

GABRA1 Blocking Peptide (C-term)

Synthetic peptide Catalog # BP21477b

Specification

GABRA1 Blocking Peptide (C-term) - Product Information

Primary Accession

P14867

GABRA1 Blocking Peptide (C-term) - Additional Information

Gene ID 2554

Other Names

Gamma-aminobutyric acid receptor subunit alpha-1, GABA(A) receptor subunit alpha-1, GABRA1

Target/Specificity

The synthetic peptide sequence is selected from aa 405-418 of HUMAN GABRA1

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

GABRA1 Blocking Peptide (C-term) - Protein Information

Name GABRA1 (HGNC:4075)

Function

Alpha subunit of the heteropentameric ligand-gated chloride channel gated by Gamma-aminobutyric acid (GABA), a major inhibitory neurotransmitter in the brain (PubMed:23909897, PubMed:25489750, PubMed:29950725, PubMed:30602789). GABA-gated chloride channels, also named GABA(A) receptors (GABAAR), consist of five subunits arranged around a central pore and contain GABA active binding site(s) located at the alpha and beta subunit interface(s) (PubMed:29950725" target="_blank">29950725, PubMed:30602789). When activated by GABA, GABAARs selectively allow the flow of chloride anions across the cell membrane down their electrochemical gradient (PubMed:23909897, PubMed:23909897, PubMed:29950725, PubMed:29950725, PubMed:30602789, PubMed:30602789, PubMed:30602789, PubMed:30602789, PubMed:30602789



Alpha-1/GABRA1-containing GABAARs are largely synaptic (By similarity). Chloride influx into the postsynaptic neuron following GABAAR opening decreases the neuron ability to generate a new action potential, thereby reducing nerve transmission (By similarity). GABAARs containing alpha-1 and beta-2 or -3 subunits exhibit synaptogenic activity; the gamma-2 subunit being necessary but not sufficient to induce rapid synaptic contacts formation (PubMed:23909897, PubMed:25489750). GABAARs function also as histamine receptor where histamine binds at the interface of two neighboring beta subunits and potentiates GABA response (By similarity). GABAARs containing alpha, beta and epsilon subunits also permit spontaneous chloride channel activity while preserving the structural information required for GABA-gated openings (By similarity). Alpha-1-mediated plasticity in the orbitofrontal cortex regulates context-dependent action selection (By similarity). Together with rho subunits, may also control neuronal and glial GABAergic transmission in the cerebellum (By similarity).

Cellular Location

Postsynaptic cell membrane {ECO:0000250|UniProtKB:P08219}; Multi-pass membrane protein. Cell membrane; Multi-pass membrane protein. Cytoplasmic vesicle membrane {ECO:0000250|UniProtKB:P62813}; Multi-pass membrane protein. Note=Mainly located in GABAergic synapses in granule cells, and also in the extrasynaptic membrane at a lower concentration. {ECO:0000250|UniProtKB:P62813}

GABRA1 Blocking Peptide (C-term) - Protocols

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

• Blocking Peptides

GABRA1 Blocking Peptide (C-term) - Images

GABRA1 Blocking Peptide (C-term) - Background

Component of the heteropentameric receptor for GABA, the major inhibitory neurotransmitter in the vertebrate brain. Functions also as histamine receptor and mediates cellular responses to histamine. Functions as receptor for diazepines and various anesthetics, such as pentobarbital; these are bound at a separate allosteric effector binding site. Functions as ligand-gated chloride channel (By similarity).

GABRA1 Blocking Peptide (C-term) - References

Schofield P.R., et al. FEBS Lett. 244:361-364(1989). Mural R.I., et al. Submitted (SEP-2005) to the EMBL/GenBank/DDBI databases. Garrett K.M., et al. Biochem. Biophys. Res. Commun. 156:1039-1045(1988). Lachance-Touchette P., et al. Eur. J. Neurosci. 34:237-249(2011). Carvill G.L., et al. Neurology 82:1245-1253(2014).