abcam

A guide to glutamate receptors

Contents

Glutamate receptors

L-Glutamate is the principle excitatory neurotransmitter in the mammalian CNS. It acts through ligand gated ion channels (ionotropic receptors) and G-protein coupled (metabotropic) receptors. These receptors are involved in excitatory synaptic transmission and synaptic plasticity which are thought to underlie learning and memory. Ionotropic glutamate receptors

Ionotropic glutamate receptors

Ionotropic glutamate receptors (iGluRs) are ligand-gated ion channels that mediate the majority of excitatory neurotransmission within the brain.

Structure

iGluRs are found on pre- and postsynaptic cell membranes, primarily within the CNS[1] and are divided into AMPA receptors, NMDA receptors and kainate receptors. These subfamilies are named according to their affinities for the synthetic agonists, AMPA (α-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate), NMDA (N-methyl-d-aspartate) and kainic acid (kainate)[2]. The delta receptor family has been classified as an iGluR by sequence homology3 (Figure 1).

Similar to other ligand-gated ion channels, iGluRs are composed of four domains: the extracellular amino-terminal domain (ATD), the extracellular ligand-binding domain (LBD), four transmembrane domains (TMD), and an intracellular carboxyl-terminal domain (CTD). At the second TMD (TMII), there is a re-entrant loop that gives rise to an extracellular N-terminus and an intracellular C-terminus (Figure 2).

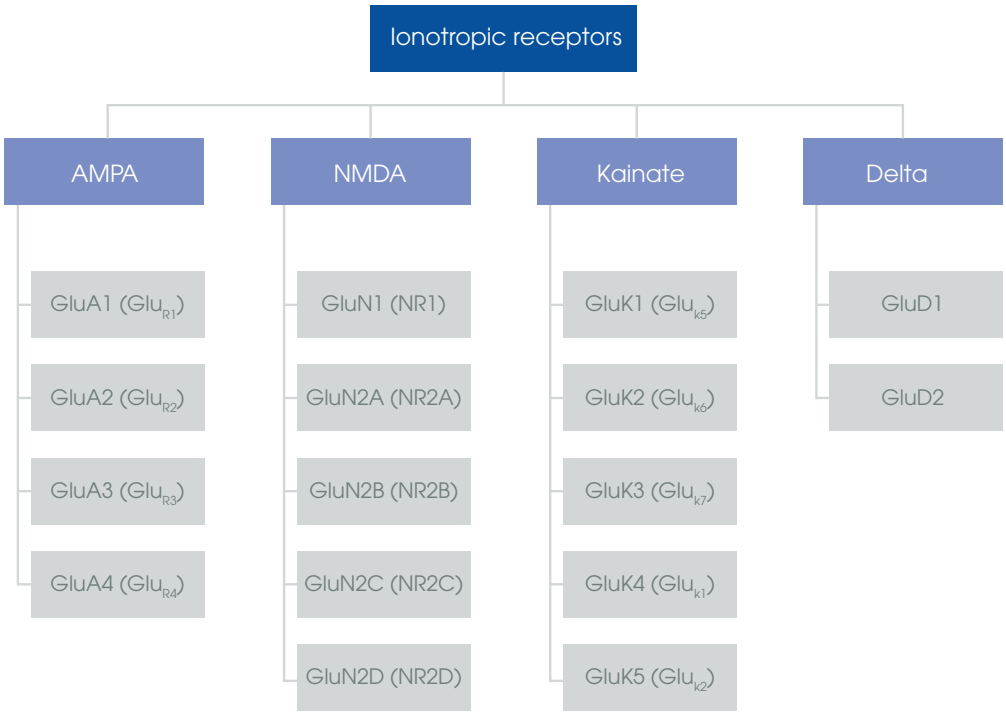


Figure 1. Diagram of the ionotropic glutamate receptor subgroups.

