Product Datasheet

Opioid Receptor (Phospho-Ser375) Antibody

Catalog No: #11313

Package Size: #11313-1 50ul #11313-2 100ul



Orders: order@signalwayantibody.com Support: tech@signalwayantibody.com

Description

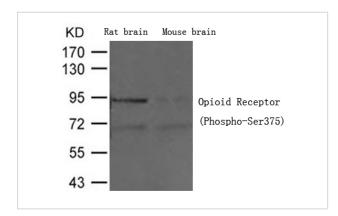
Product Name	Opioid Receptor (Phospho-Ser375) Antibody
Host Species	Rabbit
Clonality	Polyclonal
Purification	Antibodies were produced by immunizing rabbits with synthetic phosphopeptide and KLH conjugates.
	Antibodies were purified by affinity-chromatography using epitope-specific phosphopeptide. Non-phospho
	specific antibodies were removed by chromatogramphy using non-phosphopeptide.
Applications	WB
Species Reactivity	Ms Rt
Specificity	The antibody detects endogenous level of Opioid Receptor only when phosphorylated at serine375.
Immunogen Type	Peptide-KLH
Immunogen Description	Peptide sequence around phosphorylation site of serine 375(H-P-S(p)-T-A) derived from Human Opioid
	Receptor.
Conjugates	Unconjugated
Target Name	Opioid Receptor
Modification	Phospho
Other Names	MOP; MOR; LMOR; MOR1; OPRM
Accession No.	Swiss-Prot: P35372; NCBI Gene ID: 4988; NCBI mRNA: NM_000914.3 ; NCBI Protein: NP_000905.3
SDS-PAGE MW	70-90kd
Concentration	1.0mg/ml
Formulation	Supplied at 1.0mg/mL in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.02%
	sodium azide and 50% glycerol.
Storage	Store at -20°C

Application Details

Predicted MW: 70-90kd

Western blotting: 1:500~1:1000

Images



Western blot analysis of extracts from Rat brain tissue and Mouse brain tissue using Opioid Receptor (Phospho-Ser375) Antibody #11313.

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.

Note: This product is for in vitro research use only and is not intended for use in humans or animals.