CYREX® Laboratories

CLINICAL SPECIFICATIONS

LIPOPOLYSACCHARIDE (saliva)

Function:

Lipopolysaccharide (LPS) is a molecule made up of a lipid and a polysaccharide. LPS is a component of the surface membrane of gram-negative bacteria found in the gastrointestinal tract. Gram-negative bacteria include: *Escherichia coli*, *Salmonella*, *Shigella*, *Pseudomonus*, *Helicobactor*, *Legionella*, *Wolbachia*. As an endotoxin, LPS increases the negative charge of the bacterial membrane and promotes the upregulation of pro-inflammatory cytokines.[4]

Antibodies Appear:

Gram-negative bacterial infection[6]
Hemolytic uremic syndrome[1, 2]
Increase intestinal permeability[4]
Typhoid fever[3, 8]

Known Cross-Reactions: DNA-histone,[7] Ganglioside,[5] Antiphospholipid antibodies[9]

Clinical Significance:

Lipopolysaccharides (LPS) are bacterial endotoxins that elicit a strong immune response. The detection of salivary antibodies against LPS indicates mucosal immune reactivity to LPS. Upregulated antibodies to LPS indicate gut dysbiosis.[6] If the patient is experiencing recent-onset high fever, weakness and stomach pains, or has bloody diarrhea, the elevated salivary LPS may be an indication of Typhoid fever or *Escherichia coli* infection respectively. In an *S typhi* LPS study, researchers found the level of salivary anti-LPS IgA was significantly higher in typhoid cases compared to febrile and healthy control and also revealed that the maximum sensitivity of salivary anti-LPS IgA are in the second and third weeks of illness, with the levels falling thereafter.[8] Salivary IgA and IgM antibodies were found to decline to their normal levels 45 to 90 days after the acute phase.[3] Studies on *Escherichia coli* infections showed greater specificity and sensitivity of salivary IgA over serum antibodies during the acute phase of hemolytic uremic syndrome.[1, 2]

Suggested Reading:

1. Ludwig, et al. Saliva IgM and IgA are a sensitive indicator of the humoral immune response to *Escherichia coli* O157 lipopolysaccharide in children with enteropathic hemolytic uremic syndrome. Pediatr Res, 2002; 52:307–313.
2. Chart, et al. Analysis of saliva for antibodies to the LPS of *Escherichia coli* O157 in patients with serum antibodies to E. coli O157 LPS. J Med Microbiol, 2003, 52:569–572.
3. Herath. Early diagnosis of typhoid fever by the detection of salivary IgA. J Clin Pathol, 2003;56:694–648.
4. Maes, et al. The gut-brain barrier in major depression: intestinal mucosal dysfunction with an increased translocation of LPS from gram negative enterobacteria (leaky gut) plays a role in the inflammatory pathophysiology of depression. Neuroendocrinol Lett, 2008; 29(1):117-124.
5. Neisser, et al. Serum antibodies against gangliosides and *Campylobacter jejuni* lipopolysaccharides in Miller Fisher Syndrome. Infect Immunity, 1997; 65(10):4038-4042.
6. Poxton, et al. Antibodies to lipopolysaccharide.Immunol Methods, 1995; 186:1-15.
7. Sumazaki, et al. Monoclonal antibody against bacterial lipopolysaccharide cross-reacts with DNA-histone. Clin exp Immunol, 1986; 66:103-110.
8. Zaka-ur-Rab, et al. Evaluation of salivary anti-*Salmonella typhi* lipopolysaccharide IgA ELISA for serodiagnosis of typhoid fever in children. Arch Dis Child, 2012; 97(3):236-238.
9. Cheng, et al. IgA antiphospholipid antibodies in normal human saliva cross-react with bacterial lipopolysaccharide. Int Arch Allergy Immunol, 1993; 101:297-298.

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