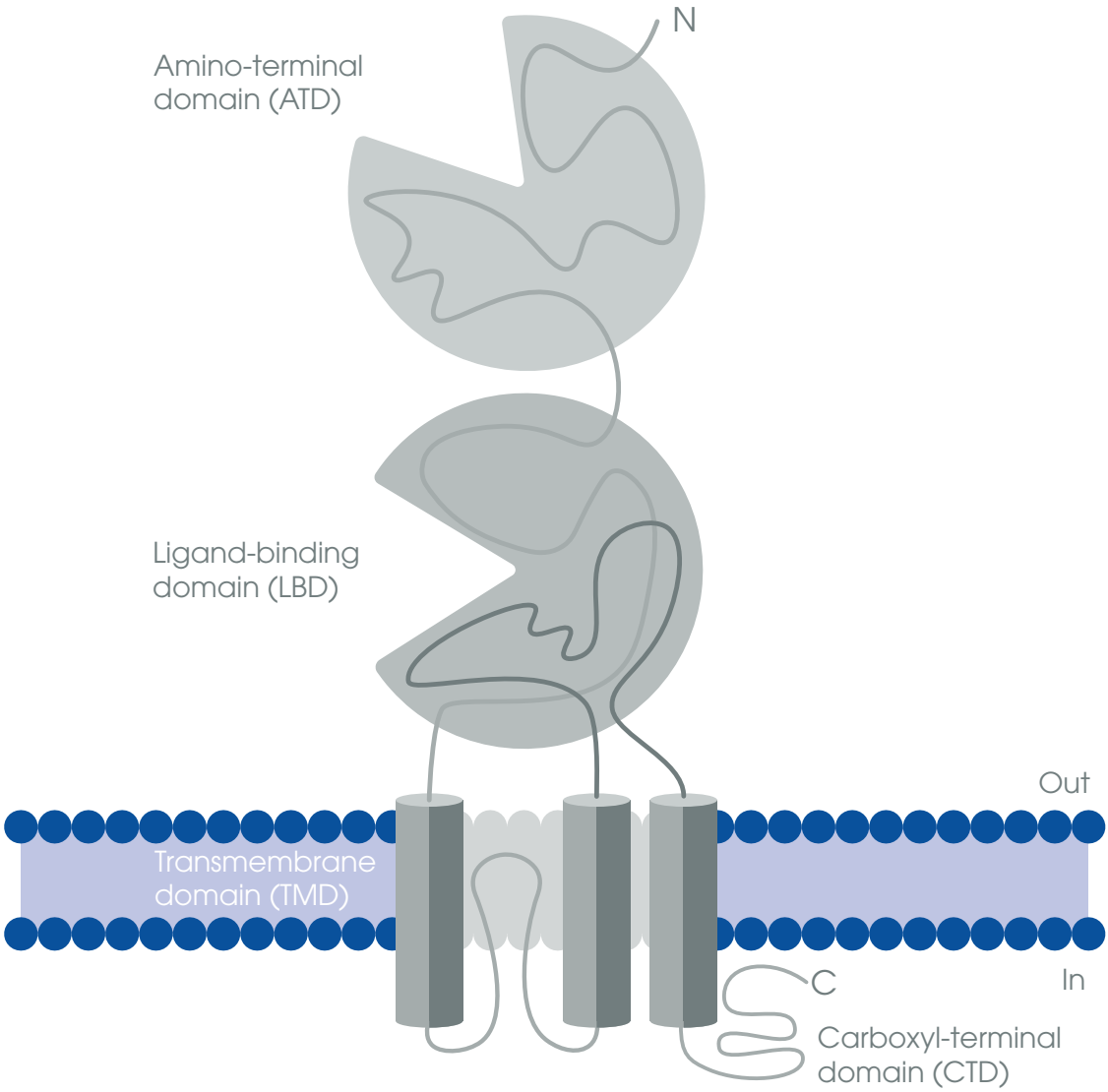


Figure 2. Schematic structure of the ionotropic glutamate receptors. (Adapted from Traynelis, S. F. *et al.*, 2010)

Function

iGluRs mediate fast excitatory neurotransmission and are involved in synaptic plasticity and our capacity to learn and form memories. As nonselective cation channels, iGluRs allow ions like Na^+, K^+ or Ca^{2+}, to pass through the channel upon binding with glutamate[1,4]. Activation of a significant number of iGluRs generates an action potential (AP). After this signal is received, excitatory amino acid transporters (EAATs) remove glutamate from the synaptic cleft, effectively turning off the signal in preparation for subsequent APs.

Prolonged stimulation of iGluRs can result in excitotoxicity as over-stimulation causes an abnormal membrane voltage potential that inhibits glutamate uptake by EAATs. Excitotoxicity is a major contributor to neurodegenerative disorders and nervous system injuries, making iGluRs an interesting target for various therapeutic developments[5].

AMPA receptors

AMPA receptors are co-expressed with NMDA receptors at most glutamatergic synapses in glia and neurons, and mediate the majority of fast excitatory neurotransmission within the CNS6. The modulation of AMPA receptors is also a primary mechanism of synaptic plasticity: increasing the number of AMPA receptors at the postsynaptic site can increase the response to an action potential[7,8].

The Ca^{2+}-permeability of AMPA receptors is dictated by the GluA2 subunit. Normally impermeable to Ca^{2+}, post-transcriptional editing of GluA2 at the TMII region (the Q/R editing site) can convert glutamine (Q) to arginine (R), rendering the receptor Ca^{2+}-permeable[4].

GluA2 proteins throughout the CNS are almost exclusively in the calcium-impermeable state. This is important because Ca^{2+} entry through AMPA receptors can trigger neuronal death and can contribute to the pathogenesis of diseases like amyotrophic lateral sclerosis (ALS)[9]. The distribution of Ca^{2+}-permeable and impermeable AMPA receptors therefore serves as an indicator of selective neuronal vulnerability.

NMDA receptors

NMDA receptors require the co-binding of glycine in addition to glutamate for activation. A glycine binding site is provided by the GluN1 and GluN3 subunits[10]. These receptors allow the flow of Ca^{2+}, in addition to Na^{+} and K^{+}, but activate significantly slower than AMPA and kainate receptors.

Mg^{2+} normally blocks the NMDA channel, meaning weak stimuli triggering glutamate-binding result in only limited Ca^{2+} conductance[1,11]. In these instances, AMPA receptors mediate the excitatory postsynaptic potential through conductance of Na^{+} and K^{+}. In the presence of strong stimuli, AMPA receptors depolarize the membrane enough to dislodge Mg^{2+} from the NMDA receptor channel. This allows NMDA receptors to respond to glutamate-binding and permit the flow of large amounts of Ca^{2+}, Na^{+} and K^{+} through the channel. NMDA receptors therefore function as molecular coincidence detectors, requiring both glutamate binding and a strong depolarizing stimulus[12].

