

**GRIN2B Blocking Peptide (C-Term)**

Synthetic peptide

Catalog # BP21859b

**Specification**

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**GRIN2B Blocking Peptide (C-Term) - Product Information**

Primary Accession

[Q13224](#)

Other Accession

[Q01097](#), [Q00960](#)**GRIN2B Blocking Peptide (C-Term) - Additional Information**

Gene ID 2904

**Other Names**

Glutamate receptor ionotropic, NMDA 2B, GluN2B, Glutamate [NMDA] receptor subunit epsilon-2, N-methyl D-aspartate receptor subtype 2B, NMDAR2B, NR2B, N-methyl-D-aspartate receptor subunit 3, NR3, hNR3, GRIN2B, NMDAR2B

**Target/Specificity**

The synthetic peptide sequence is selected from aa 1320-1332 of HUMAN GRIN2B

**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.

**GRIN2B Blocking Peptide (C-Term) - Protein Information**

Name GRIN2B

Synonyms NMDAR2B

**Function**

Component of NMDA receptor complexes that function as heterotetrameric, ligand-gated ion channels with high calcium permeability and voltage-dependent sensitivity to magnesium. Channel activation requires binding of the neurotransmitter glutamate to the epsilon subunit, glycine binding to the zeta subunit, plus membrane depolarization to eliminate channel inhibition by Mg(2+) (PubMed: [8768735](http://www.uniprot.org/citations/8768735), PubMed: [26919761](http://www.uniprot.org/citations/26919761), PubMed: [26875626](http://www.uniprot.org/citations/26875626), PubMed: [28126851](http://www.uniprot.org/citations/28126851)). Sensitivity to glutamate and channel kinetics depend on the subunit composition (PubMed: [8768735](http://www.uniprot.org/citations/8768735))

target="\_blank">8768735</a>, PubMed:<a href="http://www.uniprot.org/citations/26875626" target="\_blank">26875626</a>). In concert with DAPK1 at extrasynaptic sites, acts as a central mediator for stroke damage. Its phosphorylation at Ser-1303 by DAPK1 enhances synaptic NMDA receptor channel activity inducing injurious Ca<sup>2+</sup> influx through them, resulting in an irreversible neuronal death. Contributes to neural pattern formation in the developing brain. Plays a role in long-term depression (LTD) of hippocampus membrane currents and in synaptic plasticity (By similarity).

#### **Cellular Location**

Cell membrane; Multi-pass membrane protein {ECO:0000250|UniProtKB:Q00960}. Postsynaptic cell membrane {ECO:0000250|UniProtKB:Q00960}; Multi-pass membrane protein {ECO:0000250|UniProtKB:Q00960}. Late endosome {ECO:0000250|UniProtKB:Q01097}. Lysosome {ECO:0000250|UniProtKB:Q01097}. Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:Q01097}. Note=Co-localizes with the motor protein KIF17 along microtubules. {ECO:0000250|UniProtKB:Q01097}

#### **Tissue Location**

Primarily found in the fronto-parieto-temporal cortex and hippocampus pyramidal cells, lower expression in the basal ganglia.

### **GRIN2B Blocking Peptide (C-Term) - Protocols**

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

- [Blocking Peptides](#)

### **GRIN2B Blocking Peptide (C-Term) - Images**

### **GRIN2B Blocking Peptide (C-Term) - Background**

NMDA receptor subtype of glutamate-gated ion channels with high calcium permeability and voltage-dependent sensitivity to magnesium. Mediated by glycine. In concert with DAPK1 at extrasynaptic sites, acts as a central mediator for stroke damage. Its phosphorylation at Ser-1303 by DAPK1 enhances synaptic NMDA receptor channel activity inducing injurious Ca<sup>2+</sup> influx through them, resulting in an irreversible neuronal death (By similarity).

### **GRIN2B Blocking Peptide (C-Term) - References**

Adams S.L.,et al.Biochim. Biophys. Acta 1260:105-108(1995).  
Hess S.D.,et al.J. Pharmacol. Exp. Ther. 278:808-816(1996).  
Mandich P.,et al.Submitted (FEB-1997) to the EMBL/GenBank/DDBJ databases.  
Mandich P.,et al.Genomics 22:216-218(1994).  
Schito A.M.,et al.Neurosci. Lett. 239:49-53(1997).