

PRODUCT INFORMATION

Tag	C-Flag Tag
Target	GRIA2
Synonyms	GLUR2, GLURB, GluA2, GluR-K2, HBGR2, NEDLIB, gluR-2, gluR-B
Description	Human GRIA2 full length protein-synthetic nanodisc
Delivery	6~8weeks
Uniprot ID	P42262
Expression Host	HEK293
Protein Families	Ion Channels: Glutamate Receptors
Protein Pathways	N/A
Molecular Weight	The human full length GRIA2 protein has a MW of 98.8kDa
Formulation & Reconstitution	Lyophilized from nanodisc solubilization buffer (20 mM Tris-HCl, 150 mM NaCl, pH 8.0). Normally 5% - 8% trehalose is added as protectants before lyophilization. Please see Certificate of Analysis for specific instructions. Do not use solvents with a pH below 6.5 or those containing high concentrations of divalent metal ions (greater than 5 mM) in subsequent experiments. Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.
Storage & Shipping	Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.
Background	Glutamate receptors are the predominant excitatory neurotransmitter receptors in the mammalian brain and are activated in a variety of normal neurophysiologic processes. This gene product belongs to a family of glutamate receptors that are sensitive to alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA), and function as ligand-activated cation channels. These channels are assembled from 4 related subunits, GRIA1-4. The subunit encoded by this gene (GRIA2) is subject to RNA editing (CAG->CGG; Q->R) within the second transmembrane domain, which is thought to render the channel impermeable to Ca ²⁺ . Human and animal studies suggest that pre-mRNA editing is essential for brain function, and defective GRIA2 RNA editing at the Q/R site may be relevant to amyotrophic lateral sclerosis (ALS) etiology. Alternative splicing, resulting in transcript variants encoding different isoforms, (including the flip and flop isoforms that vary in their signal transduction properties), has been noted for this gene. [provided by RefSeq, Jul 2008]
Usage	Research use only
Conjugate	Unconjugated

