

# GluR-δ2 (C-19): sc-26118

## BACKGROUND

Glutamate receptors mediate most excitatory neurotransmission in the brain and play an important role in neural plasticity, neural development and neurodegeneration. Ionotropic glutamate receptors are categorized into NMDA receptors and kainate/AMPA receptors, both of which contain glutamate-gated, cation-specific ion channels. Kainate/AMPA receptors co-localize with NMDA receptors in many synapses and consist of seven structurally related subunits designated GluR-1 to -7 as well as GluR-δ2. The kainate/AMPA receptors are primarily responsible for the fast excitatory neuro-transmission by glutamate whereas the NMDA receptors are functionally characterized by a slow kinetic and a high permeability for  $Ca^{2+}$  ions. The NMDA receptors consist of five subunits:  $\epsilon$  1, 2, 3, 4 and one  $\zeta$  subunit. The  $\zeta$  subunit is expressed throughout the brainstem whereas the four epsilon subunits display limited distribution. In mice, mutations in the gene encoding GluR-δ2 (GRID2) cause the Lurcher phenotype. The gene encoding human GluR-δ2 maps to chromosome 4q22.1.

## REFERENCES

1. Choi, D.W., et al. 1990. The role of glutamate neurotoxicity in hypoxic-ischemic neuronal death. *Annu. Rev. Neurosci.* 13: 171-182.
2. Nakanishi, S., et al. 1992. Molecular diversity of glutamate receptors and implications for brain function. *Science* 258: 597-603.
3. Stern, P., et al. 1992. Fast and slow components of unitary EPSCs on stellate cells elicited by focal stimulation in slices of rat visual cortex. *J. Physiol.* 449: 247-278.
4. Bliss, T.V., et al. 1993. A synaptic model of memory: long-term potentiation in the hippocampus. *Nature* 361: 31-39.

## CHROMOSOMAL LOCATION

Genetic locus: GRID2 (human) mapping to 4q22.1; Grid2 (mouse) mapping to 6 C1.

## SOURCE

GluR-δ2 (C-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of GluR-δ2 of human origin.

## PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-26118 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## STORAGE

Store at 4° C, \*\*DO NOT FREEZE\*\*. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.

## APPLICATIONS

GluR-δ2 (C-19) is recommended for detection of GluR-δ2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

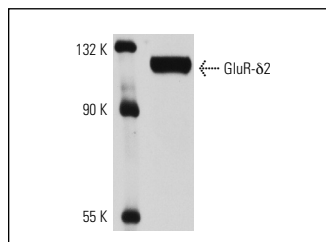
GluR-δ2 (C-19) is also recommended for detection of GluR-δ2 in additional species, including canine, bovine and avian.

Suitable for use as control antibody for GluR-δ2 siRNA (h): sc-42491, GluR-δ2 siRNA (m): sc-42492, GluR-δ2 shRNA Plasmid (h): sc-42491-SH, GluR-δ2 shRNA Plasmid (m): sc-42492-SH, GluR-δ2 shRNA (h) Lentiviral Particles: sc-42491-V and GluR-δ2 shRNA (m) Lentiviral Particles: sc-42492-V.

Molecular Weight of GluR-δ2: 110 kDa.

Positive Controls: rat cerebellum extract: sc-2398.

## DATA



GluR-δ2 (C-19): sc-26118. Western blot analysis of GluR-δ2 expression in rat cerebellum tissue extract.

## SELECT PRODUCT CITATIONS

1. Uemura, T., et al. 2007. Regulation of long-term depression and climbing fiber territory by glutamate receptor δ2 at parallel fiber synapses through its C-terminal domain in cerebellar Purkinje cells. *J. Neurosci.* 27: 12096-12108.
2. Hansen, K.B., et al. 2009. Modulation of the dimer interface at ionotropic glutamate-like receptor δ2 by D-serine and extracellular calcium. *J. Neurosci.* 29: 907-917.
3. Mandolesi, G., et al. 2009. GluR-δ2 expression in the mature cerebellum of hotfoot mice promotes parallel fiber synaptogenesis and axonal competition. *PLoS ONE* 4: e5243.
4. Verkerk, A.J., et al. 2009. Mutation in the AP4M1 gene provides a model for neuroaxonal injury in cerebral palsy. *Am. J. Hum. Genet.* 85: 40-52.

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