

GRIA4 / GLUR4 Antibody (Internal)
Goat Polyclonal Antibody
Catalog # ALS12864**Specification**

GRIA4 / GLUR4 Antibody (Internal) - Product Information

Application	WB, IHC-P, E
Primary Accession	P48058
Reactivity	Human, Mouse, Rat, Rabbit, Monkey, Chicken, Horse, Bovine, Dog
Host	Goat
Clonality	Polyclonal
Calculated MW	101kDa KDa
Dilution	WB~~1:1000 IHC-P~~N/A E~~N/A

GRIA4 / GLUR4 Antibody (Internal) - Additional Information**Gene ID** 2893**Other Names**

Glutamate receptor 4, GluR-4, GluR4, AMPA-selective glutamate receptor 4, GluR-D, Glutamate receptor ionotropic, AMPA 4, GluA4, GRIA4, GLUR4

Target/Specificity

Human GRIA4 / GLUR4. This antibody is expected to recognize all reported isoforms (NP_000820.3; NP_001070711.1; NP_001070712.1). Reported variants NP_001070712.1 and NP_001106283.1 represent identical protein.

Reconstitution & Storage

Store at -20°C. Minimize freezing and thawing.

Precautions

GRIA4 / GLUR4 Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

GRIA4 / GLUR4 Antibody (Internal) - Protein Information**Name** GRIA4 {ECO:0000303|PubMed:29220673, ECO:0000312|HGNC:HGNC:4574}**Function**

Ionotropic glutamate receptor that functions as a ligand- gated cation channel, gated by L-glutamate and glutamatergic agonists such as alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA), quisqualic acid, and kainic acid (By similarity). L-glutamate acts as an excitatory neurotransmitter at many synapses in the central nervous system and plays an important role in fast excitatory synaptic transmission (By similarity). Binding of the excitatory neurotransmitter L-glutamate induces a conformation change, leading to

the opening of the cation channel, and thereby converts the chemical signal to an electrical impulse upon entry of monovalent and divalent cations such as sodium and calcium. The receptor then desensitizes rapidly and enters a transient inactive state, characterized by the presence of bound agonist (By similarity). In the presence of CACNG8, shows resensitization which is characterized by a delayed accumulation of current flux upon continued application of L-glutamate (PubMed:21172611).

Cellular Location

Cell membrane {ECO:0000250|UniProtKB:P19493}; Multi-pass membrane protein {ECO:0000250|UniProtKB:P19493} Postsynaptic cell membrane {ECO:0000250|UniProtKB:P19493}; Multi-pass membrane protein {ECO:0000250|UniProtKB:P19493}. Cell projection, dendrite {ECO:0000250|UniProtKB:P19493}. Postsynaptic cell membrane {ECO:0000250|UniProtKB:P42262}; Multi-pass membrane protein {ECO:0000250|UniProtKB:P42262}

GRIA4 / GLUR4 Antibody (Internal) - Protocols

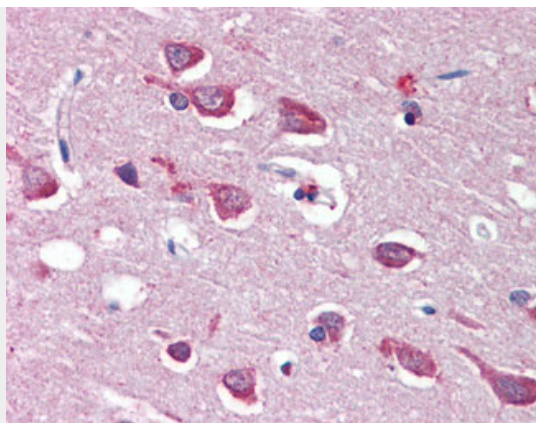
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

GRIA4 / GLUR4 Antibody (Internal) - Images



Antibody (0.3 ug/ml) staining of Human Cerebellum lysate (35 ug protein in RIPA buffer).



Anti-GRIA4 / GLUR4 antibody IHC of human brain, cortex.

GRIA4 / GLUR4 Antibody (Internal) - Background

Receptor for glutamate that functions as ligand-gated ion channel in the central nervous system and plays an important role in excitatory synaptic transmission. L-glutamate acts as an excitatory neurotransmitter at many synapses in the central nervous system. Binding of the excitatory neurotransmitter L- glutamate induces a conformation change, leading to the opening of the cation channel, and thereby converts the chemical signal to an electrical impulse. The receptor then desensitizes rapidly and enters a transient inactive state, characterized by the presence of bound agonist. In the presence of CACNG4 or CACNG7 or CACNG8, shows resensitization which is characterized by a delayed accumulation of current flux upon continued application of glutamate.

GRIA4 / GLUR4 Antibody (Internal) - References

Fletcher E.J.,et al.Recept. Channels 3:21-31(1995).
Taylor T.D.,et al.Nature 440:497-500(2006).
Kato A.S.,et al.Neuron 68:1082-1096(2010).