# GluR1 Antibody
Cat. No. CG1176

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|---|---|
| **Applications:** | **WB, IF** |
| **Detected MW:** | **101 kDa** |
| **Species & Reactivity:** | **Human, Mouse, Rat** |
| **Isotype:** | **Rabbit IgG** |

## BACKGROUND

AMPA receptors are members of the ionotropic class of glutamate receptors, which also includes NMDA and kainate receptors. The classification of glutamate receptors is based on their activation by different pharmacologic agonists. AMPA receptors mediate fast excitatory synaptic transmission in the CNS and play a key role in hippocampal synaptic long-term potentiation (LTP) and depression (LTD), which are two well established cellular models of learning and memory. AMPA receptors consist of GluR1-4 subunits which assemble as homomers or heteromers to form functional AMPA receptors. Each subunit possesses transmembrane regions, and all arranged to form a ligand-gated ion channel. The subunit composition determines the physiological properties of AMPA receptors: those containing the GluR2 subunit show low permeability to $Ca^{2+}$ whereas those lacking this subunit show high $Ca^{2+}$ permeability.[1]

L-glutamate acts as an excitatory neurotransmitter at many synapses in the central nervous system. Binding of the excitatory neurotransmitter L-glutamate induces a conformation change, leading to the opening of the cation channel, and thereby converts the chemical signal to an electrical impulse. The receptor then desensitizes rapidly and enters a transient inactive state. Phosphorylation of the GluR1 subunit of AMPA receptors is modulated during LTP and LTD and is critical for LTD and LTP expression and the retention of memories.[2] AMPA receptor peak response open probability can be increased by PKA through phosphorylation of GluR1 Ser845.[3] In addition, regulation of GLuR1 expression is a critical mechanism for induction of long-term potentiation (LTP) in the hippocampus. It was demonstrated this mTOR acts downstream BDNF in the hippocampus and controls the increase of synaptic GluR1 necessary for memory consolidation.[4]


**References**:
1. Mayer, M.L.: Curr. Opin. Neurobiol. 15:282-8, 2005
2. Lee, H. K. et al: Cell 112:631-43, 2003
3. Banke, T.G. et al: J. Neurosci. 20:89-102, 2000
4. Slipczuk, L. et al: PLoS ONE 4:e6007, 2009


## TECHNICAL INFORMATION

**Source:**
GluR1 Antibody is a rabbit antibody raised against a short peptide from N-terminal sequence of human GluR1.

**Specificity and Sensitivity:**
This antibody detects endogenous GluR1 proteins without cross-reactivity with other family members.

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

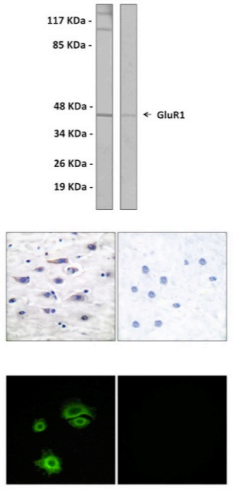
**Storage**:
Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

## APPLICATIONS

| Application: | *Dilution: |
|---|---|
| **WB** | 1:500-1:1,000 |
| **IP** | n/d |
| **IHC** | n/d |
| **ICC** | n/d |
| **FACS** | n/d |
| **IF** | 1:100-1:500 |

*****Optimal dilutions must be determined by end user.*

## QUALITY CONTROL DATA




**Top**: Immunoblotting analysis of extracts from COS7 cells, using Anti-GLUR1, C-Terminal antibody. The lane on the left was treated with the Anti-GLUR1, C-Terminal antibody. The lane on the right (negative control) was treated with both Anti-GLUR1, C-Terminal antibody and the synthesized immunogen peptide.
**Middle**: Immunohistochemistry analysis of paraffin-embedded human brain tissue using Anti-GLUR1, C-Terminal antibody. Cells on the left were treated with the Anti-GLUR1, C-Terminal antibody. Cells on the right (negative control) were treated with both Anti-GLUR1, C-Terminal antibody and the synthesized immunogen peptide.
**Bottom**: Immunofluorescence of A549 cells using Anti-GLUR1, C-Terminal antibody. Cells on the left were treated with the Anti-GLUR1, C-Terminal antibody. Cells on the right (negative control) were treated with both Anti-GLUR1, C-Terminal antibody and the synthesized immunogen peptide.