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OPRM1 Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP9130c

Specification

OPRM1 Antibody (Center) Blocking Peptide - Product Information

Primary Accession P35372

OPRM1 Antibody (Center) Blocking Peptide - Additional Information

Gene ID 4988

Other Names
Mu-type opioid receptor, M-OR-1, MOR-1, Mu opiate receptor, Mu opioid receptor, MOP, hMOP, OPRM1, MOR1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP9130c was selected from the Center region of human OPRM1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

OPRM1 Antibody (Center) Blocking Peptide - Protein Information

Name OPRM1

OPRM1 Antibody (Center) Blocking Peptide - Background

OPRM1 is the mu opioid receptor, which is the primary site of action for the most commonly used opioids, including morphine, heroin, fentanyl, and methadone. It is also the primary receptor for endogenous opioid peptides beta-endorphin (see POMC, MIM 176830) and the enkephalins (see PENK, MIM 131330). The OPRM1 receptor is a membrane of the G protein-coupled receptor family (Bond et al., 1998 [PubMed 9689128]). There are at least 3 types of opioid receptors, mu, kappa (OPRK1; MIM 165196), and delta, each with a distinct pharmacologic profile.

OPRM1 Antibody (Center) Blocking Peptide - References

Bare,L.A., et.al., FEBS Lett. 354 (2), 213-216 (1994)Wang,J.B., et.al., FEBS Lett. 338 (2), 217-222 (1994)

Synonyms MOR1

Function
Receptor for endogenous opioids such as beta-endorphin and endomorphin. Receptor for natural and synthetic opioids including morphine, heroin, DAMGO, fentanyl, etorphine, buprenorphin and methadone (PubMed:7905839, PubMed:7957926, PubMed:7891175, PubMed:12589820, PubMed:9689128). Agonist binding to the receptor induces coupling to an inactive GDP-bound heterotrimeric G-protein complex and subsequent exchange of GDP for GTP in the G-protein alpha subunit leading to dissociation of the G-protein complex with the free GTP-bound G-protein alpha and the G-protein beta-gamma dimer activating downstream cellular effectors (PubMed:7905839). The agonist- and cell type-specific activity is predominantly coupled to pertussis toxin-sensitive G(i) and G(o) G alpha proteins, GNAI1, GNAI2, GNAI3 and GNAO1 isoforms Alpha-1 and Alpha-2, and to a lesser extent to pertussis toxin-insensitive G alpha proteins GNAZ and GNA15 (PubMed:12068084). They mediate an array of downstream cellular responses, including inhibition of adenylate cyclase activity and both N-type and L-type calcium channels, activation of inward rectifying potassium channels, mitogen-activated protein kinase (MAPK), phospholipase C (PLC), phosphoinositide/protein kinase (PKC), phosphoinositide 3-kinase (PI3K) and regulation of NF-kappa-B. Also couples to adenylate cyclase stimulatory G alpha proteins. The selective temporal coupling to G- proteins and subsequent signaling can be regulated by RGSZ proteins, such as

RGS9, RGS17 and RGS4. Phosphorylation by members of the GPRK subfamily of Ser/Thr protein kinases and association with beta-arrestins is involved in short-term receptor desensitization. Beta- arrestins associate with the GPRK-phosphorylated receptor and uncouple it from the G-protein thus terminating signal transduction. The phosphorylated receptor is internalized through endocytosis via clathrin-coated pits which involves beta-arrestins. The activation of the ERK pathway occurs either in a G-protein-dependent or a beta-arrestin-dependent manner and is regulated by agonist-specific receptor phosphorylation. Acts as a class A G-protein coupled receptor (GPCR) which dissociates from beta-arrestin at or near the plasma membrane and undergoes rapid recycling. Receptor down-regulation pathways are varying with the agonist and occur dependent or independent of G- protein coupling. Endogenous ligands induce rapid desensitization, endocytosis and recycling whereas morphine induces only low desensitization and endocytosis. Heterooligomerization with other GPCRs can modulate agonist binding, signaling and trafficking properties. Involved in neurogenesis. Isoform 12 couples to GNAS and is proposed to be involved in excitatory effects (PubMed:20525224). Isoform 16 and isoform 17 do not bind agonists but may act through oligomerization with binding-competent OPRM1 isoforms and reduce their ligand binding activity (PubMed:16580639).

Cellular Location
Cell membrane; Multi-pass membrane protein. Cell projection, axon {ECO:0000250|UniProtKB:P97266}. Perikaryon {ECO:0000250|UniProtKB:P97266}. Cell projection, dendrite {ECO:0000250|UniProtKB:P97266}. Endosome {ECO:0000250|UniProtKB:P97266}. Note=Is rapidly internalized after agonist binding. {ECO:0000250|UniProtKB:P97266}

Tissue Location
Expressed in brain. Isoform 16 and isoform

17 are detected in brain.

OPRM1 Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides