CYREX® Laboratories

CLINICAL SPECIFICATIONS

ROTAVIRUS

Pathogen Type:

Human rotaviruses belong to the family of *Reoviridae*. Rotavirus is a double-stranded RNA virus that is commonly associated with gastroenteritis in children.

Associated With:

Gastroenteritis[1]
Celiac disease[2]
Type 1 diabetes[3]
Uveitis[4]

Known Cross-Reactions: Celiac peptide;[5] glutamic acid decarboxylase-65, tyrosine phosphatase;[3] retinal S-antigen;[4] *Parietaria officinalis* pollen allergen of *Urticaceae* (a weed)[6]

Clinical Significance:

The detection of antibodies to Rotavirus indicates the patient has increased risk of gastrointestinal disorders, type 1 diabetes, eye autoimmunity. Infection with rotavirus, a double-stranded RNA virus, is commonly associated with antibody production and can occasionally spread beyond the gastrointestinal tract, causing systemic infection and strong immune response against the virus.[7] While infection with rotavirus may subside after several days or weeks, the immune response against the virus may continue and hence antibodies are detected in blood for a long period of time. Upon replication in intestinal tissue, rotaviruses are able to alter the gastrointestinal environment. This alteration can lead to intestinal inflammation, breaking of intestinal tight junction structures and increased intestinal permeability to large molecules. Due to cross-reactivity with extraintestinal tissue proteins, Rotavirus infection may lead to autoimmunity beyond the gut.

This array tests for IgG immune reactivity associated with Rotavirus. This is not a measurement of acute infection. Equivocal or out-of-range results indicate IgG antibody reactivity to the tested antigen. We tested 288 blood donor sera against Rotavirus antigens at optimal dilution, 10% of these donors were IgG reactive.

References:

1. Lin, et al. Disease caused by rotavirus infection. Open Virol J, 2014; 8:14-19.
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3. Honeyman, et al. Evidence for molecular mimicry between human T cell epitopes in rotavirus and pancreatic islet autoantigens. J Immunol, 2010; 184(4):2204-2210.
4. Wildner and Diedrichs-Möhring. Autoimmune uveitis induced by molecular mimicry of peptides from rotavirus, bovine casein and retinal S-antigen. Eur J Immunol, 2003; 33:2577-2587.
5. Kagnoff, et al. Possible role for a human adenovirus in the pathogenesis of Celiac disease. J Exp Med, 1984; 160:1544-1557.
6. di Somma, et al. Cross-reactivity between the major Parietaria allergen and rotavirus VP4 protein. Allergy, 2003; 58:503–510.
7. Nakano, et al. Sudden death from systemic rotavirus infection and detection of nonstructural rotavirus proteins. J Clin Microbiol, 2011; 49(12):4382-4385.