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Datasheet for ABIN6257095 anti-CNR1 antibody (N-Term)

3 Images



Overview

Quantity:	100 μL
Target:	CNR1
Binding Specificity:	N-Term
Reactivity:	Human, Rat, Mouse
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This CNR1 antibody is un-conjugated
Application:	Western Blotting (WB), ELISA, Immunohistochemistry (IHC), Immunofluorescence (IF), Immunocytochemistry (ICC)

Product Details

Immunogen:	A synthesized peptide derived from human CNR1, corresponding to a region within N-terminal amino acids.
Isotype:	lgG
Specificity:	CNR1 Antibody detects endogenous levels of total CNR1.
Predicted Reactivity:	Bovine,Horse,Sheep,Chicken
Purification:	The antiserum was purified by peptide affinity chromatography using SulfoLink TM Coupling Resin (Thermo Fisher Scientific).

Target Details

Target:	CNR1
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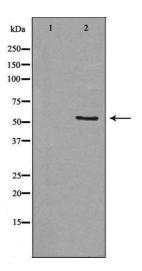
CNR1 (CNR1 Products)
Description: G-protein coupled receptor for endogenous cannabinoids (eCBs), including N-
arachidonoylethanolamide (also called anandamide or AEA) and 2-arachidonoylglycerol (2-AG),
as well as phytocannabinoids, such as delta9-tetrahydrocannabinol (THC) (PubMed:15620723,
PubMed:27768894, 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 Ca2+ channels in a constitutive, as well as agonist-
dependent manner (PubMed:17895407). In cerebral vascular smooth muscle cells,
cannabinoid-induced inhibition of voltage-gated Ca2+ 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

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Target Details

	 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 proinflammatory 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). Gene: CNR1
Molecular Weight:	53 kDa
Gene ID:	1268
UniProt:	P21554
Pathways:	Feeding Behaviour
Application Details	
Application Notes:	WB 1:500-1:1000, IHC: 1:50-1:200, IF/ICC 1:100-1:500, ELISA(peptide) 1:20000-1:40000
Restrictions:	For Research Use only
Handling	
Format:	Liquid
Concentration:	1 mg/mL
Buffer:	Rabbit IgG in phosphate buffered saline , pH 7.4, 150 mM NaCl, 0.02 % sodium azide and 50 % glycerol.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	-20 °C
Storage Comment:	Store at -20 °C. Stable for 12 months from date of receipt.
Expiry Date:	12 months

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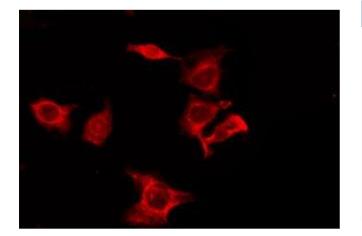
Western Blotting

Image 1. Western blot analysis of extracts from COS-7cells using CNR1 antibody. The lane on the left is treated with the antigen-specific peptide.



Immunohistochemistry

Image 2. ABIN6275980 at 1/100 staining Human brain cancer tissue by IHC-P. The sample was formaldehyde fixed and a heat mediated antigen retrieval step in citrate buffer was performed. The sample was then blocked and incubated with the antibody for 1.5 hours at 22;ãC. An HRP conjugated goat anti-rabbit antibody was used as the secondary



Immunofluorescence (fixed cells)

Image 3. ABIN6275980 staining 293T by IF/ICC. The sample were fixed with PFA and permeabilized in 0.1% Triton X-100,then blocked in 10% serum for 45 minutes at 25jãC. The primary antibody was diluted at 1/200 and incubated with the sample for 1 hour at 37jãC. An Alexa Fluor 594 conjugated goat anti-rabbit IgG (H+L) Ab, diluted at 1/600, was used as the secondary antibod

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