H. pylori

Stomach ulcers are NOT caused by excess stomach acid, but rather by the bacterium Helicobacter pylori:

That H. pylori is the cause of peptic ulcer-associated gastritis was first determined in 1979, and was unequivocally proven by 1981. One of the researchers who discovered the connection between H. pylori and stomach ulcers was so frustrated at his being ignored by the medical establishment that he actually infected himself with H. pylori by ingesting a culture of bacterium, resulting in immediate gastritis, which he subsequently cured with the appropriate antibiotic.

Gastritis and peptic ulcer have thus been conclusively shown to be infectious diseases, and have absolutely nothing to do with too much stomach acid. In fact, H. pylori thrives in a low acid environment, and even creates its own low acid environment as it damages the lining of the stomach. Its helical shape allows it to burrow into the stomach's mucoid lining.

To avoid the acidic environment of the stomach, H. pylori burrows in deeply enough to reach the epithelial cells underneath, where it is less acidic. H. pylori is able to sense the pH gradient in the mucus and move to the less acidic region. The burrowing beneath the mucoid lining not only protects the bacteria from the healthy secretion of stomach acid, but also prevents the constantly flowing mucus lining of the stomach from sweeping the critters away.

The H. pylori adherence to the surface of stomach epithelial cells is acid-sensitive, and can be fully reversed by increased pH (less acid). This responsiveness to pH changes enables adherence while also allowing an effective escape from stomach acid, which is designed not only to prepare foods for digestion, but also to protect from microbial invasion. --- The means by which the bacteria neutralize stomach acid in its immediate environment is by producing large amounts of urease, which breaks down the urea present in the stomach into carbon dioxide and ammonia. These react with the strong stomach acid to produce a neutralized area around the bacteria.

Interestingly, 60% of all people worldwide have H. pylori colonized in their upper GI tract. However, 80-90% of those harboring H. pylori never experience symptoms. In those who do suffer symptoms, the gastritis symptoms tend to be chronic stomach pain, nausea, bloating, and sometimes belching --- or even vomiting or black stool. If the chronic blood loss is significant, the person may become anemic --- with weakness, fatigue, and often visual disturbances. The majority of those with H. pylori-related symptoms will experience episodic extreme exacerbation of their non-ulcer dyspepsia with severe ulceration. The triggers for these acute exacerbations are not always clear.

----- If the inflammation is more toward the pyloric sphincter, the infection tends to lead to duodenal ulcers, while inflammation higher in the body of the stomach not only leads to gastic ulcers, but gives a 1-2% risk of gastric carcinoma.

H. pylori is also associated with colorectal polyps and colorectal cancer.

There is a strong correlation (and perhaps a causative relationship) between H. pylori infection and the following conditions: rosacea, seborrheic dermatitis, and many ocular diseases including ocular rosacea, blepharitis, glaucoma, central serous chorioretinopathy, and others. ----- What is the link between H. pylori infection and these other diseases?

The suggested etiological role for H. pylori in these other inflammatory conditions involves a chronic inflammatory response systemically provoked by the upper GI inflammation. Gastric epithelial cells release cytokines such as Interleukins, which act as proinflammatory stimuli --- then promote the systemic release of other cytokines, which contribute to an overall inflammatory state --- and, these inflammatory cytokines in response to H. pylori are combined with a chronic histamine reaction from mast cells throughout the body's epithelial tissues.

----- The chronic inflammation also provokes free radical and lipid peroxide generation involving reactive oxygen species such as hydroxyl radicals, peroxides, and peroxynitrites. It has very definitely been shown that H. pylori infection increases both serum and tissue levels of nitric oxide and peroxynitrite --- inducing vasodilation, inflammation, and immune modulation.

Sacca, et al. H. pylori infection and eye diseases: A systematic review. Medicine, 2014.

