CYREX® Laboratories

CLINICAL SPECIFICATIONS

NATIVE + DEAMIDATED α-GLIADIN-33-MER

Function:

Gliadin is a simple, alcohol-soluble peptide 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.[4]

Antibodies Appear:

Autism[6]
Celiac disease[1,2,3,4]
Celiac Sprue[5]
Wheat allergy[3]

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;[7] Cerebellar;[6,7] *Candida albicans*;[8] 55 kDa Nuclear autoantigen[9]

Clinical Significance:

Gliadin contains the toxic peptides associated with Celiac disease (CD).[1] Detection of antibodies to gliadin may indicate abnormal mucosal immune response and intestinal barrier dysfunction. Coupled with Transglutaminase-2 antibodies testing, Gliadin antibody assay results can assist with differentiating CD and non-celiac gluten sensitivity (NCGS). If both are IgA positive, the patient most likely has CD, which must be confirmed by biopsy. If Gliadin is positive and Transglutaminase negative the patient could be suffering from gluten-reactivity (GR) without enteropathy. If Transglutaminase is positive and Gliadin is negative the patient could be suffering from autoimmunity other than CD and GR. Epithelial translocation of α-Gliadin-33-mer, and the subsequent uptake of 33-mer, is higher in untreated CD than in Celiac patients on a gluten-free diet.[5] 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 triggering an immune response and perpetuating intestinal inflammation.[2] In a study measuring T cell responses to an array of wheat antigens, 33-mer and 17-mer elicited the strongest and most consistent responses.[1]

References:

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3. Palosuo. Update on wheat hypersensitivity. Curr Opin Allergy Clin Immunol, 2003; 3:205-209.
4. 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.
5. Schumann, et al. Mechanisms of epithelial translocation of the a2-gliadin-33 mer in coeliac sprue. Gut, 2008; 57:747-754.
6. Vojdani, et al. Immune response to dietary proteins, gliadin and cerebellar peptide in children with autism. Nutr Neurosci, 2004; 7(3):151-161.
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8. Corouge, et al. Humoral immunity links *Candida albicans* infection and celiac disease. PLoS One, 2015; 10(3):e0121776.
9. Natter, et al. IgA cross-reactivity between a nuclear autoantigen and wheat proteins suggests molecular mimicry as a possible pathomechanism in celiac disease. Eur J Immunol, 2001; 31:918-928.

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