NMDAR1 (Phospho-Ser896) Antibody

#11104

Catalog Number: 11104-1, 11104-2

Amount: 50μg/50μl, 100μg/100μl

Swiss-Prot No. :Q05586

Form of Antibody: Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl,0.02% sodium azide and 50% glycerol.

Storage/Stability: Store at -20℃/1 year

Immunogen: The antiserum was produced against synthesized phosphopeptide derived from Human NMDAR1 around the phosphorylation site of serine 896 (R-R-S^{P}-S-K).

Purification: The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phosphorylation site.

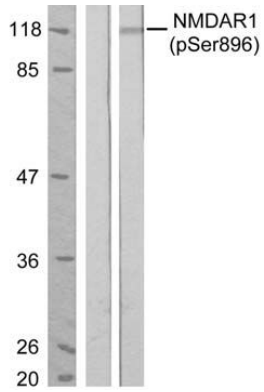
Specificity/Sensitivity:NMDAR1 (phospho-Ser896) antibody detects endogenous levels of NMDAR1 only when phosphorylated at serine 896.

Reactivity: Human,Mouse,Rat

Applications:

Predicted MW: 120 kd

WB **:**1:500~1:1000



Estradiol + -

Western blot analysis of extract from MCF7 cells, untreated or treated with estradiol (100nM, 20min), using NMDAR1 (phospho-Ser896) antibody (#11104).

Background :

NMDA receptors are members of the ionotropic class of glutamate receptors, which also includes Kainate and AMPA receptors. NMDA receptors consist of NR1 subunits combined with one or more NR2 (A-D) or NR3 (A-B) subunits. The ligand-gated channel is permeable to cations including Ca2+, and at resting membrane potentials NMDA receptors are inactive due to a voltage-dependent blockade of the channel pore by Mg2+. NMDA receptor activation, which requires binding of glutamate and glycine, leads to an influx of Ca2+ into the postsynaptic region where it activates several signaling cascades, including pathways leading to the induction of long-term potentiation (LTP) and depression (LTD). NMDA receptors have a critical role in excitatory synaptic transmission and plasticity in the CNS. They govern a range of physiological conditions including neurological disorders caused by excitotoxic neuronal injury, psychiatric disorders and neuropathic pain syndromes.

References:

Tyszkiewicz JP, et al. J Physiol. 2004 Feb 1; 554(Pt 3): 765-777