CYREX® Laboratories

CLINICAL SPECIFICATIONS

21 HYDROXYLASE (ADRENAL CORTEX)

Function:

The Adrenal gland is the major gland for coping with stress. Its two parts consist of the adrenal medulla, which releases epinephrine and small amounts of norepinephrine, and the adrenal cortex, which releases epinephrine androgen hormones and corticosteroids including cortisol and aldosterone. This gland is the communicator between the body's brain and immune and metabolic system.

Antibodies Appear:

Addison's Disease[1, 2]
Adrenal Autoimmunity[1, 2]
Adrenal Insufficiency[1, 2]
Atrophic Gastritis[1]
Autoimmune Endocrine Disorders[1, 2]
Diabetes Insipidus[1]
Graves' Disease[1]
Hashimoto's Thyroiditis[1, 2]
Vitiligo[1]

Known Cross-Reactions: gliadin[5]

Clinical Significance:

There exists a high co-morbidity between Celiac disease and Addison's disease,[4] an illness resulting from a near total destruction of the adrenal cortex.[3] Thus, patients with Celiac should be tested for Addison's and vice versa. The cause of antibody production may be due to environmental factors such as bacterial or viral infections, or haptenic toxic chemicals binding to human tissue, causing modification of self-antigens and the subsequent production of autoantibodies. Antibodies against Adrenal Gland Antigens, specifically 21-hydroxylase, 17-hydroxylase and Cytochrome P-450 side chain cleavage assay, have been detected in patients with adrenal gland insufficiencies or failure (Addison's disease), or adrenal fatigue.[1, 2, 3] Antibodies associated with adrenalitis are considered predictive markers due to the detection of antibodies 5 to 10 years before the onset of disease.[2] Levels of adrenal autoantibodies correlate with the severity of adrenal dysfunction prior to the onset of the clinical condition.[2] These antibodies can predict progression to clinical disease and have been used to classify the cause of primary adrenal failure.[1]

References:

1. Betterle, et al. II. Adrenal cortex and steroid 21-hydroxylase autoantibodies in children with organ-specific autoimmune diseases: markers of high progression to clinical Addison's disease. J Clin Endocrinol Metab, 1997; 82:939-942.
2. Laureti, et al. Levels of adrenocortical autoantibodies correlate with the degree of adrenal dysfunction in subjects with preclinical Addison's disease. J clin Endocrinol Metab, 1998; 83:3507-3511.
3. Nigam, et al. Prevalence of adrenal antibodies in Addison's disease among North Indian Caucasians. Clin Endocrinol, 2003; 59:593-598.
4. O'Leary, et al. Coeliac disease and autoimmune Addison's disease: A clinical pitfall. Q J Med, 2002; 95:79-82.
5. Vojdani and Tarash. Cross-reaction between gliadin and different food and tissue antigens, Food Nutri Sci, 2013; 4:20-32.