CYREX® Laboratories

CLINICAL SPECIFICATIONS

CLOSTRIDIUM DIFFICILE

Pathogen Type:

Clostridium difficile (*C. difficile*) is a spore-forming gram-positive bacterium, which infects and colonizes the large intestine.

Associated With:

Colitis[1]
Megacolon[2]
Ileus[2]
Irritable bowel disorder[3]
Ulcerative colitis[3]
Crohn's disease[3]

Known Cross-Reactions:

Clinical Significance:

The detection of antibodies to *C. difficile* indicates the patient has increased risk of gastrointestinal disorders including irritable bowels, ulcerative colitis and Crohn's disease. *C. difficile* is a spore-forming gram-positive anaerobic bacillus, and the leading cause of antibiotic-associated nosocomial diarrhea and colitis in the industrialized world.[4] *C. difficile* infection has been around for more than 30 years, and is most often acquired in hospital settings.[5] *C. difficile* produces potent toxins, triggers inflammation, and causes significant systemic complications.[6] The use of stomach acid blockers allow *C. difficile* spores to transit through the stomach into the gut, where the anaerobic environment and the presence of bile salts, allows the spores to germinate into the toxin-producing vegetative state.[7,8] Studies have shown an increase in the prevalence and severity of *C. difficile* infection among inflammatory bowel disease (IBD) patients and patients with IBD are more likely to have serum antibodies to *C. difficile* toxin B.[3] Measurement of IgG antibody against *C. difficile* is highly recommended in patients before, during or after hospitalization in order to determine colonization with this bacterium.[9]

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

References:

1. Bartlett, et al. Antibiotic-associated pseudomembranous colitis due to toxin-producing Clostridia. N Engl J Med, 1978; 298(10):531-534.
2. Rupnik, et al. *Clostridium difficile* infection: new developments in epidemiology and pathogenesis. Nat Rev Microbiol, 2009; 7:526-536.
3. Shakir, et al., Determination of serum antibodies to *Clostridium difficile* toxin B in patients with inflammatory bowel disease. Gastroenterol Hepatol (N Y), 2012; 8(5):313–317.
4. Bartlett. Narrative review: the new epidemic of *Clostridium difficile*-associated enteric disease. Ann Intern Med 2006; 145: 758–764.
5. Musa, et al. *Clostridium difficile* infection and liver disease. J Gastrointestin Liver Dis, 2010; 19(3):303-310.
6. Khanna and Pardi. The growing incidence and severity of *Clostridium difficile* infection in inpatient and outpatient settings. Expert Rev Gastroenterol Hepatol, 2010; 4(4):409-416.
7. Tleyjeh, et al. Association between proton pump inhibitor therapy and *Clostridium difficile* infection: a contemporary systematic review and meta-analysis. PLoS ONE, 2012; 7:e50836.
8. Francis, et al. Bile acid recognition by the *Clostridium difficile* germinant receptor, CspC, is important for establishing infection. PLoS Pathog, 2013; 9:e1003356.
9. Ghose. *Clostridium difficile* infection in the twenty-first century. Emerging Microbes and Infections, 2013; 2:e62.