ARG51787
anti-Opioid Receptor phospho (Ser375) antibody

Package: 100 µl, 50 µl
Store at: -20°C

Summary

Product Description	Rabbit Polyclonal antibody recognizes Opioid Receptor phospho (Ser375)
Tested Reactivity	Hu, Ms, Rat
Tested Application	WB
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Target Name	Opioid Receptor
Species	Human
Immunogen	Peptide sequence around phosphorylation site of serine 375(H-P-S(p)-T-A) derived from Human Opioid Receptor.
Conjugation	Un-conjugated
Alternate Names	MOP; Mu opiate receptor; OPRM; MOR; LMOR; MOR1; MOR-1; Mu opioid receptor; hMOP; Mu-type opioid receptor; M-OR-1

Application Instructions

Application table

Application	Dilution
WB	1:500 - 1:1000

Application Note: * The dilutions indicate recommended starting dilutions and the optimal dilutions or concentrations should be determined by the scientist.

Properties

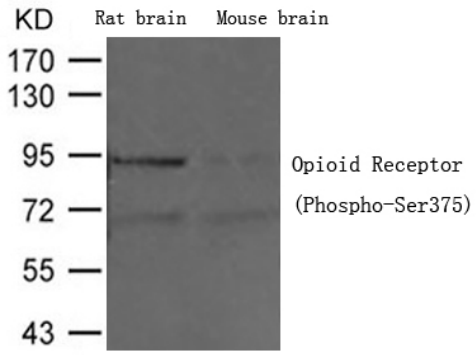
Form	Liquid
Purification	Antibodies were produced by immunizing rabbits with KLH-conjugated synthetic phosphopeptide. Antibodies were purified by affinity-chromatography using epitope-specific phosphopeptide. In addition, non-phospho specific antibodies were removed by chromatogramphy using non-phosphopeptide.
Buffer	PBS (without Mg2+ and Ca2+, pH 7.4), 150mM NaCl, 0.02% Sodium azide and 50% Glycerol.
Preservative	0.02% Sodium azide
Stabilizer	50% Glycerol
Concentration	1 mg/ml
Storage instruction	For continuous use, store undiluted antibody at 2-8°C for up to a week. For long-term storage, aliquot and store at -20°C. Storage in frost free freezers is not recommended. Avoid repeated freeze/thaw cycles. Suggest spin the vial prior to opening. The antibody solution should be gently mixed before use.
Note	For laboratory research only, not for drug, diagnostic or other use.

Bioinformation

Gene Symbol	OPRM1
Gene Full Name	opioid receptor, mu 1
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.
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. 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. [UniProt]
Research Area	Cell Biology and Cellular Response antibody; Neuroscience antibody
Calculated Mw	45 kDa
PTM	Phosphorylated. Differentially phosphorylated in basal and agonist-induced conditions. Agonist-mediated phosphorylation modulates receptor internalization. Phosphorylated by GRK2 in a agonist-dependent manner. Phosphorylation at Tyr-168 requires receptor activation, is dependent on non-receptor protein tyrosine kinase Src and results in a decrease in agonist efficacy by reducing G-protein coupling efficiency.

Phosphorylated on tyrosine residues; the phosphorylation is involved in agonist-induced G-protein-independent receptor down-regulation. Phosphorylation at Ser-377 is involved in G-protein-dependent but not beta-arrestin-dependent activation of the ERK pathway (By similarity).
Ubiquitinated. A basal ubiquitination seems not to be related to degradation. Ubiquitination is increased upon formation of OPRM1:OPRD1 oligomers leading to proteasomal degradation; the ubiquitination is diminished by RTP4.

Images



ARG51787 anti-Opioid Receptor phospho (Ser375) antibody WB image

Western blot: Extracts from Rat brain tissue and Mouse brain tissue stained with ARG51787 anti-Opioid Receptor phospho (Ser375) antibody.