SANTA CRUZ BIOTECHNOLOGY, INC.

VGAT (F-2): sc-393373

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Synaptic transmission involves the controlled exocytosis of vesicles containing specific neurotransmitters. Usually, neurotransmitters are synthesized in the cytoplasm of the cell and must be transported into synaptic vesicles for release. The vesicular GABA transporter (VGAT) is responsible for loading γ-aminobutyric acid (GABA), an inhibitory neurotransmitter, from neuronal cytoplasm into synaptic vesicles and is expressed only in the nerve endings of inhibitory neurons that contain GABA and/or glycine. During neocortical development, VGAT expression barely precedes the maturation of inhibitory synaptogenesis, suggesting that it may contribute to the development of neocortical GABAergic circuitry. VGAT may also play a role in epileptogenesis and the recovery mechanisms that occur after a spontaneous seizure.

CHROMOSOMAL LOCATION

Genetic locus: SLC32A1 (human) mapping to 20q11.23; Slc32a1 (mouse) mapping to 2 H1.

SOURCE

VGAT (F-2) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 71-98 near the N-terminus of VGAT of human origin.

PRODUCT

Each vial contains 200 µg IgM kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-393373 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

VGAT (F-2) is recommended for detection of VGAT of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for VGAT siRNA (h): sc-61782, VGAT siRNA (m): sc-61783, VGAT siRNA (r): sc-270411, VGAT shRNA Plasmid (h): sc-61782-SH, VGAT shRNA Plasmid (m): sc-61783-SH, VGAT shRNA Plasmid (r): sc-270411-SH, VGAT shRNA (h) Lentiviral Particles: sc-61782-V, VGAT shRNA (m) Lentiviral Particles: sc-61783-V and VGAT shRNA (r) Lentiviral Particles: sc-270411-V.

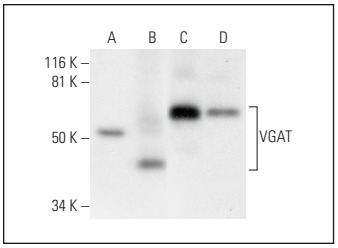
Molecular Weight of VGAT: 57 kDa.

Positive Controls: VGAT (h): 293T Lysate: sc-116499, mouse brain extract: sc-2253 or rat brain extract: sc-2392.

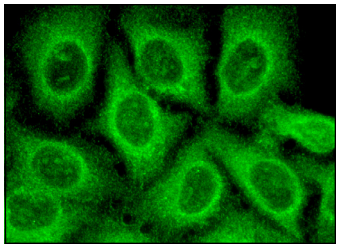
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



VGAT (F-2): sc-393373. Western blot analysis of VGAT expression in non-transfected: sc-117752 (**A**) and human VGAT transfected: sc-116499 (**B**) 293T whole cell lysates and mouse brain (**C**) and rat brain (**D**) tissue extracts.



VGAT (F-2): sc-393373. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Leshchyns'ka, I., et al. 2015. Aβ-dependent reduction of NCAM2-mediated synaptic adhesion contributes to synapse loss in Alzheimer's disease. Nat. Commun. 6: 8836.
2. Li, Y., et al. 2017. Synaptic adhesion molecule Pcdh-γC5 mediates synaptic dysfunction in Alzheimer's disease. J. Neurosci. 37: 9259-9268.
3. Zheng, J., et al. 2020. Interneuron accumulation of phosphorylated Tau impairs adult hippocampal neurogenesis by suppressing GABAergic transmission. Cell Stem Cell 26: 331-345.e6.
4. Tang, Y., et al. 2020. Increased GABAergic development in iPSC-derived neurons from patients with sporadic Alzheimer's disease. Neurosci. Lett. 735: 135208.
5. Lacaille, H., et al. 2021. Preterm birth alters the maturation of the GABAergic system in the human prefrontal cortex. Front. Mol. Neurosci. 14: 827370.
6. Aloi, M.S., et al. 2023. Microglia specific deletion of miR-155 in Alzheimer's disease mouse models reduces Amyloid-β pathology but causes hyperexcitability and seizures. J. Neuroinflammation 20: 60.
7. Martinovic, J., et al. 2023. Prolonged zaleplon treatment increases the expression of proteins involved in GABAergic and glutamatergic signaling in the rat hippocampus. Brain Sci. 13: 1707.
8. Su, M., et al. 2024. Synaptic adhesion molecule protocadherin-γC5 mediates β-Amyloid-induced neuronal hyperactivity and cognitive deficits in Alzheimer's disease. J. Neurochem. 168: 1060-1079.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.