CYREX® Laboratories

CLINICAL SPECIFICATIONS

NATIVE + DEAMIDATED α-GLIADIN-33-MER (saliva)

Function:

Gliadin is a glycoprotein. It is an alcohol-soluble protein present in wheat and occurring in various forms (α-, γ-, and ω-gliadins). α-Gliadin-33-mer is produced by natural digestion processes. It is resistant to proteolytic degradation and stimulates T cells.[6]

Antibodies Appear:

Celiac disease[1, 2, 3, 4, 5]

Known Cross-Reactions: 21 Hydroxylase, Asialoganglioside, Corn, Cytochrome P450, Dairy proteins, Glutamic Acid Decarboxylase, Myelin Basic Protein, Millet, Myocardial Peptide, Oats, Osteocyte, Ovary, Rice, Synapsin, Thyroid Peroxidase, Yeast;[9] Cerebellar[8, 9]

Clinical Significance:

Gliadin contains the toxic peptides associated with Celiac Disease (CD).[1] Assessing immune reactivity to a variety of wheat proteins increases the sensitivity for wheat/gluten reactivity. Detection of salivary antibodies to gliadin may indicate abnormal mucosal immune response and early onset of intestinal barrier dysfunction. Coupled with Transglutaminase-2 antibodies testing, Gliadin antibody assay results can assist with diagnosing potential CD and Gluten Reactivity (GR). If both are positive, the patient is at risk for developing CD. If Gliadin is positive and Transglutaminase-2 negative the patient could be suffering from GR. Epithelial translocation of α-gliadin-33-mer, and the subsequent uptake of 33-mer, is higher in untreated Celiac disease than in Celiac patients on a gluten-free diet.[7] In patients with active CD, CD71, a protein that is required for iron delivery, is overexpressed in the intestinal epithelium. In a recent study,[2] intestinal transport of 33-mer peptides was blocked by polymeric and secretory IgA (SIgA) and by soluble CD71 receptors. Retrotranscytosis of SIgA–gliadin complexes may therefore promote the entry of harmful gliadin peptides into the intestinal mucosa, and subsequently trigger an immune response and perpetuate intestinal inflammation.[2] Inflammation may trigger autoimmunity.

Suggested Reading:

1. Al-Bayaty, et al. Salivary and serum antibodies to gliadin in the diagnosis of celiac disease. J Oral Pathol Med, 1989; 18:578-581.
2. Matsiak-Budnik, et al. Secretory IgA mediates retrotranscytosis of intact gliadin peptides via the transferrin receptor in celiac disease. JEM, 2008; 205(1):143-154.
3. Hakeem, et al. Salivary IgA antigliadin antibody as a marker for coeliac disease. Arch Dis Childhood, 1992; 67:724-727.
4. Marinello, et al. Celiac disease screening: exploring the iceberg with salivary antigliadin antibodies. J Ped Gastroenterol Nutr, 2001; 32:227–228.
5. Matsiak-Budnik, et al. Secretory IgA mediates retrotranscytosis of intact gliadin peptides via the transferrin receptor in celiac disease. JEM, 2008; 205(1):143-154.
6. Qiao, et al. Antigen presentation to celiac lesion-derived T cells of a 33-mer gliadin peptide naturally formed by gastrointestinal digestion. J Immunol, 2004; 173:1757-1762.
7. Schumann, et al. Mechanisms of epithelial translocation of the α2-gliadin-33mer in coeliac sprue. Gut, 2008; 57:747-754.
8. Vojdani, et al. Immune response to dietary proteins, gliadin and cerebellar peptide in children with autism. Nutr Neurosci, 2004; 7(3):151-161.
9. Vojdani and Tarash. Cross-reaction between gliadin and different food and tissue antigens. Food Nutri Sci, 2013; 4:20-32.