bs-0325R

Rabbit Anti-GAD65 Polyclonal Antibody

Primary Antibodies

Background:
Glutamic Acid Decarboxylase (GAD) catalyzes the conversion of L glutamate to g-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the brain, and a putative paracrine signal molecule in pancreatic islets. GAD has a restricted tissue distribution. It is highly expressed in the cytoplasm of GABAergic neurons in the central nervous system (CNS) and pancreatic beta cells. It is also present in other non-neuronal tissues such as testis, oviduct and ovary. GAD is also transiently expressed in non-GABAergic cells of the embryonic and adult nervous system, suggesting its involvement in development and plasticity.
GAD exists as two isoforms, GAD65 and GAD67 (molecular masses of 65 and 67 kD, respectively) that are encoded by two different genes. GAD65 is an ampiphilic, membraneanchored protein, (585 amino acid residues) and is encoded on human chromosome 10. GAD67 is a cytoplasmic protein (594 amino acid residues) and is encoded on chromosome 2. There is 64% amino acid identity between the two isoforms, with the highest diversity located at the N terminus, which in GAD65 is required for targeting the enzyme to GABA-containing secretory vesicles. The two isoforms appear to have distinct intraneuronal distribution in the brain. GAD65 has been identified as an autoantigen in insulindependent diabetes mellitus (IDDM) and stiff-man syndrome (SMS), IDDM is an autoimmune disease that results from T cell mediated destruction of pancreatic insulin-secreting beta cells. Islet-reactive T cells and antibodies primarily to GAD65 (also named beta cell autoantigen) can be detected in peripheral blood of 80% of recent-onset IDD patients and in pre-diabetic high-risk subjects before onset of clinical symptoms. This suggests that GAD may be an important marker in the early stages of the disease. Also, autoantibodies to GAD65 and GAD67 are detected in animal models of IDDM, including the non-obese diabetes (NOD) mouse. In the NOD mouse, T cell reactivity is initially restricted to the C terminal regions of GAD65, but later spreads to other parts of GAD65. Stiff-man syndrome (SMS), a rare disorder of the CNS, is characterized by progressive rigidity of the body musculature with painful spasms, due to impairment of the GABAergic neurotransmission.
High-titer autoantibodies directed against GAD 65 and GABAergic neurons (nerve terminals) have been detected in the serum and cerebrospinal fluid (CSF) in 60% of patients with the syndrome. Strikingly, many of the SMS patients also developed late-onset IDDM.

Source/Purification:
KLH conjugated synthetic peptide derived from human GAD65. Was purified by Protein A and peptide affinity chromatography.

Storage: Prepared as lyophilized powder or liquid and shipped on ice. Store at -20℃ for one year.

Reconstitution:
If the antibody is in liquid form, no reconstitution needed.

Reconstitution is only required for the lyophilized antibody. Please refer to the reconstitution instruction card in the package.

Size: 100ul or 100ug lyophilized

Concentration: 1ug/uL

Host: Rabbit

Reactivities:
Human,Mouse,Rat,Chicken,Pig,

Application:

- WB(1:100-500)
- ELISA(1:500-1000)
- IP(1:20-100)
- IHC-P(1:100-500)
- IHC-F(1:100-500)
- IF(1:100-500)
- Not yet tested in other applications. Optimal working dilutions must be determined by the end user.

Antibody Type: Polyclonal

Isotype: IgG

Molecular Weight: 65kDa

Preservatives:
10ug/uL BSA and 0.1% NaN3.

For research use only. CAUTION: Not for human or animal therapeutic or diagnostic use.