

## CLINICAL SPECIFICATIONS

# HELICOBACTER PYLORI

### Pathogen Type:

*Helicobacter Pylori* (*H. pylori*) is a gram-negative microaerophilic bacterium usually found in the stomach, but may be present in other parts of the human body. *H. pylori* was previously named *Campylobacter pylori*.

### Associated With:

Chronic superficial gastritis<sup>1,2</sup>  
 Type B atrophic gastritis<sup>1</sup>  
 Gastric ulcers<sup>1,2</sup>  
 Duodenal ulcers<sup>1,2</sup>  
 Ischemic heart disease<sup>3,4</sup>  
 Type 1 diabetes<sup>5,6</sup>  
 Thyroid disease<sup>7</sup>

**Known Cross-Reactions:** Parietal cells;<sup>8</sup> autoimmune pancreatitis peptide and ubiquitin-protein ligase E3 component n-recognin 2;<sup>9</sup> Thyroid Peroxidase (TPO), Gastric Epithelial Cells<sup>10</sup>

### Clinical Significance:

The detection of antibodies to *H. pylori* indicates the patient has increased risk of gastrointestinal disorders, neurological disorders, rheumatic diseases, thyroid autoimmunity and lupus. Two major groups of *H. pylori* have been classified.<sup>1</sup> The maintenance group colonizes and remains in within the host. Indeed, greater than 80% of individuals infected with *H. pylori* are asymptomatic and thus the bacterium can be considered to play an important role in the gastrointestinal biome.<sup>11</sup> The virulence group produces a specific enzyme, CagA, which contributes to the pathogenic effects of the bacterium. The pathogenic effects of the virulence group includes gastric inflammation, disruption of the gastric mucosal barrier, and alteration of gastric physiology.<sup>1</sup> The most common etiological cause for autoimmune thyroiditis is infection with *H. pylori* and *Yersinia enterocolitica*. Bacterial antigen and thyroid tissue cross-reactivity has been proposed as a mechanism of *H. pylori*-induced autoimmune thyroid disorders.<sup>10</sup> Systemic inflammation caused by *H. pylori* can lead to extra-intestinal autoimmunities effecting heart, pancreas and thyroid tissues.

This array tests for IgG immune reactivity associated with Helicobacter Pylori. 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 H. pylori antigens at optimal dilution, 12% of these donors were IgG reactive.

### References:

1. Dunn, et al. *Helicobacter pylori*. Clin Microbiol Rev, 1997; 10(4):720-741.
2. Kusters, et al. Pathogenesis of *Helicobacter pylori* infection. Clin Microbiol Rev, 2006; 19(3):449-490.
3. Danesh, et al. *Helicobacter pylori* infection and early onset myocardial infarction: case-control and sibling pairs study. BMJ, 1999; 319:1157-1162.
4. Pietroiusti, et al. Cytotoxin associated gene-A-positive *Helicobacter pylori* strains are associated with atherosclerotic stroke. Circulation, 2002; 106:580-584.
5. Salardi, et al. *Helicobacter pylori* and type 1 diabetes mellitus in children. J Pediatr Gastroenterol Nutr, 1999; 28:307-309.
6. Coker, et al. *Helicobacter pylori* seropositivity in children with diabetes mellitus type 1. J Trop Pediatr, 2001; 47:123-124.
7. Larizza, et al. *Helicobacter pylori* infection and autoimmune thyroid disease in young patients: the disadvantage of carrying the human leukocyte antigen-DRB1\*0301 Allele. J Clin Endocrinol Metabol, 2006; 91(1):176-179.
8. Claeys, et al. The gastric H<sup>+</sup>,K<sup>+</sup>-ATPase is a major autoantigen in chronic *Helicobacter pylori* gastritis with body mucosa atrophy. Gastroenterology, 1998; 115:340-347.
9. Frulloni, et al. Identification of a novel antibody associated with autoimmune pancreatitis. N Engl J Med, 2009; 361(22):2135-2142.
10. Ko, et al. Monoclonal antibodies against *Helicobacter pylori* cross-react with human tissue. Helicobacter, 1997; 2(4):210-215.
11. Blaser. Who are we? Indigenous microbes and the ecology of human diseases. EMBO Reports, 2006; 7(10): 956-960.