磷酸化 μ-型阿片受体抗体

产品货号： mlR10532

英文名称： Phospho-mu Opioid Receptor (Ser377)

中文名称： 磷酸化 μ-型阿片受体抗体

别　　名： Mu Opioid Receptor (phospho S377)(human); Mu Opioid Receptor (phospho S375)(mo, rat); Phospho-mu Opioid Receptor (Ser375)(mo, rat); LMOR; MOR 1; mor; MOR1; Mu opiate receptor; mu type opioid receptor; Mu type opioid receptor MOR 1; muOR; Opioid receptor mu 1; Opioid receptor mu; OPRM; OPRM1; OPRM_HUMAN.

研究领域： 细胞生物　免疫学　神经生物学　信号转导　转录调节因子

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应： Human, Mouse, Rat, Dog, Pig, Cow, Horse,

产品应用： WB=1:500-2000 ELISA=1:500-1000 IHC-P=1:400-800 IHC-F=1:400-800 ICC=1:100-500 IF=1:100-500（石蜡切片需做抗原修复）

not yet tested in other applications.

optimal dilutions/concentrations should be determined by the end user.

分 子 量： 44kDa

细胞定位： 细胞膜

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原： KLH conjugated synthesised phosphopeptide derived from human mu Opioid Receptor around the phosphorylation site of Ser377:HP(p-S)TA

亚　　型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件： Store at -20 ° C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20° C. 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.

PubMed : PubMed

产品介绍 background:

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]

Function:

Receptor for endogenous opioids such as beta-endorphin and endomorphin. 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. Heterooligomerization with other GPCRs can modulate agonist binding, signaling and trafficking properties. Involved in neurogenesis.

Subunit:

Forms homooligomers and heterooligomers with other GPCRs, such as OPRD1, OPRK1, OPRL1, NPFFR2, ADRA2A, SSTR2, CNR1 and CCR5 (probably in dimeric forms). Interacts with PPL; the interaction disrupts agonist-mediated G-protein activation. Interacts (via C-terminus) with DNAJB4 (via C-terminus). Interacts with calmodulin; the interaction inhibits the constitutive activity of OPRM1; it abolishes basal and attenuates agonist-stimulated G-protein coupling. Interacts with FLNA. Interacts with PLD2. Interacts with RANBP9 and WLS. Interacts with GPM6A. Interacts with RTP4. Interacts with SYP and GNAS. Interacts with RGS9, RGS17 and RGS20. Interacts with RGS4. Interacts with PPP1R9B and HINT1.

Subcellular Location:

Cell membrane; Multi-pass membrane protein.

Tissue Specificity:

Brain. Is expressed in the cerebral cortex, caudate putamen, nucleus accumbens, septal nuclei, thalamus, hippocampus, and habenula. Not detected in cerebellum.

Similarity:

Belongs to the G-protein coupled receptor 1 family.

SWISS:

P35372

Gene ID:

4988

Important Note:

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