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OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain)

Rabbit Polyclonal Antibody
Catalog # ALS10074

Specification

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - Product Information

Application	**IHC-P**
Primary Accession	P35372
Reactivity	**Human, Monkey**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**45kDa KDa**
Dilution	**IHC-P~~N/A**

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - 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
Human OPRM1 / Mu Opioid Receptor. BLAST analysis of the peptide immunogen showed no homology with other human proteins.

Reconstitution & Storage
Long term: -70°C; Short term: +4°C

Precautions
OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) is for research use only and not for use in diagnostic or therapeutic procedures.

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - Protein Information

Name OPRM1

Synonyms MOR1

Function
Receptor for endogenous opioids such as beta-endorphin and endomorphin (PubMed:10529478, PubMed:12589820, PubMed:7891175, PubMed:7905839, PubMed:7957926, PubMed:9689128). Receptor for natural and synthetic opioids including morphine, heroin, DAMGO, fentanyl, etorphine,

buprenorphin and methadone (PubMed:10529478, PubMed:10836142, PubMed:12589820, PubMed:19300905, PubMed:7891175, PubMed:7905839, PubMed:7957926, PubMed:9689128). Also activated by enkephalin peptides, such as Met-enkephalin or Met-enkephalin-Arg-Phe, with higher affinity for Met-enkephalin-Arg-Phe (By similarity). 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 (By similarity). Also couples to adenylate cyclase stimulatory G alpha proteins (By similarity). The selective temporal coupling to G- proteins and subsequent signaling can be regulated by RGSZ proteins, such as RGS9, RGS17 and RGS4 (By similarity). Phosphorylation by members of the GPRK subfamily of Ser/Thr protein kinases and association with beta-arrestins is involved in short-term receptor desensitization (By similarity). Beta-arrestins associate with the GPRK-phosphorylated receptor and uncouple it from the G-protein thus terminating signal transduction (By similarity). The phosphorylated receptor is internalized through endocytosis via clathrin-coated pits which involves beta-arrestins (By similarity). 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 (By similarity). 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 (By similarity). Receptor down-regulation pathways are varying with the agonist and occur dependent or independent of G-protein coupling (By similarity). Endogenous ligands induce rapid desensitization, endocytosis and recycling (By similarity). Heterooligomerization with other GPCRs can modulate agonist binding, signaling and trafficking properties (By similarity).

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.

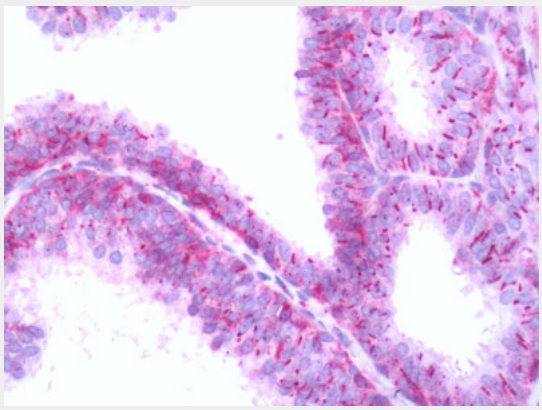
Volume
50 µl

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - Images



Human Seminal Vesicle: Formalin-Fixed, Paraffin-Embedded (FFPE)

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - Background

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. 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. 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 extend to pertussis toxin-insensitive G alpha proteins GNAZ and GNA15. 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. 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.

OPRM1 / Mu Opioid Receptor Antibody (Extracellular Domain) - References

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