The amount of Ca^{2+} entering the cell, as modulated by NMDA receptors, affects an array of local signal transduction complexes: Ca^{2+} can act as a secondary messenger in several signaling cascades. For example, activation of calcium/calmodulin-dependent kinase II (CaMKII), upregulation of AMPA receptor expression at the synaptic membrane, and subsequent phosphorylation of the GluA2 AMPA receptor subunit, can result in synaptic enhancement and long-term potentiation[5].

Since NMDA receptors are present on both excitatory and inhibitory neurons, excessive activation can lead to excitotoxicity and neuronal death (as seen in Huntingdon's disease), or a reduced activity that disturbs the balance of excitation/inhibition (as seen in schizophrenia)[13].

Kainate receptors

Traditionally, kainate receptors have been grouped with AMPA receptors as non-NMDA receptors, sharing many similar agonists and antagonists, but are now known to be a separate group[14].

Kainate receptors require extracellular Na^{+} and Cl^{-} for their activation[15,16]. They are found throughout the CNS where they are usually co-expressed with AMPA and NMDA receptors, although in some regions such as the retina for example, they exist independently. Unlike AMPA and NMDA receptors, kainate receptors can also signal through G-proteins, behaving like metabotropic receptors: canonical signaling (ionotropic) is responsible for membrane depolarization, postsynaptic responses, and neurotransmitter release; while non-canonical (metabotropic) signaling activates G-proteins to affect membrane excitability, neuronal and circuit maturation, and neurotransmitter release[17].

Postsynaptically, kainate receptors work much like AMPA and NMDA receptors, propagating the excitatory postsynaptic current. Presynaptically, they modulate the release of neurotransmitters at both excitatory and inhibitory synapses[14]. Kainate receptors also play a critical role in synaptic plasticity and are linked to a number of neurological diseases such as epilepsy, schizophrenia, and autism, yet their involvement in brain pathologies remain unclear[17].

Product highlight

Name	D-AP5 (ab120003)
Description	Competitive NMDA receptor glutamate site antagonist
Purity	> 99%
Solubility	Soluble in water to 100 mM
Pack sizes	1 mg, 10 mg, 50 mg, 100 mg
Number of citations	125

Product highlight

Name	Anti-Glutamate Receptor 1 (AMPA subtype) antibody (ab31232)
Target	GluA1
Description	Rabbit polyclonal to Glutamate Receptor 1 (AMPA subtype)
Tested applications	Western blot, Immunohistochemistry
Species reactivity	Mouse, Rat, Human, Marmoset (common)
Immunogen	Synthetic peptide derived from within residues 850 to the C-terminus of Human Glutamate Receptor 1 (AMPA subtype).
Blocking peptide	Human Glutamate Receptor 1 (AMPA subtype) peptide (ab28424)

Browse the full range of glutamate-related products at:
www.abcam.com/glutamate-products

Metabotropic glutamate receptors

Metabotropic glutamate receptors (mGluRs) are class C, G-protein-coupled receptors (GPCRs) in the CNS that play a role in modulating synaptic transmission and neuronal excitability.

Structure

mGluRs provide a mechanism through which glutamate can modulate cell excitability and synaptic transmission via second messenger signaling pathways. These receptors lack ion channels, and instead affect other channels through the activation of intermediate molecules called G-proteins[18]. mGluRs are divided into three groups based on their sequence similarity, pharmacology and signaling mechanisms: Group I ($mGlu_1$ and $mGlu_5$ receptors), Group II ($mGlu_2$ and $mGlu_3$ receptors) and Group III ($mGlu_4$, $mGlu_6$, $mGlu_7$ and $mGlu_8$ receptors)[19] (Figure 3).

