CYREX® Laboratories

CLINICAL SPECIFICATIONS

CAMPYLOBACTER JEJUNI

Pathogen Type:

Campylobacter jejuni (*C. jejuni*) is a gram-negative and microaerophilic bacterium commonly found in animal feces. *C. jejuni* infection is one of the most commonly identified bacterial causes of acute gastroenteritis worldwide.

Associated With:

Diarrheal disease[1]
Campylobacter-induced Guillain-Barré syndrome[2,3]
Campylobacter-induced Miller Fisher syndrome[3]

Known Cross-Reactions: *C. jejuni* Lipopolysaccharides (LPS) and lipo-oligosaccharides (LOS) with asialoganglioside[2]

Clinical Significance:

The detection of antibodies to *C. jejuni* indicates the patient may have increased risk of bowel disorders, neurological disorders and arthritis. Typically, infection with *C. jejuni* results in an acute, gastrointestinal illness characterized by diarrhea, fever, and abdominal cramps and resolves without clinical intervention.[1] In persons with compromised immune systems, *Campylobacter* occasionally spreads to the bloodstream and causes a serious life-threatening infection. Extraintestinal manifestations of *C. jejuni* infection are rare and may include meningitis, endocarditis, septic arthritis, osteomyelitis, neonatal sepsis[1] and even Guillain-Barré syndrome (GBS)[2,3] or Miller Fisher syndrome.[3] Molecular mimicry between microbial antigens and human tissue and in this particular case, between *C. jejuni* and ganglioside, serve as the causative mechanism for GBS.[4,5] Due to this mimicry, antibodies, autoreactive T-helper cells, singly or in combination, can be induced by infection in order to destroy the invading pathogen, but also attack the host tissue (ganglioside).[6] The majority of GBS patients report or present symptoms of an infectious disease such as watery diarrhea, weeks preceding neurological symptoms associated with GBS.[3,7] This short interval between the acute infection and presentation of symptoms enable identification of the triggering pathogenic agents.

This array tests for IgG immune reactivity associated with *Campylobacter jejuni*. 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 *C. jejuni* antigens at optimal dilution, 15% of these donors were IgG reactive.

References:

1. Allos. *Campylobacter jejuni* infections: update on emerging issues and trends. Clin Infect Dis, 2001; 32:1201–1206.
2. Yuki, et al. A bacterium lipopolysaccharide that elicits Guillain-Barré syndrome has a GM1 ganglioside–like structure. J Exp Med, 1993; 178:1771–1775.
3. Yuki and Koga. Bacterial infections in Guillain–Barré and Fisher syndromes. Curr Opin Neurol, 2006; 19:451–457.
4. Ang, et al. The Guillain-Barré syndrome: a true case of molecular mimicry. Trends Immunol, 2004; 25(2):61-66.
5. Yu, et al. Ganglioside molecular mimicry and its pathological roles in Guillain-Barré syndrome and related diseases. Infect Immun, 2006; 74(12):6517-6527.
6. Huizinga, et al. Sialylation of *Campylobacter jejuni* endotoxin promotes dendritic cell-mediated B cell responses through CD14-dependent production of IFN-β and TNF-α. J Immunol, 2013; 191(11):5636-5645.
7. Ropper. *Campylobacter* diarrhea and Guillain-Barré syndrome. Arch Neurol, 1988; 45(6):655-656.