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NMDAR2B (YD35507) Rabbit mAb

货号: **AYD16566**

产品信息

反应	Human, Mouse, Rat
宿主	Rabbit
克隆性	Monoclonal
预测反应	
应用	WB IHC-P IP
推荐浓度	
理论分子量	166kDa/166kDa/166kDa
实测分子量	
形式	Liquid
保存条件	Store at -20°C. Avoid freeze / thaw cycles. Buffer: PBS with 0.75% BSA,50% glycerol,pH7.3.
偶联物	Unconjugated
阳性对照	
细胞定位	Cell membrane, Postsynaptic cell membrane, Cell projection, dendrite, Late endosome, Lysosome, Cytoplasm, cytoskeleton
纯化	亲和纯化

抗原信息

抗原信息	
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靶点信息

研究背景	<p>Component of N-methyl-D-aspartate (NMDA) receptors (NMDARs) that function as heterotetrameric, ligand-gated cation channels with high calcium permeability and voltage-dependent block by Mg(2+) (PubMed:24272827, PubMed:24863970, PubMed:26875626, PubMed:26919761, PubMed:27839871, PubMed:28095420, PubMed:28126851, PubMed:38538865, PubMed:8768735). Participates in synaptic plasticity for learning and memory formation by contributing to the long-term depression (LTD) of hippocampus membrane currents (By similarity). Channel activation requires binding of the neurotransmitter L-glutamate to the GluN2 subunit, glycine or D-serine binding to the GluN1 subunit, plus membrane depolarization to eliminate channel inhibition by Mg(2+) (PubMed:24272827, PubMed:24863970, PubMed:26875626, PubMed:26919761, PubMed:27839871, PubMed:28095420, PubMed:28126851, PubMed:38538865, PubMed:8768735). NMDARs mediate simultaneously the potassium efflux and the influx of calcium and sodium (By similarity). Each GluN2 subunit confers differential attributes to channel properties, including activation, deactivation and desensitization kinetics, pH sensitivity, Ca2(+) permeability, and binding to allosteric modulators (PubMed:26875626, PubMed:28095420, PubMed:28126851, PubMed:38538865, PubMed:8768735). 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 Ca2+ influx through them, resulting in an irreversible neuronal death (By similarity) Component of N-methyl-D-aspartate (NMDA) receptors (NMDARs) that function as heterotetrameric, ligand-gated cation channels with high calcium permeability and voltage-dependent block by Mg(2+) (PubMed:26912815). Participates in synaptic plasticity for learning and memory formation by contributing to the long-term depression (LTD) of hippocampus membrane currents (PubMed:8789948). Channel activation requires binding of the neurotransmitter L-glutamate to the GluN2 subunit, glycine or D-serine binding to the GluN1 subunit, plus membrane depolarization to eliminate channel inhibition by Mg(2+) (Probable) (PubMed:1377365, PubMed:20141836, PubMed:7790891). NMDARs mediate simultaneously the potassium efflux and the influx of calcium and sodium (By similarity). Each GluN2 subunit confers differential attributes to channel properties, including activation, deactivation and desensitization kinetics, pH sensitivity, Ca2(+) permeability, and binding to allosteric modulators (By similarity). In concert with DAPK1 at extrasynaptic sites, acts as a central mediator for stroke damage (PubMed:20141836). Its phosphorylation at Ser-1303 by DAPK1 enhances synaptic NMDA receptor channel activity inducing injurious Ca2+ influx through them, resulting in an irreversible neuronal death (PubMed:20141836) Component of N-methyl-D-aspartate (NMDA) receptors (NMDARs) that function as heterotetrameric, ligand-gated cation channels with high calcium permeability and voltage-dependent block by Mg(2+) (PubMed:11929923, PubMed:19910922, PubMed:21677647, PubMed:24607230, PubMed:24876489, PubMed:27135925, PubMed:7524561). Participates in synaptic plasticity for learning and memory formation by contributing to the long-term depression (LTD) of hippocampus membrane currents (By similarity). Channel activation requires binding of the neurotransmitter L-glutamate to the GluN2 subunit, glycine or D-serine binding to the GluN1 subunit, plus membrane depolarization to eliminate channel inhibition by Mg(2+) (PubMed:11929923, PubMed:19910922, PubMed:21677647, PubMed:24607230, PubMed:24876489, PubMed:27135925, PubMed:7524561). NMDARs mediate simultaneously the potassium efflux and the influx of calcium and sodium (By similarity). Each GluN2 subunit confers differential attributes to channel properties, including activation, deactivation and desensitization kinetics, pH sensitivity, Ca2(+) permeability, and binding to allosteric modulators (PubMed:10436042, PubMed:11929923, PubMed:24607230, PubMed:9463421). In concert with DAPK1 at extrasynaptic sites, acts as a central mediator for stroke damage (By similarity). Its phosphorylation at Ser-1303 by DAPK1 enhances synaptic NMDA receptor channel activity inducing injurious Ca2+ influx through them, resulting in an irreversible neuronal death (By similarity)</p>
基因ID	2904
基因名	GRIN2B, Grin2b
Swiss	Q13224 (https://www.uniprot.org/uniprotkb/Q13224/entry), Q01097 (https://www.uniprot.org/uniprotkb/Q01097/entry), Q00960 (https://www.uniprot.org/uniprotkb/Q00960/entry)
别名	NMDAR2B (YD35507),NMDAR2B (YD35507) Rabbit mAb,GRIN2B,Glutamate [NMDA] receptor subunit epsilon-2,N-methyl D-aspartate receptor subtype 2B,N-methyl-D-aspartate receptor subunit 3,NMDAR2B

产品验证

实验步骤

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