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phospho-mu Opioid Receptor (Ser375) Rabbit pAb

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Catalog # AP52191

Product Information

Application	WB
Primary Accession	P33535
Predicted	Rat, Pig, Dog, Human, Mouse, Horse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	44494
Physical State	Liquid
Immunogen	KLH conjugated Synthesised phosphopeptide derived from rat mu Opioid Receptor around the phosphorylation site of Ser375
Epitope Specificity	HP(p-S)TA
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Cell membrane; Multi-pass membrane protein.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	This gene encodes one of three opioid receptors. The mu opioid receptor is the principal target of endogenous opioid peptides and opioid analgesic agents such a s beta-endorphn and enkephalins. The NM_001008503.1:c.118A>G allele had been associated with opioid and alcohol addiction and variations in pain sensitivity but evidence is conflicting. Multiple transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Jun 2012]

Additional Information

Gene ID	25601
Other Names	Mu-type opioid receptor, M-OR-1, MOR-1, Opioid receptor B, Oprm1, Ror-b
Dilution	WB=1:500-2000,ELISA=1:5000-10000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	Oprm1

Synonyms Ror-b

Function Receptor for endogenous opioids such as beta-endorphin and endomorphin (PubMed:11060299, PubMed:15944153, PubMed:16682964, PubMed:17384143, PubMed:17947509, PubMed:1846076, PubMed:18558479, PubMed:21292762, PubMed:7595566, PubMed:7678862, PubMed:8051154, PubMed:8240812, PubMed:8393525, PubMed:9224819, PubMed:9572309). Receptor for natural and synthetic opioids including morphine, heroin, DAMGO, fentanyl, etorphine, buprenorphin and methadone (PubMed:11060299, PubMed:15944153, PubMed:16682964, PubMed:17384143, PubMed:17947509, PubMed:1846076, PubMed:18558479, PubMed:21292762, PubMed:7595566, PubMed:7678862, PubMed:8051154, PubMed:8240812, PubMed:8393525, PubMed:9224819, PubMed:9572309). Also activated by enkephalin peptides, such as Met-enkephalin or Met-enkephalin-Arg-Phe, with higher affinity for Met-enkephalin-Arg-Phe (PubMed:8624732). 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:16682964, PubMed:9224819). 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:9224819, PubMed:9572309). 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 (PubMed:15944153, PubMed:21292762, PubMed:7595566, PubMed:9572309). Also couples to adenylate cyclase stimulatory G alpha proteins (PubMed:7595566). 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 (PubMed:11060299, PubMed:17384143, PubMed:17947509, PubMed:18558479). 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 (PubMed:11278523, PubMed:11896051, PubMed:15944153). 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 (PubMed:11060299, PubMed:17384143, PubMed:17947509, PubMed:18558479). Endogenous ligands induce rapid desensitization, endocytosis and recycling. Heterooligomerization with other GPCRs can modulate agonist binding, signaling and trafficking properties (PubMed:16682964, PubMed:17384143).

Cellular Location Cell membrane; Multi-pass membrane protein {ECO:0000250|UniProtKB:P42866}. 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	Brain. Is expressed in the cerebral cortex, caudate putamen, nucleus accumbens, septal nuclei, thalamus, hippocampus, and habenula. Not detected in cerebellum

Background

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

References

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Wang J.-B.,et al.Proc. Natl. Acad. Sci. U.S.A. 90:10230-10234(1993).
Chen Y.,et al.Mol. Pharmacol. 44:8-12(1993).
Bunzow J.R.,et al.Submitted (SEP-1993) to the EMBL/GenBank/DDBJ databases.
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