduction in cyclic AMP (PubMed:1718258, PubMed:21895628, PubMed:27768894). 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. 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. In the hypothalamus, inhibits leptin-induced reactive oxygen species (ROS) formation and mediates cannabinoid-induced increase in SREBF1 and FASN gene expression. 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. 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. Also reduces excitatory synaptic transmission (By similarity). 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 (PubMed:17895407). 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. In adipose tissue, CNR1 signaling leads to increased expression of SREBF1, ACACA and FASN genes (By similarity). 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. May also affect de novo cholesterol synthesis and HDL-cholesteryl ether uptake. Peripherally modulates energy metabolism (By similarity). 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 (By similarity). In response to cannabinoid anandamide, elicits a pro- inflammatory response in macrophages, which involves NLRP3 inflammasome activation and IL1B and IL18 secretion (By similarity). 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).

Cellular Location
Cell membrane; Multi-pass membrane protein. Membrane raft. Mitochondrion outer membrane {ECO:0000250|UniProtKB:P47746}. Cell projection, axon {ECO:0000250|UniProtKB:P20272}. Presynapse {ECO:0000250|UniProtKB:P20272}. Note=Unexpectedly, in the mitochondria, the C-terminus is located in the mitochondrial intermembrane space, a compartment topologically considered as extracellular. In canonical seven-transmembrane G-protein coupled receptors, the C-terminus is cytosolic (By similarity). Found on presynaptic axon terminals in some GABAergic neurons in the somatosensory cortex (By similarity) {ECO:0000250|UniProtKB:P20272, ECO:0000250|UniProtKB:P47746}

Tissue Location
Widely expressed, with highest levels in fetal and adult brain. Expression levels of isoform 2 and isoform 3 are much lower than those of isoform 1.

CNR1 Blocking Peptide (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

CNR1 Blocking Peptide (C-term) - Images

CNR1 Blocking Peptide (C-term) - Background

Involved in cannabinoid-induced CNS effects. Acts by inhibiting adenylate cyclase. Could be a receptor for anandamide. Inhibits L-type Ca(2+) channel current. Isoform 2 and isoform 3 have altered ligand binding.

CNR1 Blocking Peptide (C-term) - References

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Gerard C.,et al.Biochem. J. 279:129-134(1991).
Shire D.,et al.J. Biol. Chem. 270:3726-3731(1995).
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