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ProductInformation

Anti-VGLUT2 (KS-22)

Developed in Rabbit IgG Fraction of Antiserum

Product Number V 2639

Product Description

Anti-VGLUT2 (KS-22) is developed in rabbit using as immunogen a synthetic peptide corresponding to the C-terminus of rat VGLUT2 (amino acids 561-582) conjugated to KLH. The VGLUT2 sequence is highly conserved in mouse (90% identity) and human (72% identity) and has no homology to VGLUT1. Whole antiserum is fractionated and then further purified by ion-exchange chromatography to provide the IgG fraction of antiserum that is essentially free of other rabbit serum proteins.

Anti-VGLUT2 (KS-22) recognizes VGLUT2 (65 kDa) by immunoblotting. Staining of the VGLUT2 band in immunoblotting is specifically inhibited with the VGLUT2 immunizing peptide (amino acids 561-582).

Glutamate is the major excitatory neurotransmitter in the mammalian central nervous system (CNS). Packaging and storage of glutamate in glutaminergic neuronal vesicles requires an ATP-dependent vesicular glutamate uptake system, which utilizes the electrochemical proton gradient as a driving force. ^{2, 3} VGLUT1 (vesicular glutamate transporter 1, originally termed BNPI and characterized as a Na⁺/P_i transporter) is a 60 kDa protein responsible for vesicular glutamate uptake. 4-7 A second VGLUT isoform has been cloned, which has 82% identity to VGLUT1 and named differentiation-associated Na⁺/P_I transporter (DNPI) or VGLUT2 (65 kDa).⁸⁻¹¹ In contrast to the Na⁺-dependent plasma membrane glutamate transporters, VGLUT1 is highly specific for L-glutamate. It displays low substrate affinity (1 mM) and is stimulated by a physiologically relevant concentration of Cl ions.

VGLUT2 is a highly selective glutamate transporter, H⁺-dependent and requires Cl⁻ ions. Overexpression of VGLUT1/BNPI in mammalian cell lines results in transport of glutamate into isolated intracellular vesicles. VGLUT1 expression is confined to

subpopulations of glutaminergic axon terminals where it is exclusively localized on small synaptic vesicles.^{6, 7} VGLUT1-containing synaptic vesicles, immuno-isolated from rat brain are enriched in glutamate-uptake activity but display only marginal GABA uptake activity.6 VGLUT1-expressing cells release glutamate in a quantal manner monitored by reporter cells expressing a non-desensitizing variant of the AMPA glutamate receptor. In addition, GABAergic neurons in culture release glutamate in addition to GABA when VGLUT1 is exogenously expressed. 6 The mRNA of VGLUT2 displays a complementary distribution to VGLUT1. VGLUT2 is enriched in synaptic vesicles and selective for a distinct class of glutaminergic nerve terminals.¹² In general, VGLUT1 is associated with neuronal pathways that exhibit activity-dependent potentiation. whereas VGLUT2 is expressed in sensory and autonomic pathways that display high fidelity neurotransmission. VGLUT1 and VGLUT2 isoforms may account for glutamate uptake by synaptic vesicles from all glutaminergic neurons.

Reagent

Anti-VGLUT2 (KS-22) is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at -20 °C. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile

A minimum working dilution of 1:2,000 is determined by immunoblotting using an extract of synaptic vesicle (SV) fraction of rat brain.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilution by titration.

References

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