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ProductInformation

Anti-GABA(A) Receptor (α6 subunit)
Developed in Rabbit
Affinity Isolated Antibody

Product Number **G 5544**

Product Description

Anti-GABA(A) receptor α6 subunit (γ-aminobutyric acid receptor type A α6 subunit, GABRA6) is developed in rabbit using a highly purified peptide QLEDEGNFYSE-NVSRILDN corresponding to amino acid residues 20-37 of rat GABA(A) receptor α6 subunit with an additional C-terminal cysteine as the immunogen. The epitope is identical in mouse and rat and highly conserved in human (17/19 residues identical) antigens. The antibody was affinity isolated on immobilized immunogen.

Anti-GABA(A) receptor α6 subunit specifically recognizes the GABA(A) receptor α6 subunit protein and may be used for the detection of the GABA(A) receptor α6 subunit protein in rat brain membrane extracts by immunoblotting.

The inhibitory neurotransmitter GABA (γ-aminobutyric acid) signals through two distinct types of pre- and postsynaptic receptors, GABA(A) and GABA(B). Both GABA receptors can mediate depression of synaptic transmission and contribute to the inhibition controlling neuronal excitability.[1] GABA(A) and GABA(B) receptors differ with regard to their ionic characteristics and pharmacological properties. The GABA(A) receptor is an ionotropic receptor that forms the GABA gated chloride channel and consists of several heterogeneous subunits with membrane recognition sites for benzodiazapenes.[2] Over the past decade, a family of GABA(A) receptor subtypes has been delineated. These subtypes are generated by the co-assembly of five polypeptides selected from the α1-α6, β1-β3, γ1-γ3, δ, ε, π, and θ subunits.[3]

The gene transcripts and the polypeptides have distinct patterns of spatial expression such that the GABA(A) receptor subtypes have defined localizations that are presumed to reflect their physiological function. For example, serotonergic and GABAergic neurons selectively express distinct patterns of ⟨ subunits, suggesting they possess distinct GABA(A) receptor subtypes.[4] Serotonergic neurons express strong α3 immunoreactivity but show no α1 immunoreactivity. In contrast, GABAergic neurons express both α1 and α3 subunits. GABA(A) receptor subtypes also vary with respect to developmental expression patterns.[5] Developmental changes in the GABA(A) receptor subunit composition and the resulting pharmacology will be important in understanding the type of GABA-mediated transmission that takes place between neuronal contacts in the neonatal and, ultimately, the mature brain.

Reagents

Anti-GABA(A) receptor α6 subunit is supplied lyophilized from phosphate buffered saline, pH 7.4, with 1% bovine serum albumin and 0.05 % sodium azide as preservative.

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 hazardous and safe handling.

Preparation Instructions

Reconstitute the lyophilized vial with 0.05 ml or 0.2 ml deionized water, depending on the package size purchased. Antibody dilutions should be made in buffer containing 1-3 % bovine serum albumin. Centrifuge all antibody preparations before use (10000 x g 5 min).

Storage/Stability

Lyophilized powder can be stored intact at room temperature for several weeks. For extended storage, it should be stored at .20 °C or below. Once reconstituted, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in “frost-free” freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution

samples should be discarded if not used within 12 hours.

Product Profile

The recommended working dilution is 1-1.5 µg/ml (1:200-1:300) for immunoblotting.

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Note: In order to obtain best results and assay sensitivities of different techniques and preparations, we recommend determining optimal working dilutions by titration test.

References

1. Kerr, D.I. and Ong, J., Pharmacol. Ther., 67, 187-246 (1995).
2. Kostowski, W., Pol. J. Pharmacol., 47, 237-246 (1995).
3. Whiting, P.J. et al., Ann. N.Y. Acad. Sci., 868, 645-653 (1999).
4. Gao, B. et al., Neuroscience, 54, 881-892 (1993).
5. Carlson, B.X. et al., Eur. J. Pharmacol., 352, 1-14 (1998).

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