GAT-1 Antibody / GABA Transporter 1 / SLC6A1 (N-Terminal Region) (R32967)

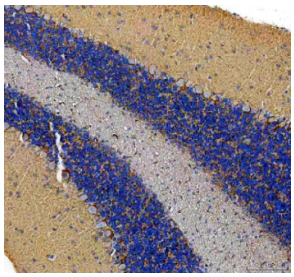
Catalog No.	Formulation	Size
R32967	0.5mg/ml if reconstituted with 0.2ml sterile DI water	100 ug

Bulk quote request

Availability	1-3 business days
Species Reactivity	Human, Mouse, Rat
Format	Antigen affinity purified
Host	Rabbit
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit IgG
Purity	Antigen affinity
Buffer	Lyophilized from 1X PBS with 2% Trehalose
UniProt	P30531
Localization	Membrane
Applications	Western Blot: 0.5-1ug/ml Immunohistochemistry (FFPE): 2-5ug/ml Immunofluorescence: 5ug/ml
Limitations	This GAT-1 antibody is available for research use only.



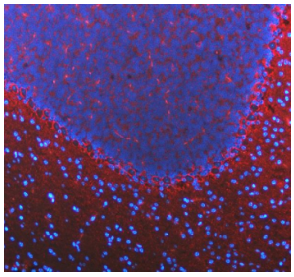
IHC staining of mouse cerebellum tissue with GAT-1 antibody, HRP-secondary and DAB substrate. Strong membrane associated brown labeling is observed along the periphery of the granular layer and in scattered neuronal processes, consistent with the expected distribution of the GABA transporter GAT-1. Hematoxylin counterstain shows nuclei in blue.



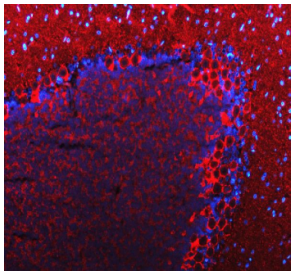
Immunohistochemical staining of FFPE rat cerebellum tissue with TCP1 antibody, HRP-secondary and DAB substrate. HIER: boil tissue sections in pH8 EDTA for 20 min and allow to cool before testing.



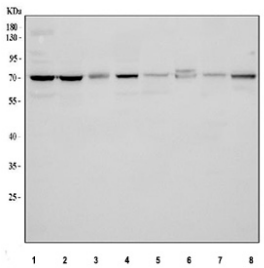
Immunohistochemical staining of FFPE rat brain tissue with TCP1 antibody, HRP-secondary and DAB substrate. HIER: boil tissue sections in pH8 EDTA for 20 min and allow to cool before testing.



Immunofluorescent staining of FFPE mouse cerebellum tissue with GAT-1 antibody (red) and DAPI nuclear stain (blue). HIER: steam section in pH8 EDTA buffer for 20 min.



Immunofluorescent staining of FFPE rat cerebellum tissue with GAT-1 antibody (red) and DAPI nuclear stain (blue). HIER: steam section in pH8 EDTA buffer for 20 min.



Western blot testing of 1) human HeLa, 2) human RT4, 3) human K562, 4) human U251, 5) rat brain, 6) rat C6, 7) mouse brain, and 8) mouse Neuro 2a cell lysate with GAT-1 antibody. Predicted molecular weight is approximately 67 kDa, and a single band was detected at approximately 70 kDa, consistent with the known glycosylation dependent migration pattern of SLC6A1/GAT-1 on SDS PAGE.

Description

GAT-1 antibody detects GABA Transporter 1, encoded by the SLC6A1 gene, a major neurotransmitter transporter responsible for the high-affinity reuptake of gamma-aminobutyric acid (GABA) from synaptic and extrasynaptic spaces. The UniProt recommended name is Sodium- and chloride-dependent GABA transporter 1. As one of the primary mechanisms controlling inhibitory neurotransmission in the central nervous system, GAT-1 plays an essential role in shaping synaptic strength, limiting tonic inhibition, modulating neuronal excitability, and maintaining the balance between excitation and inhibition in neural circuits.

SLC6A1 belongs to the solute carrier 6 family of sodium- and chloride-dependent neurotransmitter transporters. GAT-1 is

an integral membrane protein of approximately 599 amino acids with twelve transmembrane helices, extracellular glycosylation sites important for trafficking and stability, and intracellular regulatory regions that control transporter localization and function. By coupling GABA uptake to the electrochemical gradients of sodium and chloride, GAT-1 rapidly clears GABA following synaptic release, ensuring precise temporal control of inhibitory signaling.

