

Product Datasheet

Anti-NMDA NR2B Subunit (Tyr1472)



Overview	
Catalog #	p1516-1472
Host Species	Rabbit Polyclonal
Format	Antigen Affinity Purified from Pooled Serum
Applications	WB 1:1000 ICC 1:100
Species Tested	Mouse, Rat
Expected Reactivity	Bovine, Canine, Chicken, Human, Non-Human Primate, Zebrafish
Immunogen	Synthetic phospho-peptide corresponding to amino acid residues surrounding Tyr1472 of the NR2B subunit of the rat NMDA receptor, conjugated to keyhole limpet hemocyanin (KLH).
Molecular Weight	180 kDa
Cite this Antibody	PhosphoSolutions Cat# p1516-1472, RRID: AB_2492182

Images



Western blot of rat hippocampal lysate showing specific immunolabeling of the ~180 kDa NR2B subunit of the NMDAR phosphorylated at Tyr¹⁴⁷² in the first lane (-). Phosphospecificity is shown in the second lane (+) where immunolabeling is eliminated by lysate treatment with λ phosphatase (400 units/100uL lysate for 30 min).

Target Description	The ion channels activated by glutamate that are sensitive to N-methyl-Daspartate (NMDA) are designated NMDA receptors (NMDAR). The NMDAR plays an essential role in memory, neuronal development and it has also been implicated in several disorders of the central nervous system including Alzheimer's, epilepsy and ischemic neuronal cell death (Grosshans et al., 2002; Wenthold et al., 2003; Carroll and Zukin, 2002). The NMDA receptor is also one of the principal molecular targets for alcohol in the CNS (Lovinger et al., 1989; Alvestad et al., 2003; Snell et al., 1996). Channels with physiological characteristics are produced when the NR1 subunit is combined with one or more of the NMDAR2 (NR2 A-D) subunits (Ishii et al., 1993). Overexpression of the NR2B-subunit of the NMDA Receptor has been associated with increases in learning and memory while aged, memory impaired animals have deficiencies in NR2B expression (Clayton et al., 2002a; Clayton et al., 2002b). Recent work suggests that phosphorylation of Tyr-1472 on NR2B may regulate the functional expression the receptor in LTP and other forms of plasticity (Nakazawa et al., 2001; Roche et al., 2001).
Specificity	Specific for endogenous levels of the ~180 kDa NMDAR NR2B-subunit protein phosphorylated at Tyr1472. Immunolabeling is completely eliminated by treatment with λ -phosphatase.
Production/Purification	Prepared from pooled rabbit serum by affinity purification via sequential chromatography on phospho and non-phosphopeptide affinity columns.
Quality Control	Western blots performed on each lot.
Buffer	10 mM HEPES (pH 7.5), 150 mM NaCl, 100 μg per ml BSA and 50% glycerol.
Storage	Storage at -20°C is recommended, as aliquots may be taken without freeze/thawing due to presence of 50% glycerol.
Stability	After date of receipt, stable for at least 1 year at -20°C.

Significant Citations

He, R.B., Li, L., Liu, L.Z., Ma, Y.J., Fan, S.J., Liu, L.R., Li, W.B. and Xian, X.H., 2023. Ceftriaxone improves impairments in synaptic plasticity and cognitive behavior in APP/PS1 mouse model of Alzheimer's disease by inhibiting extrasynaptic NMDAR-STEP61 signaling. Journal of *Neurochemistry*, *166*(2):215-232.

Su, L., Bai, X., Niu, T., Zhuang, X., Dong, B., Wang, G. and Yu, Y., 2021. P2Y1 purinergic receptor inhibition attenuated remifentanil-induced postoperative hyperalgesia via decreasing NMDA receptor phosphorylation in dorsal root ganglion. Brain Research Bulletin, 177, pp.352-362.

Higginbotham, J.A., Wang, R., Richardson, B.D., Shiina, H., Tan, S.M., Presker, M.A., Rossi, D.J. and Fuchs, R.A., 2021. CB1 receptor signaling modulates amygdalar plasticity during context-cocaine memory reconsolidation to promote subsequent cocaine seeking. *Journal of Neuroscience*, *41(4)*, pp.613-629.

Xu, J., Kurup, P., Zhang, Y., Goebel-Goody, S.M., Wu, P.H., Hawasli, A.H., Baum, M.L., Bibb, J.A. and Lombroso, P.J., 2009. Extrasynaptic NMDA receptors couple preferentially to excitotoxicity via calpain-mediated cleavage of STEP. *Journal of Neuroscience*, *29*(*29*), pp.9330-9343.

Alvestad, R.M., Grosshans, D.R., Coultrap, S.J., Nakazawa, T., Yamamoto, T. and Browning, M.D., 2003. Tyrosine dephosphorylation and ethanol inhibition of N-methyl-D-aspartate receptor function. *Journal of Biological Chemistry*, *278(13)*, pp.11020-11025.

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