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Mu Opioid Receptor (pS375) Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP51408

Product Information

Application	WB
Primary Accession	P35372
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	44779

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
Dilution	WB~~1:1000
Format	0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%
Storage	Store at -20 °C.Stable for 12 months from date of receipt

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.

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.

References

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