

Anti-GABA Transporter GAT-3

produced in rabbit, affinity isolated antibody

Catalog Number **G8407**

Anti-GABA Transporter GAT-3 is produced in rabbit using as immunogen a highly purified peptide corresponding to amino acid residues 607-627 of rat GAT-3. The antibody was affinity isolated using immobilized immunogen.

Anti-GABA Transporter GAT-3 recognizes GAT-3 protein from rat brain using immunohistochemistry on frozen or vibratome sections. The epitope is located in the predicted C-terminal domain of rat GAT-3.

γ -Aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the central nervous system and a neuromodulator in certain peripheral tissues.^{1,2} GABA is also present in non-nervous structures, and plays a major role in the regulation of blood pressure, decreases in heart rate and depression in respiration, effects on the immune system as well as modulation of cell proliferation, protein synthesis, and metabolism.³ GABA is formed following decarboxylation of L-glutamic acid by an enzyme, glutamic acid decarboxylase (GAD). GABA mediates neuronal inhibition by binding to the GABA/benzodiazepine receptor and opening an integral membrane chloride channel.

Several distinct transporters have been identified for the selective uptake of GABA. These include the GABA Transporters GAT-1, GAT-2, GAT-3 and BGT-1 transporters, in addition to the taurine transporter, a related family member. The homologous transporters each have a predicted molecular weight of 67 kDa and are predicted to contain 12 transmembrane regions. These proteins define a family of Na⁺ and Cl⁻ co-dependent transporters.^{4,5,6}

The γ -aminobutyric acid (GABA) transporter GAT-1 is the prototype of neurotransmitter transporters that includes those of dopamine and serotonin.⁷ GAT-1 maintains low synaptic concentrations of neurotransmitter by coupling GABA uptake to sodium and chloride fluxes.⁸ GAT-1 catalyzes the electrogenic transport of GABA with one chloride and two sodium ions.⁹

Inhibition of the re-uptake of GABA by potent and selective inhibitors of the GABA transporter enhances GABA activity. This property can be used therapeutically in epilepsy or psychiatric disorders.¹⁰

Reagents

Supplied lyophilized from 1% bovine serum albumin and $\leq 0.02\%$ sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Preparation Instructions

Reconstitute the lyophilized contents of the vial with 100 μ L sterile deionized water. Antibody dilutions should be made in buffer containing 1-3% bovine serum albumin.

Storage/Stability

Prior to reconstitution, store at -20°C . After reconstitution, the stock antibody solution may be stored at $2-8^{\circ}\text{C}$ for up to 2 weeks. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or 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

Antibody specificity was examined in the rat hippocampus and thalamus.¹¹ Suggested working dilution for frozen and vibratome sections is 1:4,000-1:8,000 for biotin-streptavidin/peroxidase detection. Low levels of glutaraldehyde (0.1%-0.3%) may be used in conjunction with paraformaldehyde for tissue perfusion fixation.

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. Erdo, S. and Kiss, B., In: GABAergic Mechanisms in the Mammalian Periphery, Erdo, S. and Bowery, N., (Eds.), Raven Press, New York, p. 5 (1986).
2. Erdo, S. and Wolff, J., *J. Neurochem.*, **54**, 363 (1990).
3. Okada, Y., In: GABAergic Mechanisms in the Mammalian Periphery, Erdo, S. and Bowery, N., (Eds.), Raven Press, New York, p. 223 (1986).
4. Kanner, B.I., *J. Exp. Biol.*, **196**, 237 (1994).
5. Borden, L.A., *Neurochem. Int.*, **29**, 335 (1996).
6. Miller, J.W. et al. 1997. Reith M.E.A. (Ed.). Humana Press, Totowa, N.J. pp. 101-149.
7. Uhl, G. R., *Trends Neurosci.*, **15**, 265 (1992).
8. Bennett, E.R. et al., *J. Biol. Chem.*, Aug., (2000) [in press].
9. Mager, S. et al., *Neuron*, **10**, 177 (1993).
10. Soudijn, W. and van Wijngaarden, I., *Curr. Med. Chem.*, **7**, 1063 (2000).
11. Minelli. A. et al., *J. Neurosci.*, **15**, 7734 (1995).

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