Inhibition of acid secretion with proton pump inhibitors or H2 blockers increases symptoms of gastritis in H. pylori infected subjects. H. pylori infection contributes to under-secretion of stomach acid. (Side Note --- data on gastric acid secretion in patients with esophagitis (GERD) suggest that acid secretion is normal or slightly diminished, and not elevated as is commonly believed.) Proton pump inhibitors increase the tendency of H. pylori to cause atrophic gastritis. H. pylori actually impairs gastric secretion functions.

Pajares, et al. Helicobacter pylori: Its discovery and relevance for medicine. Rev Esp Enfrem Dig, 2006.

Calamj. Helicobacter pylori modulation of gastric acid. Yale J Biol Med, 1999.

Konturek, et al. Helicobacter pylori and impaired gastric secretory functions. J Physiol Pharmacol, 1997.

- Those with low hydrochloric acid are those who are subject to H. pylori infections (and stomach ulcers), as well as yeast overgrowth of the GI tract. (--- Note that H. pylori is the cause of stomach ulcers, and H. pylori infection is both caused by and causes hypochlorhydria. Trying to cure gastric/peptic ulcers with proton pump inhibitors or even over-the-counter antacids is absolute insanity.)

H. pylori damages the stomach and the duodenum by several mechanisms. First, the ammonia produced to neutralize the stomach acid in its immediate environment is toxic to the upper GI lining. But there are many other mechanisms --- including many toxic secretions that either break down the stomach lining directly, or initiate an extreme inflammatory irritation. Ulcers in the stomach and duodenum result when the consequences of inflammation allow stomach acid and the gastric digestive enzyme pepsin to overwhelm the protective mucous of the gut membranes.

The location of an ulcer matches the area of H. pylori colonization, which depends on the distribution of the acidity in the stomach. In individuals producing abundant hydrochloric acid, H. pylori colonizes in the pyloric area (just before the stomach opens into the duodenum) so as to avoid the acid-secreting parietal cells at the upper entrance to the stomach. But in people producing normal or reduced amounts of stomach acid, H. pylori can also colonize the rest of the stomach.

As you can see, there is a paradoxical relationship between H. pylori infection, stomach acid secretion, and ulceration. Initially, H. pylori colonizes in the lowest acid stomach environment it can find, and then carves out an area of ultra low stomach acid as the environment where it can fully penetrate and thrive beneath the mucosa. The paradox is that H. pylori needs an acid concentration gradient --- from extreme acidity to less acidity --- to direct its invasion and ultimate adherence to the epithelium beneath the mucoid lining. So, once H. pylori is established, its further spread is inhibited by neutralizing stomach acid such that the bacteria does not have the metabolic stimulation it needs to expand its penetration in both width and depth. That explains the paradox of why antacids --- which chronically make the condition worse, will actually give some relief as they remove the acid concentration gradient that H. pylori requires to direct its invasion.

Once H. pylori-associated gastritis is established and then advances to the point of ulceration, there is yet another factor contributing to this high-low acid paradox. The inflammatory response to bacteria colonizing in the pyloric area induces the secretion of the exocrine hormone gastrin. In the absence of ulceration, gastrin is an essential part of upper GI digestive function. The thought of food, the aroma of food, and the initial tasting of certain foods

activates the upper GI tract --- and an important part of that activation is stimulation of gastrin release. Gastrin stimulates the parietal cells to secrete more acid into the stomach lumen as an aide to healthy digestion.

--- But when there is an ulcer, the existing inflammatory response causes over-release of gastrin, and thus overstimulation of stomach acid production. The excess acid does not harm the healthy portion of the stomach, but where there is H. pylori, it facilitates its invasive activity. It is in these cases of H. pylori colonization in the pyloric area that the inflammation tends to cause duodenal in addition to or instead of gastric ulcers.

----- Gastrin production is particularly activated by caffeine, alcohol, and sugar. That explains why it is a tradition in many cultures to have coffee or tea at the beginning of a meal, or an alcoholic aperitif (such as Sherry) before a meal. But in those with ulcers, coffee or alcohol or sugar on an empty stomach can activate an extreme flare up of symptoms.

