

**Antibody to GAD65, B96**
HUMAN MONOCLONAL

Catalog Number: AB-N13
Quantity: 50 micrograms
Format: Purified on a Protein A column; PBS (0.14 M Sodium Chloride; 0.003 M Potassium Chloride; 0.002 M Potassium Phosphate; 0.01 M Sodium Phosphate; pH 7.4), no preservative.
Host: Human
Isotype: IgG₁ lambda light chain
Clone: B96
Immunogen: peripheral blood B cells immortalized with Epstein-Barr virus

Background: Antibodies to glutamic acid decarboxylase-65 (GAD65) are present in autoimmune disorders such as insulin-dependent (type1) diabetes mellitus (IDDM), stiff man syndrome, and polyendocrine autoimmune disease. Autoantibodies to GAD65 are present in 60-70% of individuals with newly diagnosed IDDM, and thus are important markers for disease activity. These autoantibodies usually recognize conformation-dependent regions on GAD65 and rarely bind to the second isoform, GAD67. Autoantibodies to GAD67 are found in only 15% of recent-onset IDDM patients, and most of this binding can be blocked with GAD65, suggesting shared epitopes between the two isoforms of GAD.

Specificity & Preparation: This antibody is specific for the 65-kDa isoform of glutamic acid decarboxylase (GAD65), targeting the IDDM-E2 region (amino acids 451-570), and does not bind GAD67. To create this cell line, peripheral blood B-cells were obtained from a donor who tested positive for GAD65 autoantibodies and were immortalized with Epstein-Barr virus.

Usage: Applications include immunoprecipitation (antibody supernatant 1:5,000-1:10,000)¹, immunohistochemistry¹, and protein footprinting (10 μ l serum).¹ This antibody does not work in Western blotting¹.

Storage: Store the antibody at -20°C for one year. Avoid repeated freezing and thawing. Gently spin down material 5-10 seconds in a microfuge before use.

**Selected References:**

1. Tremble J, Morgenthaler NG, Vlug A, Powers AC, Christie MR, Scherbaum WA, Banga JP (1997) Human B cells secreting immunoglobulin G to glutamic acid decarboxylase-65 from a nondiabetic patient with multiple autoantibodies and Graves' disease: a comparison with those present in type1 diabetes. *J Clin Endocrinol Metab* 82(8):2664-2670.

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