

Cannabinoid receptor 1 Antibody

Mouse Monoclonal Antibody [Clone CNR1/14469]

Catalog No	Format	Size
1268-MSM1-P0	Purified Ab with BSA and Azide at 200ug/ml	20 ug
1268-MSM1-P1	Purified Ab with BSA and Azide at 200ug/ml	100 ug
1268-MSM1-P1ABX	Purified Ab WITHOUT BSA or Azide at 1.0mg/ml	100 ug

Applications	Tested Dillution	Note
Immunofluorescence (IF)	1-3ug/ml	

Product Details

Clone	CNR1/14469
Immunogen	Recombinant fragment (around aa 388-472) of human CNR1 protein
Host	Mouse
Clonality	Monoclonal
Isotype / Light Chain	IgG2a / Kappa
Mol. Weight of Antigen	52.86kDa
Cellular Localization	Axon, Cell membrane, Cell projection, Membrane raft, Mitochondrion outer membrane, Presynapse
Species Reactivity	Human
Positive Control	Widely expressed, with highest levels in fetal and adult brain

**Optimal dilution for a specific application should be determined.*

Product Images for Cannabinoid receptor 1 Antibody

Specificity & Comments

G-protein coupled receptor for endogenous cannabinoids (eCBs), including N-arachidonylethanolamide (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, GSK, ACACA, ACACB and FASN genes. May also affect de novo cholesterol synthesis and HDL-cholesterol 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)., Binds both 2-arachidonoylglycerol (2-AG) and anandamide., Only binds 2-arachidonoylglycerol (2-AG) with high affinity. Contrary to its effect on isoform 1, 2-AG behaves as an inverse agonist on isoform 2 in assays measuring GTP binding to membranes., Only binds 2-arachidonoylglycerol (2-AG) with high affinity. Contrary to its effect on isoform 1, 2-AG behaves as an inverse agonist on isoform 3 in assays measuring GTP binding to membranes.

Limitations and Warranty

This antibody is available for research use only and is not approved for use in diagnosis. There are no warranties, expressed or implied, which extend beyond this description. Company is not liable for any personal injury or economic loss resulting from this product.

Supplied As

200ug/ml of Ab purified from Bioreactor Concentrate by Protein G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

Storage and Stability

Antibody with azide - store at 2 to 8 °C. Antibody without azide - store at -20 to -80 °C. Antibody is stable for 24 months. Non-hazardous. No MSDS required.