The standard and very effective treatment for H. pylori is a seven-day "triple therapy" consisting of a proton pump inhibitor plus two antibiotics --- generally Clarithromycin and either Amoxicillin or Metronidazole. As stated above, use of proton pump inhibitors or other antacids <u>alone</u> is a losing proposition --- guaranteeing that, even though symptoms improve temporarily, the H. pylori is quite happy with the low acid environment, and gastritis continues to cause degenerative atrophy of the mucoid lining. --- But <u>temporary</u> (seven-day) use of a proton pump inhibitor along with the antibiotics will guarantee that the infection is eradicated.

The most effective and simplest 7-day regimen consists of Aciphex 20 mg. + amoxicillin 1000 mg. + clarithromycin 500 mg. === all 3 taken together, twice daily, for 7 days. ----- Note the use of Aciphex and not the most common proton pump inhibitor, Prilosec. Prilosec when used as part of this 7 day regimen often causes gastrointestinal distress, and is not as effective as Aciphex.

Are there alternative treatments that can avoid the PPI and the antibiotics? Yes, absolutely. Bismuth suppresses H. pylori. So, an over the counter remedy such as Pepto Bismol will give both symptomatic relief and tend to control the H. pylori invasion. And as far as symptomatic relief goes, even baking soda (1/2 tsp in 4 oz. of water) is quite effective in giving temporary relief. And if you combine some combination of Arm and Hammer baking soda and/or Pepto Bismol with your NUTRI-SPEC products, you will have a winning therapy in a high percentage of cases.

L. reuteri (one of the probiotics in your Immuno-Synbiotics) <u>suppresses H. pylori infection</u> in humans and decreases the occurrence of dyspeptic symptoms. Bifidobacteria also inhibit H. pylori. So, Immuno-Synbiotic,

IMMUNE RESTORE, containing both L. reuteri and Bifidobacteria, is ideal for those with gastritis/ulcer. ----- Also, A Good Thyme taken orally according to the instructions will combat H. pylori.

The combination of Immuno-Synbiotic and A Good Thyme (not taken at the same time) is very effective (though not as effective as the 2 antibiotics and the bismuth) in eradicating H. pylori, while helping to eliminate the cause. Very often you will need to supplement with Proton Plus as a source of hydrochloric acid at some point. But when the condition is acute, you may need to supplement with Baking Soda.

So --- there are 2 ways to approach H. pylori. First, is to go with the A Good Thyme and Immuno-Synbiotic and perhaps the Baking Soda eventually replaced by Proton Plus and see if you get the job done. If not, you can fall back on the 2 antibiotics and the bismuth. The other approach is to use the bismuth and 2 antibiotics for a short time first, and then follow through with the Immuno-Synbiotic, Good Thyme, and Proton Plus afterwards to re-establish normal GI function.

A large part of the benefit from L. reuteri and Bifidobacteria is because they so effectively induce the production of butyrate in the gut. Butyrate powerfully suppresses H. pylori infections. In fact, in addition to Immuno-Synbiotic Immune Restore, butyrate as a concentrated supplement can be used in difficult cases. Butyrate is an antimicrobial which destroys the cell envelope of H. pylori by inducing the body's regulatory T cell expression.

One further consideration regarding H. pylori ----- Is H. pylori always "bad"? --- There is substantial evidence that the presence of H. pylori in more than 60% of the people worldwide is not necessarily a pathological phenomenon. It appears likely that H. pylori is part of our natural microbiota. Evidence suggests that in those with a "healthy" population of H. pylori in the stomach there is a decreased incidence of asthma, rhinitis, atopic dermatitis, inflammatory bowel disease, GERD, and esophageal cancer --- all by influencing systemic immune responses. Consider the possibility that the 10-20% of the 60% who have H. pylori colonization only suffer gastritis because they lack the anti-inflammatory capacity to control the bacteria. It may be that those who have a significant colony of H. pylori would be well protected by using the NUTRI-SPEC adaptogens Adapto-Max, Oxy-Max, Taurine, Immuno-Synbiotic, Oxy Tonic, Electro Tonic, and Oxy D+ as indicated by NUTRI-SPEC Protocols.