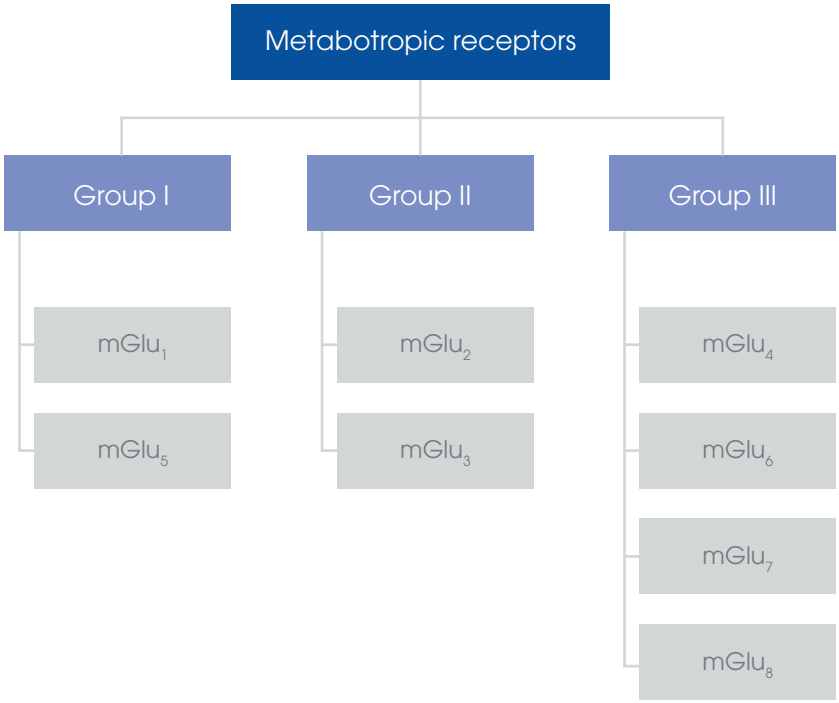


Figure 3. Diagram of the metabotropic glutamate receptor subgroups.

mGluRs consist of seven transmembrane-spanning domains, an extracellular N-terminal domain and an intracellular C-terminus. The N-terminal domain is formed by a pair of hinged domains, termed the Venus fly-trap domain, and is responsible for binding glutamate and activating the receptor[20]. The C-terminal is important in modulating G-protein-coupling (Figure 4).

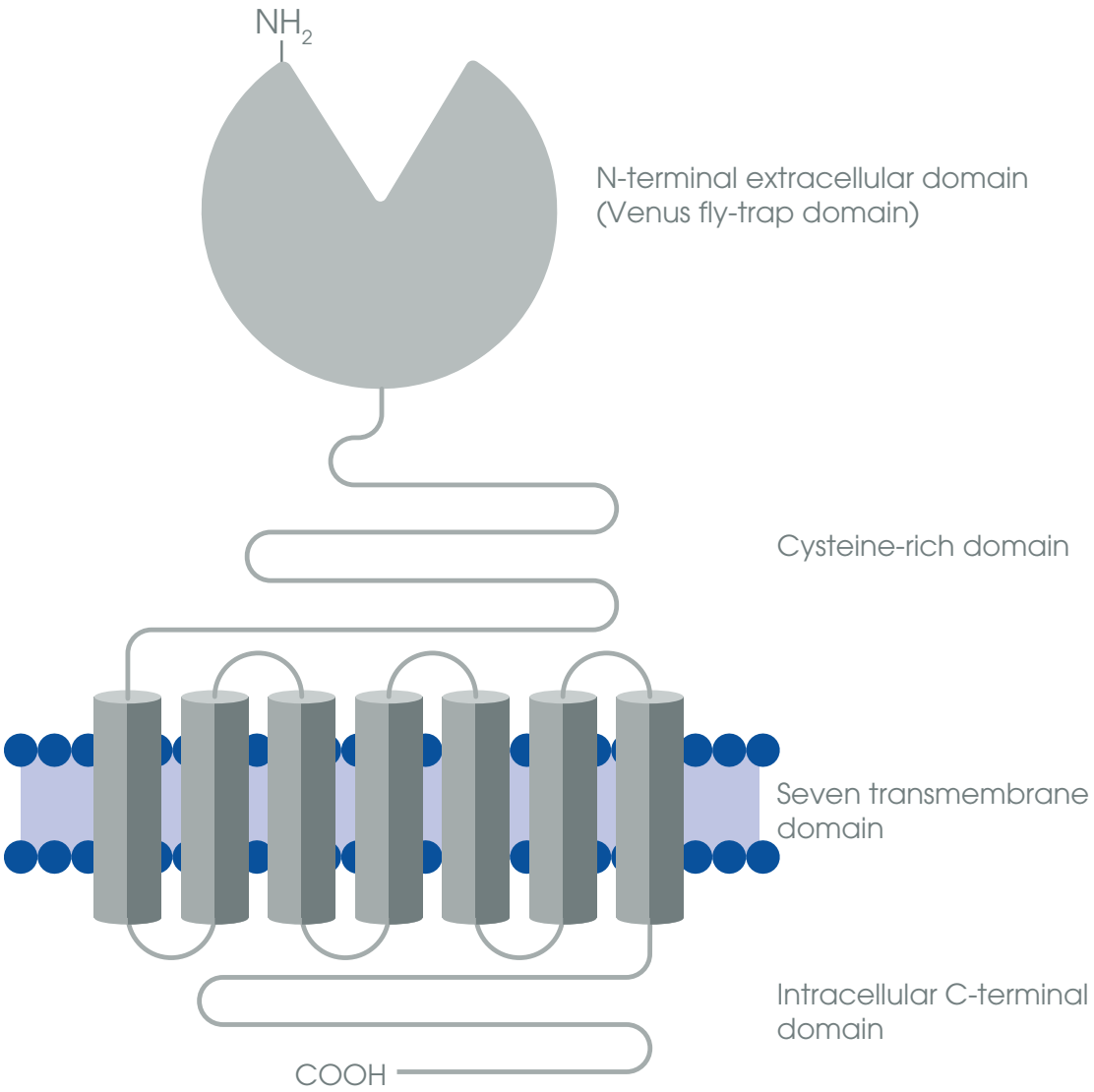


Figure 4: Schematic structure of the metabotropic glutamate receptors. (Adapted from Kenny, P. J. & Markou, 2004)

Function

mGluRs function as neuromodulators that can modulate neuronal excitability or neurotransmitter release[18,21]. In general, Group I mGluRs increase neuron excitability, whereas Groups II and III tend to suppress neuronal excitability[22]. Group I mGluRs are coupled to G_{aq} proteins, and activate phospholipase C[18]. Groups II and III receptors are coupled to $G_{i/o}$ proteins, leading to adenylyl cyclase inhibition and cAMP formation, limiting downstream protein kinase A (PKA) activation[18].

