Cyrex™ Laboratories

LIPOPOLYSACCHARIDE

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.[1 6]

ANTIBODIES APPEAR:

Chronic fatigue syndrome[2]
Gram-negative bacterial infection[4]
Increase intestinal permeability[1 2]
Major depression[1]
Miller Fisher syndrome[3]
Short bowel syndrome[3]

KNOWN CROSS REACTIONS:

DNA-histone,[5] Ganglioside[3]

CLINICAL SIGNIFICANCE:

Lipopolysaccharides (LPS) is a bacterial endotoxin that elicits a strong immune response.[4] The detection of antibodies against LPS indicates infiltration of macromolecule-sized endotoxins into the intestinal barrier and the systemic circulation. For better clinical evaluations, LPS should be measured in conjunction with antibodies against tight junction proteins, occludin/zonulin, and epithelial structure proteins from the actomyosin network. If antibodies to LPS alone are elevated while antibody levels for occludin/zonulin and actomyosin are negative, the patient may have gut flora dysbiosis. When both LPS and occludin/zonulin antibodies are positive and actomyosin antibody is not detected, there is likely a breakdown in intestinal barrier integrity caused by infiltration of bacterial antigens through the paracellular pathway. Results showing elevations in LPS and actomyosin antibody levels, but not in occludin/zonulin, indicate a high possibility of breakdown in the intestinal barrier integrity by bacterial antigens through the transcellular pathway.

References:

1. Maes M, 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.
2. Maes M, et al. Increased serum IgA and IgM against lipopolysaccharide of enterobacteria in chronic fatigue syndrome (CFS): indication of the involvement of gram-negative enterobacteria in the etiology of CFS and for the presence of an increased gut-intestinal permeability. J Affect Disord, 2007; 99(1-3):237-240.
3. Neisser A, et al. Serum antibodies against gangliosides and Campylobacter jejuni lipopolysaccharides in Miller Fisher Syndrome. Infect Immunity, 1997; 65(10):4038-4042.
4. Poxton IR, et al. Antibodies to lipopolysaccharide. J Immunol Methods, 1995; 186:1-15.
5. Sumazaki R, et al. Monoclonal antibody against bacterial lipopolysaccharide cross-reacts with DNA-histone. Clin exp Immunol, 1986; 66:103-110.
6. Ziegler TR, et al. Detectable serum flagellin and lipopolysaccharide and upregulated anti-flagellin and lipopolysaccharide immunoglobulins in human short bowel syndrome. Am J Physiol Regul Integr Comp Physiol, 2008; 294:R402-R410.

5040 N. 15th Avenue, Suite 107, Phoenix, AZ 85015 | Tel 602.759.1245 | Fax 602.759.8331 | **www.CyrexLabs.com**