Diagnostic Solutions laboratory

Helicobacter pylori Virulence Factors

Quick Reference Guide

The virulence factor genes on GI-MAP® are found exclusively on the genome of *H. pylori*.

These genes code for proteins that will predispose one to more serious *H. pylori* infections.
The chart below provides details of each virulence factors tested on the GI-MAP.

Gene Acronym	Gene Name	Genetic Characteristics	Associations with Disease
BabA	Blood Group Antigen Binding Adhesion	• Promotes DNA breakage in host cell • Improves *H. pylori* adherence ("stickiness") to epithelial cells • May promote other virulence factors, especially CagA	May promote carcinogenesis
CagA	Cytotoxin Associated Gene A	• Promotes *H. pylori* adhesion and colonization • Affects barrier function of gastric epithelial tight junctions • Promotes loss of cell polarity • Antagonizes VacA • Evades the immune system and affects the activity of dendritic cells and B-cells. • Considered part of the "pathogenicity island" which includes VirB and VirD virulence factors. This is a closely associated group of genes that work synergistically and often transfer as a unit.	Promotes carcinogenesis, strong association. Also associated with peptic ulcer disease
DupA	Duodenal Ulcer-Promoting Gene A	• Promotes inflammation	Associated with duodenal ulcers, specifically

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Gene Acronym	Gene Name	• Genetic Characteristics	Associations with Disease
IceA	Induced by Contact with Epithelium A	• Transcription of this gene is only initiated after adhesion to the gastric epithelium • Promotes inflammation and associated with elevated IL-8	Associated with dyspepsia and gastric & duodenal ulcers NOT associated with gastric cancer
OipA	Outer Inflammatory Protein A	• Promotes inflammation • Drives IL-8 production	Associated with carcinogenesis and peptic ulcer disease
VacA	Vacuolating Toxin A	• Enters the host cell by endocytosis • Affects mitochondrial function • Disrupts tight junctions • Causes a programmed necrosis by inducing the production of large vacuoles inside the host cells; inducing cellular swelling; disrupting cell barrier thus causing nutrient leakage • Facilitates nutrient acquisition (iron, minerals, amino acids, etc.) • Inhibits antigen presentation in vitro • Antagonizes CagA	Associated with gastric inflammation, peptic ulcer disease, and gastric cancers
VirB & VirD		• Part of the CagA "pathogenicity island" • Both genes can potentiate CagA virulence factor by aiding in its transmission to host epithelial cels • In the absence of CagA, these virulence factors are unlikely to change clinical outcome of *H. pylori* infections.	Evaluate next to CagA virulence factors. VirB & VirD, if positive, can potentiate CagA virulence and clinical associations

How To Target Treatments in the Presence of Virulence Factors:

Generally, when virulence factors are present, the treatment goal will be to fully eradicate the *H. pylori* population. This can be confirmed by retesting the full GI-MAP, the pathogen panel, or the *H. pylori* panel 4–6 weeks after completing treatment. The goal is to achieve a result of <dl on the retest. The exception to this may be VirB and VirD if they are found in isolation (without CagA present).

Below is a chart of treatment considerations for each of the virulence factors. These would be used in addition to the standard treatments for *H. pylori* alone.

Virulence Factor	Special Treatment Considerations
BabA	More aggressive treatment may be warranted; consider the use of adhesion inhibitions, particularly cranberry
CagA	Target inflammatory support, promote T-cell activity, consider curcumin, resveratrol/red wine, ginger, Nigella sativa, low salt diet
DupA	Consider the use of demulcents for mucosal protection
IceA	Inflammatory support, consider the use of adhesion inhibitors
OipA	Inflammatory support
VacA	Mitochondrial support, consider Nigella sativa, green tea, red wine/resveratrol, Scutellaria baicalensis
VirB & VirD	No additional treatments necessary

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