MUCOSAL GLUTEN REACTIVITY SCREEN

ARRAY 2
INTESTINAL ANTIGENIC PERMEABILITY SCREEN

ARRAY 3
WHEAT/GLUTEN PROTEOME REACTIVITY AND AUTOIMMUNITY

ARRAY 4
GLUTEN-ASSOCIATED CROSS-REACTIVE FOODS AND FOODS SENSITIVITY

ARRAY 5
NEUROAUTOIMMUNITY PANEL

ARRAY 1

ORAL FLUID TEST

ARRAY 1 – Antibody

MUCOSAL GLUTEN REACTIVITY SCREEN™

- Evaluate overall mucosal immune system
- Screen for possible Gluten Sensitivity or Celiac disease
- Detect Gluten Sensitivity or Celiac Autoantibodies
- Identify defective oral tolerance
- A cost-effective test to monitor efficacy of treatment strategy

WHERE INNOVATION HAPPENS™

CYREX™ Laboratories

ARRAY 1

MUCOSAL GLUTEN REACTIVITY SCREEN™

- Secretory IgA
- Gliadin IgA + IgM combined
- Transglutaminase IgA + IgM combined

Oral Fluid Specimen

The mucosal layer of the gastrointestinal tract consists of a non-specific barrier (bacteria, gastric acid, mucus, defensins, and enzymes), and a specific immunological barrier (secretory IgA). These barriers act as a first line of defense against the intrusion of food antigens, pathogenic bacteria, and other environmental factors. Beneficial microbiota and secretory IgA protect gastrointestinal barriers against invasion by preventing antigen adhesion to the cell wall, which otherwise would be followed by eventual penetration into the gut's intrinsic layer.

Although mucosal surfaces are exposed to many dietary proteins and bacterial agents, the immune system normally will not react to these antigens. Unresponsiveness or tolerance to these antigens is maintained by three principal mechanisms: anergy (functional unresponsiveness), apoptosis (deletion through programmed cell death), and immune suppression by regulatory T-cells.

The breakdown of mucosal immune tolerance results in a gut-associated lymphoid tissue reaction to luminal antigens. This reaction may lead to the production of IgA and IgM antibodies against gliadin, tissue transglutaminase, and endogenous bacteria, as well as an increase of pro-inflammatory cytokines in oral fluid (saliva).

The resulting inflammatory conditions in the bowel can contribute significantly to gastrointestinal tissue damage and additional autoimmunity. It is important to know that when gliadin peptides are left undigested due to compromised laminal and brush border enzymes, they can be endocytosed and presented to the immune system. These special immune cells can then orchestrate an adaptive immune response to the gluten.

In healthy individuals, the gliadin peptide can be further digested during their transepithelial transport. Consequently, only minute amounts of intact peptides reach the villous lamina propria. The presence of gliadin peptides can drive a tolerogenic immune response and initiate the production of IgA in both Peyer's patches and mesenteric lymph nodes. In healthy individuals, dietary peptides are complexed to secretory IgA, and thus, are trapped in the mucosal layer, where they are prevented from attaching to the intestinal wall. In patients with Celiac disease, up-regulation of gut-specific receptors and their abnormal expression at the apical surface of enterocytes allows the luminal capture of IgA-gliadin complexes. These complexes enter enterocytes with gut receptors through the endosomal recycling pathway, which quickly delivers the intact complex into the basolateral compartment. This entry of large amounts of gliadin peptides ignites a localized adaptive inflammatory response.

The elevation of the gliadin antibody in secretion contributes to enhanced transportation of undigested gliadin peptides to the submucosa. This contributes to a massive expansion of intraepithelial lymphocytes, and a cytolytic attack of the epithelium on one hand, and the transfer of gliadin antigens to the regional lymph nodes and into circulation, on the other hand. Such an immune response against gliadin peptides results in IgG and IgA antibody production in the blood, the measurement of which is the laboratory hallmark of Celiac disease.

Children and adults who are suspected of gluten intolerance should be screened first by gliadin and transglutaminase IgA + IgM in oral fluid. If the results of gliadin or transglutaminase do not correlate with each other; for example, the salivary gliadin IgA + IgM are positive, but the transglutaminase antibody is negative, or both antibodies are borderline elevated, then a more comprehensive evaluation of an immune reaction to transglutaminase and the peptides of wheat should be performed, available as Cyrex's Wheat/Gluten Proteome Reactivity & Autoimmunity (Array #3). Confirmation of a typical villous atrophy of the duodenal mucosa remains the gold standard confirmation of Celiac disease.

Dysregulation of the mucosal barrier, which occurs before antigenic penetration of the gastrointestinal barrier and subsequent systemic immune response, can be assessed by measuring total secretory IgA and IgA + IgM antibodies against gliadin and transglutaminase in oral fluid.

Recommended for patients who:

- Are suspected of having mucosal abnormalities
- Have relatives with Gluten Sensitivity or Celiac disease
- Have Type 1 Diabetes
- Have Down syndrome
- Have autoimmune disorders
- Have a family history of autoimmune disorders

Specimen Requirement:
2 mL oral fluid

For the Clinical Application Guide, please visit www.CyrexLabs.com