

BEYOND ACID: HELICOBACTER PYLORI COMPLETES THE OLD "NO ACID, NO ULCER" DOGMA

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SHORT COMMUNICATION

The journey toward a cure for peptic ulcer disease (PUD) has been a long one. Both types of peptic ulcers share similar pathogenesis, diagnostic approaches, and treatment strategies, yet there are factors that distinguish them. Duodenal ulcers (DU) are characterized by a chronic and recurrent nature; more than 90% are associated with antral gastritis in the presence of the Gram-negative bacterium *Helicobacter pylori* (*H. pylori*), compared to 70% of patients with gastric ulcers (GU).

The presence of curved bacilli, spirilla, or spirochetes—has been identified in the gastric mucosa of animals and humans since 1893, as well as urease, an enzyme typical of the *Campylobacter* genus. Spirochete-like microorganisms were also identified in 43% of 242 autopsy biopsies; however, no definitive conclusion was reached due to tissue alteration caused by autolysis. Another study involving 35 partial gastrectomies identified bacteria in 37% of cases; nevertheless, the authors concluded that these were opportunistic bacteria.^[1] Perhaps due to Carl Schwarz's 1910 dogma—"no acid, no ulcer"—the optimal treatment approach involved neutralizing excess acid through dietary modifications and the management of caffeine intake, smoking, and antacid use. The development of histamine H2 antagonists revolutionized the treatment of gastric hyperacidity; histamine stimulates secretion. In 1976, cimetidine—a selective histamine H2-receptor antagonist—was introduced to the market. Treatment with cimetidine for periods of 3, 6, 9, or 12 months appears to prevent the recurrence of most ulcers that have healed during therapy; however, it does not cure PUD or eliminate the bacteria—it merely reduces acid—and relapses generally occur quickly.^[2,3]

Starting in 1979, Robin Warren, an Australian pathologist, began compiling a list of patients with histological gastritis and the presence of curved bacilli in stomach biopsies. Warren realized he needed a clinician

to follow up on these cases. During his medical rotation, Barry Marshall met Warren, who showed him the histological findings from gastric biopsies containing curved bacilli. These bacilli resembled bacteria of the genus *Campylobacter*, a known cause of gastroenteritis. After reviewing the literature, Marshall discovered that these curved bacilli had been observed as early as 1893 but had generally been overlooked. The two published their preliminary findings in a letter to the editor of *The Lancet*.^[4,5] Upon completing his rotation, Marshall continued to recruit patients undergoing stomach biopsies. Curved bacilli were found in 77% of 22 cases of gastric ulcer (GU) and in 100% of 13 patients with DU and chronic gastritis. Cultures from 11 cases revealed flagellated Gram-negative bacteria named *Campylobacter pyloridis*, which were later reclassified into the genus *Helicobacter*.^[6]

Faced with a lack of animal models, Marshall attempted to fulfill Koch's postulates by ingesting a liquid culture of the bacterium before and after an endoscopy with gastric biopsy.^[7] After three days, he developed nausea and vomiting; the endoscopy was repeated on the eighth day, and the biopsy revealed gastritis, with the culture testing positive for *Campylobacter pyloridis*. Barry Marshall was treated with metronidazole and bismuth salts. Marshall made a full recovery, demonstrating the link between the bacterium and gastritis. Bismuth salts were introduced as early as 1697 and were widely used

in the last century as a primary or adjunctive treatment for dyspepsia and peptic ulcers. Bismuth salts possess bactericidal activity, bind to peptic ulcers, inactivate pepsin, stimulate prostaglandin production, and inhibit various enzymes, among other properties.^[8]

Complete dogma

The old, incomplete dogma of 1910—"no acid, no ulcer"—was challenged by Marshall and Warren in the 1980s when they cultured the bacterium *H. pylori*, the causative agent of chronic gastritis and a precursor to PUD. Consequently, the concept should be viewed as a complete dogma—"no bacteria, no acid, no peptic ulcer"—reflecting the shift in therapeutic approach toward the use of antibiotics combined with acid-suppressing drugs. Marshall and Warren sparked a flood of publications regarding the relationship between *H. pylori*, chronic gastritis, PUD, gastric cancer, and therapeutic management. Standard triple therapy (amoxicillin+clarithromycin+omeprazole) and bismuth quadruple therapy appeared on the scene.^[9,10]

Barry Marshall and Robin Warren will always be remembered for paving the way in the study of PUD. Their place in history is well established.

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