Revision 1

Store at -20C

#82760

Silent Synapses Antibody Sampler Kit

1 Kit (6 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
AMPA Receptor 1 (GluA1) (D4N9V) Rabbit mAb	13185	20 µl	100 kDa	Rabbit IgG
Phospho-AMPA Receptor 1 (GluA1) (Ser831) (A5O2P) Rabbit mAb	75574	20 µl	100 kDa	Rabbit IgG
Phospho-AMPA Receptor 1 (GluA1) (Ser845) (D10G5) Rabbit mAb	8084	20 µl	100 kDa	Rabbit IgG
AMPA Receptor 2 (GluA2) (E1L8U) Rabbit mAb	13607	20 µl	100 kDa	Rabbit IgG
PSD95 (D27E11) XP® Rabbit mAb	3450	20 µl	95 kDa	Rabbit IgG
NMDA Receptor 1 (GluN1) (D65B7) Rabbit mAb	5704	20 µl	120 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Silent Synapses Antibody Sampler Kit provides an economical means of detecting the activation of AMPA-type glutamate receptors (AMPAR) using phospho-specific and control antibodies. AMPARs expression can be compared to other synaptic components including NMDA-type glutamate receptor subunit GluN1 and the synaptic scaffolding protein PSD95. The kit includes enough antibody to perform two western blot experiments with each primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibodies.

Background

AMPA- (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid), kainate-, and NMDA- (N-methyl-D-aspartate) receptors are the three main families of ionotropic glutamate-gated ion channels. AMPA receptors (AMPARs) are composed of four subunits (GluA1-4), which assemble as homo- or hetero-tetramers to mediate the majority of fast excitatory transmissions in the central nervous system. AMPARs are implicated in synapse formation, stabilization, and plasticity (1). In contrast to GluA2-containing AMPARs, AMPARs that lack GluA2 are permeable to calcium (2). Post-transcriptional modifications (alternative splicing, nuclear RNA editing) and post-translational modifications (glycosylation, phosphorylation) result in a very large number of permutations, fine-tuning the kinetic properties and surface expression of AMPARs representing key pathways to mediate synaptic plasticity (3). During development and mature states, some synapses exhibit “silent synapses” that lack functional AMPAR-mediated transmission. Synapses become “unsilenced” by post-translational modification of GluAs, particularly GluA1, which alters its kinetic properties and/or surface expression while other synaptic components, such as other glutamate receptors like NMDARs and postsynaptic scaffolding proteins like PSD95, remain unaltered. Conversely, reducing the AMPAR kinetic properties and surface expression can silence synapses. Key post-translational modifications implicated in regulating these processes include phosphorylation of GluA1 at Ser831 and Ser845 (4). Research studies have implicated activity-dependent changes in AMPARs in a variety of diseases, including Alzheimer’s, amyotrophic lateral sclerosis (ALS), stroke, and epilepsy (1).

Background References

1. Palmer, C.L. et al. (2005) *Pharmacol Rev* 57, 253-77.
2. Cull-Candy, S. et al. (2006) *Curr Opin Neurobiol* 16, 288-97.
3. Huganir, R.L. and Nicoll, R.A. (2013) *Neuron* 80, 704-17.
4. Diering, G.H. et al. (2016) *Proc Natl Acad Sci U S A* 113, E4920-7.

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