CYREX® Laboratories

CLINICAL SPECIFICATIONS

CAMPYLOBACTER JEJUNI CDT A + B

Function:

Campylobacter jejuni (*C. jejuni*) cytolethal distending toxin (CDT) subunits A and B are endotoxins released by the gut pathogen. Non-pathogenic CdtA is utilized by pathogenic CdtB to infiltrate intestinal epithelial cells. Inside the cell, CdtB contributes to cytoskeletal damage, which may induce apoptosis (cell death). CdtB is the first bacterial toxin known to act in the nucleus of a target cell.

Associated With:

Irritable bowels[1]
D-IBS[1]
SIBO[1,2]
Gut dysbiosis[1]
Chronic functional bowel changes[2]
Macrophage, T- and B-cell dysfunction[3]

Known Cross-Reactions: $A\beta_{42}$ peptide;[4] Deoxyribonuclease-1 (DNase-1);[5] Interstitial cells of Cajal, Enteric neurons[6]

Clinical Significance:

C. jejuni is a member of the bacteria group that participates in diseases involving the disruption of a mucosal or epithelial layer. *C. jejuni* has been shown to promote inflammation by the induction of inflammatory cytokines such as interleukin-8 (IL-8), which promotes increased intestinal permeability.[reviewed in 7] Additionally, *C. jejuni* CDT damages the intestinal barrier by causing epithelial cell apoptosis.[reviewed in 7] With the barrier broken, *C. jejuni* CDTs find their way to the submucosa, regional lymph nodes, and into the circulation, where the immune system responds by producing antibodies against them. These antibodies along with the release of inflammatory cytokines put the blood-brain barrier at risk of damage.[4] Once the BBB is broken, circulating autoantigens can enter the brain and nervous system, triggering neuroautoimmune reactivity.[8] Due to the reactivity of $A\beta_{42}$ peptide antibody with *E. coli*, *Salmonella*, and in particular with *C. jejuni* CdtB, patients with circulating antibodies to *C. jejuni* CDT may be at a greater risk for Alzheimer's disease and other neurological disorders.[4]

References:

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3. Scuron et al. The cytolethal distending toxin contributes to microbial virulence and disease pathogenesis by acting as a tri-perditious toxin. Front Cell Infect Microbial, 2016; 6:168.
4. Vojdani et al. Reaction of amyloid-peptide antibody with different infectious agents involved in Alzheimer's disease. J Alzheimer's Dis, 2018; 63:847-860.
5. Guerra et al. The biology of the cytolethal distending toxins. Toxins, 2011; 3:172-190.
6. Venkata et al. Antibodies to *Campylobacter jejuni* cytolethal distending toxin subunit B (CDT-B) bind enteric neural elements in uninfected mice suggesting molecular mimicry. Gastroenterology, 2011; 140(5, Suppl 1):S521.
7. Faïs et al. Impact of CDT toxin on human diseases. Toxins (Basel), 2016; 8(7)pii:E220.
8. Vojdani. Brain-reactive antibodies in traumatic brain injury. Funct Neurol Rehabil Ergon, 2013; 3(2-3):173-181.

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