Group I: $mGlu_1$ and $mGlu_5$

Group I mGluRs are primarily located postsynaptically and function by stimulating phospholipase C (PLC) to increase levels of diacylglycerol and inositol triphosphate. This activates protein kinase C (PKC) and the release of intracellular Ca^{2+} which ultimately inhibits presynaptic K^+ channels and delays nerve terminal repolarization. Group I mGluRs can also activate a range of downstream effectors, which may be important in the regulation of synaptic plasticity[18].

$mGlu_5$ receptors are an interesting therapeutic target for negative allosteric modulators as a potential therapy for depression, fragile X syndrome, anxiety, obsessive-compulsive disorders, and levodopa-induced dyskinesia in Parkinson's disease[23].

Group II: $mGlu_2$ and $mGlu_3$

Group II mGluRs function presynaptically to suppress neuronal excitability through the inhibition of adenylate cyclase[22]. $mGlu_2$ and $mGlu_3$ subtypes share a high degree of sequence similarity and are highly expressed in the hippocampus, cortex, nucleus accumbens, striatum and amygdala[24]. These receptors are potential novel targets in the treatment of anxiety disorders and schizophrenia[25-27].

Group III: $mGlu_4$, $mGlu_6$, $mGlu_7$ and $mGlu_8$

Group III mGluRs function in the same manner as Group II, being expressed presynaptically and suppressing neuronal excitability by inhibiting adenylate cyclase[22]. $mGlu_4$ and $mGlu_7$ receptors are widely distributed in the brain[24], $mGlu_6$ receptors are localized to the retina[28], and $mGlu_8$ receptors are primarily found at low levels in the hippocampus, hypothalamus and olfactory bulb[24]. Group III mGluRs play a role in synaptic remodeling and persistent drug seeking and addiction, exerting their effect via an as yet undefined presynaptic mechanism – possibly also extending to postsynaptic modulation[29].

Product highlight

Name	(S)-3,5-DHPG (ab120007)
Description	Selective group I mGlu agonist
Purity	> 99%
Solubility	Soluble in water to 50 mM
Pack size	5 mg, 10 mg
Number of citations	17

Product highlight

Name	Anti-Metabotropic Glutamate Receptor 5 antibody (EPR2425Y) (Alexa Fluor® 488) (ab196481)
Target	$mGlu_5$
Description	Rabbit monoclonal (EPR2425Y) to Metabotropic Glutamate Receptor 5 (Alexa Fluor® 488)
Tested applications	Flow Cytometry, Immunocytochemistry/ Immunofluorescence
Species reactivity	Mouse
Immunogen	Synthetic peptide (the amino acid sequence is considered to be commercially sensitive) corresponding to Human Metabotropic Glutamate Receptor 5 aa 1150 to the C-terminus (C terminal).
Blocking peptide	Metabotropic Glutamate Receptor 5 peptide (ab139974)

This product is a recombinant rabbit monoclonal antibody, produced using Abcam's RabMAb® technology. RabMAb® technology is covered by the following U.S. Patents, No. 5,675,063 and/or 7,429,487. Alexa Fluor® is a registered trademark of Life Technologies. Alexa Fluor® dye conjugates contain(s) technology licensed to Abcam by Life Technologies.

Browse the full range of glutamate-related products at:
www.abcam.com/glutamate-products

Protocols and webinars

We are committed to supporting our customers through helpful resources including protocols and webinars. Highlighted below are a few examples of content relevant to glutamate research.

Protocols

Inducing plasticity of astrocytic receptors by manipulation of neuronal firing rates protocol

Our high quality products are used in this video protocol to induce homeostatic plasticity in neurons for the study of plasticity of astrocytic G-protein-coupled receptors (GPCRs). In this video you will learn:

- Acute hippocampal slice preparation
- Bolus loading of astrocytes with Ca^{2+} indicator
- Recording spontaneous and Gq GPCR agonist-evoked astrocytic Ca^{2+} activity in hippocampal slices

View this video protocol at: www.abcam.com/plasticity-protocol

Immunohistochemistry protocols

Are you new to immunohistochemistry or immunofluorescence? Perhaps you wish to try a new immunostaining method and don't know where to start? Our detailed IHC and ICC/IF protocols cover all aspects of specimen preparation for ICC/IF and IHC, whether you are staining paraffin-embedded, frozen or free-floating sections or whole tissue mounts.

