CYREX Laboratories

CLINICAL SPECIFICATIONS

GLUTAMIC ACID DECARBOXYLASE (GAD65)

Function:

Glutamic Acid Decarboxylase (GAD) a neuronal protein is an enzyme responsible for the conversion of the excitatory neurotransmitter glutamate to the inhibitory neurotransmitter γ-aminobutyric acid (GABA). GAD is also expressed by pancreatic beta cells.

Antibodies Appear:

Battan disease[6]
Celiac disease[3]
Cerebellar ataxia[4]
Gluten sensitivity[3]
Polyendocrine autoimmune syndrome[2]
Stiff-person syndrome[2]
Type 1 Diabetes[2, 4, 7]

Known Cross-Reactions: Casein;[1] Coxsackievirus;[5] Gliadin;[7] Rotavirus;[9] Cytomegalovirus;[10, 11] Rubella;[12] Buckwheat, Amaranth, Rice, Corn, Yeast, Potato, Quinoa, Oats[13]

Clinical Significance:

This enzyme is the major auto-antigen in Type I Diabetes. Researchers speculate that as a target antigen, GAD65 may directly, or indirectly, produce the T cell response cascade that results in insulin-dependent (type 1) diabetes mellitus.[7] In addition to patients with autoimmunity against islet cell antigen (Type I Diabetes), patients with neurological disorders (low GABA) may also produce high levels of antibodies against GAD.[2, 4, 6] Anti-GAD autoantibodies may result in an excess of excitatory neurotransmitters, which can lead to seizures.[5] Due to cross-reactivity between gliadin and casein,[1] patients with antibodies against GAD65 should implement a dairy-free diet. Additionally, in a study of Celiac patients,[3] 60% of the participants with Celiac disease produced GAD65, which may explain the relationship between Celiac disease and type-1 diabetes.

References:

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