

GLRB Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14931a**Specification**

GLRB Antibody (N-term) - Product Information

Application	WB,E
Primary Accession	P48167
Other Accession	P20781 , P48168 , O9GJS9 , NP_001159532.1 , NP_000815.1
Reactivity	Human
Predicted	Bovine, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	103-132

GLRB Antibody (N-term) - Additional Information**Gene ID** 2743**Other Names**

Glycine receptor subunit beta, Glycine receptor 58 kDa subunit, GLRB

Target/Specificity

This GLRB antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 103-132 amino acids from the N-terminal region of human GLRB.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

GLRB Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

GLRB Antibody (N-term) - Protein Information**Name** GLRB**Function** Glycine receptors are ligand-gated chloride channels. GLRB does not form ligand-gated

ion channels by itself, but is part of heteromeric ligand-gated chloride channels. Channel opening is triggered by extracellular glycine (PubMed:[8717357](#), PubMed:[15302677](#), PubMed:[16144831](#), PubMed:[22715885](#), PubMed:[25445488](#), PubMed:[11929858](#), PubMed:[23238346](#), PubMed:[34473954](#)). Heteropentameric channels composed of GLRB and GLRA1 are activated by lower glycine levels than homopentameric GLRA1 (PubMed:[8717357](#)). Plays an important role in the down-regulation of neuronal excitability (PubMed:[11929858](#), PubMed:[23238346](#)). Contributes to the generation of inhibitory postsynaptic currents (PubMed:[25445488](#)).

Cellular Location

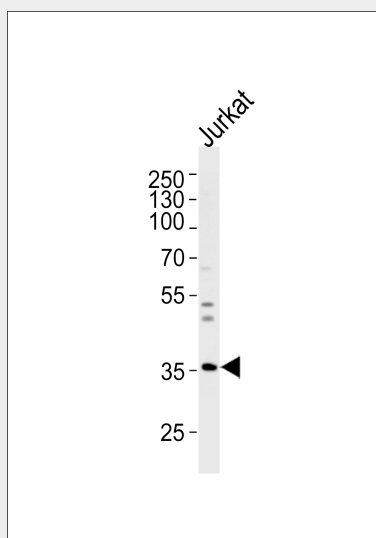
Postsynaptic cell membrane {ECO:0000250|UniProtKB:P48168}; Multi-pass membrane protein {ECO:0000250|UniProtKB:P23415}. Synapse {ECO:0000250|UniProtKB:P48168} Cell projection, dendrite {ECO:0000250|UniProtKB:P48168}. Cell membrane; Multi-pass membrane protein {ECO:0000250|UniProtKB:P23415}. Cytoplasm Note=Retained in the cytoplasm upon heterologous expression by itself Coexpression with GPHN promotes expression at the cell membrane (PubMed:12684523). Coexpression with GLRA1, GLRA2 or GLRA3 promotes expression at the cell membrane. {ECO:0000250|UniProtKB:P20781, ECO:0000269|PubMed:12684523}

GLRB Antibody (N-term) - 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](#)

GLRB Antibody (N-term) - Images



Western blot analysis of lysate from Jurkat cell line, using GLRB Antibody (N-term)(Cat. #AP14931a). AP14931a was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody. Lysate at 35ug per lane.

GLRB Antibody (N-term) - Background

This gene encodes the beta subunit of the glycine receptor, which is a pentamer composed of alpha and beta subunits. The receptor functions as a neurotransmitter-gated ion channel, which produces hyperpolarization via increased chloride conductance due to the binding of glycine to the receptor. Mutations in this gene cause startle disease, also known as hereditary hyperekplexia or congenital stiff-person syndrome, a disease characterized by muscular rigidity. Alternative splicing results in multiple transcript variants.

GLRB Antibody (N-term) - References

Joslyn, G., et al. Alcohol. Clin. Exp. Res. 34(5):800-812(2010)
Wheeler, H.E., et al. PLoS Genet. 5 (10), E1000685 (2009) :
Ziegler, E., et al. Naunyn Schmiedebergs Arch. Pharmacol. 380(4):277-291(2009)
Tabakoff, B., et al. BMC Biol. 7, 70 (2009) :
Ahrens, J., et al. Pharmacology 83(4):217-222(2009)