The SLC6A1 gene is located on chromosome 3p25.3 and is widely expressed in the brain, including cortex, hippocampus, thalamus, basal ganglia, cerebellum, and spinal cord. GAT-1 is expressed predominantly in GABAergic interneurons and their presynaptic terminals, although it is also found in astrocytes and oligodendrocyte lineage cells in certain regions. This distribution highlights its dual contribution to synaptic and glial regulation of GABA levels. Neuronal GAT-1 controls phasic inhibition by limiting GABA availability after vesicular release, while glial GAT-1 helps regulate extracellular GABA tone that contributes to tonic inhibition.

During development, GAT-1 has important roles in neuronal maturation and the refinement of inhibitory circuits. Its expression increases as GABAergic synapses mature and as chloride gradients shift toward the classical hyperpolarizing inhibitory response. Proper timing and regulation of GAT-1 expression contribute to synaptic stabilization, network formation, and the establishment of inhibitory homeostasis. Disruptions in GAT-1 expression or function during development may alter circuit formation or increase susceptibility to neurological disorders.

In adult physiology, GAT-1 is central to inhibitory neurotransmission and neuronal network stability. By regulating the decay kinetics and spatial spread of GABA, GAT-1 shapes oscillatory activity, synaptic plasticity, and information processing in cortical and subcortical circuits. GAT-1 function is dynamically regulated by neuronal activity, phosphorylation events, membrane trafficking, and interactions with scaffolding proteins. Transporter internalization or reduced surface expression can enhance extracellular GABA levels, while increased surface expression strengthens uptake and limits inhibition.

Pathologically, altered SLC6A1 function is associated with a range of neurological and psychiatric disorders. Heterozygous loss-of-function variants in SLC6A1 cause SLC6A1-related neurodevelopmental disorder, typically characterized by epilepsy, myoclonic-atonic seizures, developmental delay, autism spectrum features, and cognitive impairment. Impaired GAT-1 activity leads to elevated extracellular GABA, disrupted circuit balance, and altered network oscillations. Mouse models of SLC6A1 dysfunction show increased tonic inhibition, impaired synaptic precision, and behavioral abnormalities that mirror human phenotypes.

GAT-1 dysregulation has also been implicated in epilepsy beyond monogenic syndromes. Reduced transporter function can promote excessive inhibition and paradoxically facilitate hyperexcitability in specific circuits. Altered GAT-1 expression has been reported in temporal lobe epilepsy, absence epilepsy, and seizure models where network dynamics depend heavily on GABA transporter function.

Beyond epilepsy, SLC6A1 expression changes have been linked to anxiety disorders, depressive phenotypes, schizophrenia, and substance use disorders. Because GAT-1 influences both phasic and tonic inhibition, changes in its expression may modulate stress responses, emotional regulation, and corticolimbic circuit function. In glial biology, GAT-1 contributes to interactions between neurons and astrocytes that regulate extracellular ion balance, neurotransmitter clearance, and metabolic coupling.

In pharmacology, GAT-1 is the target of tiagabine, a clinically used GABA reuptake inhibitor that increases extracellular GABA, enhances inhibition, and reduces seizure activity. Research on tiagabine and other transporter modulators continues to expand understanding of how GAT-1 controls inhibitory tone and provides therapeutic leverage points for neurological disease.

Because of these diverse roles, GAT-1 antibody is widely used in neuroscience research to examine expression patterns, regional distribution, developmental regulation, and disease-associated changes in SLC6A1 levels. Investigators use GAT-1 antibody to study inhibitory synapse organization, transporter trafficking, astrocyte-neuron interactions, and the effects of genetic or pharmacological perturbations on GABA transport. This antibody is validated for use in relevant

research applications to detect GABA Transporter 1 expression in cells and tissues. NSJ Bioreagents provides GAT-1 antibody reagents suitable for studies of neurotransmission, neurodevelopment, epilepsy, and inhibitory circuit regulation.

Application Notes

Optimal dilution of the GAT-1 antibody should be determined by the researcher.

Immunogen

Amino acids 23-54 (ANDKPKTLVVKVQKKAADLPDRDTWKGRFDFL) were used as the immunogen for the GAT-1 antibody.

Storage

After reconstitution, the GAT-1 antibody can be stored for up to one month at 4oC. For long-term, aliquot and store at -20oC. Avoid repeated freezing and thawing.

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