Find these protocols at: www.abcam.com/IHC-protocols

Brain slice electrophysiology video protocol

Brain slice electrophysiology allows the study of a synapse or neural circuit in isolation from the rest of the brain, in controlled physiological conditions. This protocol describes the preparation of hippocampal slice and electrophysiology recording.
View this video protocol at: www.abcam.com/electrophysiology-protocol

View this video protocol at: www.abcam.com/electrophysiology-protocol

Find all our protocols at: www.abcam.com/protocols

Webinars

Investigation of Glutamate Receptors with Immunochemical Techniques Webinar

Review the advantages and limitations of different strategies applied for the production and testing of anti-GluR antibodies with Professor Elek Molnár.

Webinar Topics:

- Introduction to the molecular organization and regulation of GluRs, discussion of special challenges
- Choice of antigens for anti-GluR antibody preparation; their advantages and limitations
- Overview of some of the immunochemical approaches used for the investigation of GluRs
- Validation of antibodies and required controls for various immunochemical techniques
- Discussion of common problems and pitfalls in execution of these techniques or interpretation of the data.

Find this webinar at: www.abcam.com/glutamate-webinar

Find all our webinars at: www.abcam.com/webinars

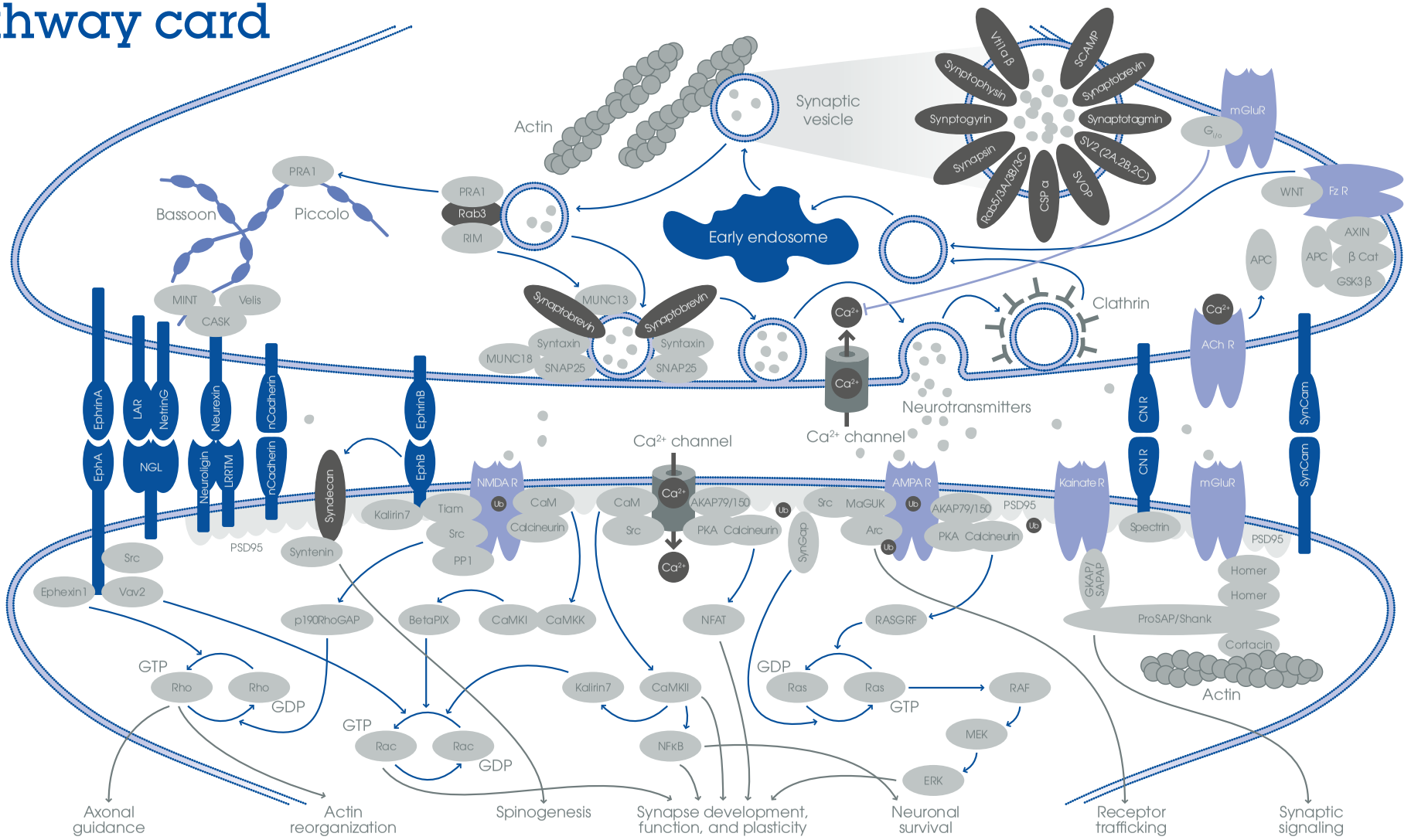
References and further reading

1. Purves, D. *et al.* in *Neuroscience* (eds. Purves, D. *et al.*) (Sinauer Associates, 2001).
2. Alexander, S. P. H. *et al.* The Concise Guide to Pharmacology 2013/14: Ligand-gated ion channels. *Br. J. Pharmacol.* **170**, 1706–1796 (2013).
3. Orth, A., Tapken, D. & Hollmann, M. The delta subfamily of glutamate receptors: Characterization of receptor chimeras and mutants. *Eur. J. Neurosci.* **37**, 1620–1630 (2013).
4. Traynelis, S. F. *et al.* Glutamate receptor ion channels: structure, regulation, and function. *Pharmacol. Rev.* **62**, 405–496 (2010).
5. Willard, S. S. & Koochekpour, S. Glutamate, glutamate receptors, and downstream signaling pathways. *Int. J. Biol. Sci.* **9**, 948–959 (2013).
6. Dingledine, R., Borges, K., Bowie, D. & Traynelis, S. F. The glutamate receptor ion channels. *Pharmacol. Rev.* **51**, 7–61 (1999).
7. Ju, W. *et al.* Activity-dependent regulation of dendritic synthesis and trafficking of AMPA receptors. *Nat. Neurosci.* **7**, 244–53 (2004).
8. Derkach, V. A., Oh, M. C., Guire, E. S. & Soderling, T. R. Regulatory mechanisms of AMPA receptors in synaptic plasticity. *Nat. Rev. Neurosci.* **8**, 101–13 (2007).
9. Van Den Bosch, L., Vandenberghe, W., Klaassen, H., Van Houtte, E. & Robberecht, W. Ca(2+)-permeable AMPA receptors and selective vulnerability of motor neurons. *J. Neurol. Sci.* **180**, 29–34 (2000).
10. Yao, Y., Belcher, J., Berger, A. J., Mayer, M. L. & Lau, A. Y. Conformational analysis of NMDA receptor GluN1, GluN2, and GluN3 ligand-binding domains reveals subtype-specific characteristics. *Structure* **21**, 1788–99 (2013).
