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Tytgat, G.N.J.

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Comment From the Editors

***Helicobacter pylori*: A Vanishing Problem?**

Few discoveries in gastroenterology have witnessed such explosive speed of development as the discovery of *Helicobacter pylori*, and the number of scientific publications continues to increase exponentially. The full genome of this organism is known, and presumably most relevant virulence factors have been identified. Discovering the most optimal therapy remains the major outstanding goal.

What More Do We Want to Know?

Most antibiotics that are active in vitro fail in vivo. Many factors contribute to clinical failure: poor diffusion in the mucous layer, the nature habitat of *H. pylori*, high intragastric acidity impairing the efficacy of the various antimicrobials, and ready induction of resistance, especially to metronidazole and clarithromycin. Primary care physicians and specialists are bewildered by the number of therapeutic regimens that have been proposed, largely on the basis of studies only published as abstracts. Many industry-supported studies are irrelevant, redundant, or poorly designed. Yet the prerequisites for a valid study are well defined. These include the need for careful analysis of primary resistance patterns in the study population. Although cure is obviously the most important primary outcome, standardized monitoring of side effects is also essential. A major confounding problem is the methodology used to monitor the efficacy of the antimicrobial therapy. Many diagnostic tests lose sensitivity after prior antibiotic therapy. It is therefore essential to use at least two independent techniques to monitor eradication efficacy including antrum and corpus biopsies, for histology, culture, rapid urease testing, etc. Monitoring induction of secondary resistance is also vital. Inclusion of

multiple antrum and corpus biopsies for determination of secondary resistance should be mandatory. Another important confounding factor is continuation of acid-suppressive therapy. Both proton pump inhibitors and H_2 -receptor antagonists interfere with the detection of remaining organisms and must be stopped at least 4 weeks before follow-up endoscopy. Acid-suppressive therapy also markedly reduces the sensitivity of the urea breath test. As larger groups of individuals are being treated with antibiotics, we are likely to see more multiresistant strains. With a more liberal use of broad-spectrum antibiotics, emergence of antimicrobial resistance in the resident microflora should also be taken into account. Future antimicrobials should be specific for the targeted pathogen. Several distinctive features of *H. pylori* could serve as selective targets such as urease, flagellar components, adhesions and receptor analogues, bacterial mechanisms for sensing, and modulating environmental signals.

Is the Pendulum Swinging Back?

Until now all attention was focused on the noxious effects of *H. pylori*. This conventional wisdom is being reevaluated in the context of the occasional almost "commensal-like" nature of the *H. pylori* infection causing only low-grade smoldering chronic inflammation. Some have suggested that it may be preferable to ignore commensal-like organisms and focus only on true pathogens identified by various immunologic markers. Is commensal-like parasitism a stable condition or does virulence shift back and forth because of other intercurrent infections or alterations in host conditions, or is it perhaps related to stress, nutrition, or other concomitant illnesses? Even if *H. pylori* can and does behave in a commensal-like fashion in many individuals, how do we reconcile this

view with the omnipresent gastric mucosal inflammation?

Does It All Matter?

Although many questions remain (what is optimal therapy, what is outcome of screening and treatment of dyspeptics in the primary care setting, where is the point of no return in the inflammation-atrophy-metaplasia-dysplasia sequence, which patients should we treat, and should we screen the whole population?), it is possible to ask whether the answers matter. At least in the developed world, *H. pylori* seems to be disappearing (at a surprisingly rapid rate) from the community, possibly preempting the answers to these questions. Infection rates in children are distinctly decreasing in the western world, and patients with *H. pylori*-associated duodenal ulcer disease are becoming increasingly rare in our endoscopy practices.

It is easy to envision that the combined effects of disappearing bacterial load from the community and putative limitation of therapy to virulent strains will decrease the demand for antimicrobial therapy. These occurrences may well jeopardize the opportunity to answer the many outstanding questions. Is it perhaps time to switch our attention to other gastrointestinal diseases of unknown etiology? Perhaps so, but in the mean time, let us hurry up to finish the remaining studies.

GUIDO N. J. TYTGAT, M.D., Ph.D.
Associate Editor, GASTROENTEROLOGY