

## GRIA3

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC IF

**Recommended Dilution:** WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

**Calculated MW:** 101kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human GRIA3

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

g

**Synonym:**

GLUR3; GLURC; GluA3; MRX94; GLUR-C; GLUR-K3

**Catalog #:** A1159

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 2892

**Isotype:** IgG

**Swiss Prot:** P42263

**Purity:** Affinity purification

For research use only.

**Background:**

AMPA- (-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid), kainite- and NMDA- (N-methyl-D-aspartate) receptors are the three main families of ionotropic glutamate-gated ion channels. AMPA receptors (AMPA-Rs) are comprised of four subunits (GluR 1-4) that assemble as homo- or hetero-tetramers and mediate the majority of fast excitatory transmissions in the CNS. AMPARs are implicated in synapse formation, stabilization and plasticity. Post-transcriptional modifications (alternative splicing and nuclear RNA editing) and post-translational modifications (glycosylation, phosphorylation) result in a very large number of permutations, fine-tuning the kinetic properties of AMPARs (1). GluR 3 knockout mice exhibited normal basal synaptic transmission and long-term depression (LTD) but enhanced long-term potentiation (LTP). In contrast, GluR 2/3 double knockout mice are impaired in basal synaptic transmission (2). Aberrant GluR 3 expression or activity is implicated in a number of diseases, including autoimmune epilepsy, X-linked mental retardation, Rett's syndrome, amyotrophic lateral sclerosis and Alzheimer disease (3).

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