11. Johnson, J. W. & Ascher, P. Voltage-dependent block by intracellular Mg2+ of N-methyl-D-aspartate-activated channels. *Biophys. J.* **57**, 1085–90 (1990).
12. Purves, D., Augustine, G. J. & Fitzpatrick, D. *Neuroscience, 4th Edition. Nature Reviews Neuroscience* (2008). doi:978-0878937257
13. Zhou, Q. & Sheng, M. NMDA receptors in nervous system diseases. *Neuropharmacology* **74**, 69–75 (2013).
14. Lerma, J. Roles and rules of kainate receptors in synaptic transmission. *Nat. Rev. Neurosci.* **4**, 481–495 (2003).
15. Bowie, D. Ion-dependent gating of kainate receptors. J. *Physiol.* **588**, 67–81 (2010).
16. Plested, A. J. R. Kainate receptor modulation by sodium and chloride. *Adv. Exp. Med. Biol.* **717**, 93–113 (2011).
17. Lerma, J. & Marques, J. M. Kainate receptors in health and disease. *Neuron* **80**, 292–311 (2013).
18. Niswender, C. M. & Conn, P. J. Metabotropic Glutamate Receptors: Physiology, Pharmacology, and Disease. *Annu Rev Pharmacol Toxicol* **50**, 295–322 (2010).
19. Alexander, S. P. H. *et al.* The Concise Guide to Pharmacology 2013/14: G protein-coupled receptors. *Br. J. Pharmacol.* **170**, 1459–581 (2013).
20. Pin, J. P., Galvez, T. & Prézeau, L. Evolution, structure, and activation mechanism of family 3/C G-protein-coupled receptors. *Pharmacol. Ther.* **98**, 325–354 (2003).
21. Pinheiro, P. S. & Mulle, C. Presynaptic glutamate receptors: physiological functions and mechanisms of action. *Nat. Rev. Neurosci.* **9**, 423–436 (2008).
22. Pin, J.-P. *et al.* Metabotropic glutamate receptors, introduction. *IUPHAR/BPS Guide to PHARMACOLOGY* (2015).

23. Jaeschke, G. *et al.* Metabotropic glutamate receptor 5 negative allosteric modulators: discovery of 2-chloro-4-(1-(4-fluorophenyl)-2,5-dimethyl-1H-imidazol-4-ylethynyl)pyridine (basimglurant, RO4917523), a promising novel medicine for psychiatric diseases. *J. Med. Chem.* **58**, 1358–71 (2015).
24. Hovelsø, N. *et al.* Therapeutic potential of metabotropic glutamate receptor modulators. *Curr. Neuropharmacol.* **10**, 12–48 (2012).
25. Swanson, C. J. *et al.* Metabotropic glutamate receptors as novel targets for anxiety and stress disorders. *Nat. Rev. Drug Discov.* **4**, 131–144 (2005).
26. Conn, P. J., Lindsley, C. W. & Jones, C. K. Activation of metabotropic glutamate receptors as a novel approach for the treatment of schizophrenia. *Trends Pharmacol. Sci.* **30**, 25–31 (2009).
27. Patil, S. T. *et al.* Activation of mGlu2/3 receptors as a new approach to treat schizophrenia: a randomized Phase 2 clinical trial. *Nat. Med.* **13**, 1102–7 (2007).
28. Nakajima, Y. *et al.* Molecular characterization of a novel retinal metabotropic glutamate receptor mGluR6 with a high agonist selectivity for L-2-amino-4-phosphonobutyrate. *J. Biol. Chem.* **268**, 11868–11873 (1993).
29. Mao, L., Guo, M., Jin, D., Xue, B. & Wang, J. Q. Group III metabotropic glutamate receptors and drug addiction. *Front. Med.* 7, 445–451 (2013).
30. Kenny, P. J. & Markou, A. The ups and downs of addiction: Role of metabotropic glutamate receptors. Trends *Pharmacol. Sci.* **25**, 265–272 (2004).

Excitatory synapse pathway card

abcam



NMDA Receptor

- Agonist: NMDA (ab120052)
- Antagonist: D-AP5 (ab120003)
- Primary antibody: Anti-NMDAR2B antibody (ab65783)

AMPA Receptor

- Agonist: (S)-AMPA (ab120005)
- Antagonist: NBQX (ab120045)
- Primary antibody: Anti-Glutamate Receptor 1 (AMPA subtype) antibody (EPR5479) (ab109450)

Kainate Receptor

- Agonist: Kainic acid (ab120100)
- Antagonist: CNQX disodium salt (ab120044)
- Primary antibody: Anti-GRIK2 antibody (EPR6307) (ab124702)

Group I mGlu receptor

- Agonist: (R,S)-3,5-DHPG (ab120020)
- Antagonist: MPEP hydrochloride (ab120008)
- Primary antibody: Anti-Metabotropic Glutamate Receptor 5 antibody (EPR2425Y) (ab76316)

Group II mGlu receptor

- Agonist: LY 379268 (ab120196)
- Antagonist: (S)-MCPG (ab120059)
- Primary antibody: Anti-Metabotropic Glutamate Receptor 2 antibody (EPR8975) (ab150387)

Group III mGlu receptor

- Agonist: L-AP4 (ab120002)
- Antagonist: MMPIP (120245)
- Primary antibody: Anti-Metabotropic Glutamate Receptor 4 antibody (ab53088)

Agonists, Antagonists, Activators and Inhibitors

NMDA receptor agonists and antagonists				
Name	Description	Purity	Citations	Product code
NMDA	Excitotoxic amino acid	> 99%	10	ab120052
D-AP5	NMDA glutamate site antagonist	> 99%	125	ab120003
AMPA receptor agonists and antagonists				
(S)-AMPA	AMPA agonist	> 98%	11	ab120005
NBQX	AMPA / kainate antagonist	> 99%	88	ab120045
Kainate receptor agonists and antagonists				
Kainic acid	Prototypic kainate receptor agonist	> 99%	31	ab120100
CNQX disodium salt	AMPA / kainate antagonist	> 99%	12	ab120044
Group I mGlu receptor agonists and antagonists				
(R,S)-3,5-DHPG	Group I mGlu receptor agonist	> 98%	3	ab120020
MPEP hydrochloride	Potent, selective mGlu5 antagonist	> 99%	21	ab120008
Group II mGlu receptor agonists and antagonists				
LY 379268	Systemically active group II mGlu agonist	> 99%	5	ab120196
(S)-MCPG	Group I / II mGlu antagonist	> 98%	9	ab120059
Group III mGlu receptor agonists and antagonists				
L-AP4	Selective group III mGlu agonist	> 99%	16	ab120002
MMPIP	mGlu7 allosteric antagonist	> 98%	2	ab120245

Primary Antibodies

NMDA receptor primary antibodies				
Name	Description	Tested applications	Species reactivity	Product code
Anti-NMDAR2B antibody ▲	Rabbit polyclonal to NMDAR2B	WB, IP, IHC, ICC/IF	Human, Mouse, Rat, Chicken, Xenopus	ab65783
AMPA receptor primary antibodies				
Anti-Glutamate Receptor 1 (AMPA subtype) antibody (EPR5479)	Rabbit monoclonal to Glutamate Receptor 1 (AMPA subtype) (EPR5479)	WB, IP, IHC	Human, Mouse, Rat	ab109450
Kainate receptor primary antibodies				
Anti-GRIK2 antibody (EPR6307)	Rabbit monoclonal to GRIK2 (EPR6307)	WB, IHC	Human, Mouse, Rat	ab124702
Group I mGlu receptor primary antibodies				
Anti-Metabotropic Glutamate Receptor 5 antibody (EPR2425Y) ▲	Rabbit monoclonal to Metabotropic Glutamate Receptor 5 (EPR2425Y)	WB, IHC, ICC/IF, Flow Cyt	Human, Mouse, Rat	ab76316
Group II mGlu receptor primary antibodies				
Anti-Metabotropic Glutamate Receptor 2 antibody (EPR8975)	Rabbit monoclonal to Metabotropic Glutamate Receptor 2 (EPR8975)	WB, IHC, ICC/IF	Human, Mouse, Rat	ab150387
Group III mGlu receptor primary antibodies				
Anti-Metabotropic Glutamate Receptor 4 antibody	Rabbit polyclonal to Metabotropic Glutamate Receptor 4	ELISA, WB, IHC, ICC/IF	Human, Mouse, Rat, Chicken, Cat	ab53088

Symbol key

Just-Add-Water: water soluble to at least 5mM or above Product also available as 1 ml water soluble pack Variation of this product available as 1 ml water soluble pack ▲ Blocking peptide available

Browse the full range of glutamate-related products at: www.abcam.com/glutamate-products