For generations, doctors believed that stomach ulcers were caused by stress, spicy foods, and alcohol. Once diagnosed, patients were promptly condemned to a life of soda crackers and milk. But in 1984, an Australian physician named Barry Marshall bucked conventional wisdom with a new idea—that peptic ulcers were caused by bacterial infections. Marshall suspected that the Helicobacter pylori bacterium was the major culprit, but without any human guinea pigs, his hypothesis was difficult to prove. So, in the spirit of scientific inquiry, he offered up his own body and downed a Petri dish of H. pylori.

To his delight, Marshall developed a serious case of gastritis, an inflammation of the stomach lining that can lead to ulcers. He then successfully treated himself with antibiotics. Further research confirmed his findings, and now peptic ulcers are routinely cured by killing off the bacteria. But Marshall’s work did more than improve the culinary life of ulcer patients; it completely changed our understanding of how microbes affect our bodies. Once scientists started really looking at H. pylori, our relationship with the bacteria only got more complicated.

A BUG’S LIFE

Most experts agree that humans have harbored H. pylori for thousands (if not tens of thousands) of years. The corkscrew-shaped bacterium typically enters the body through the mouth, and it’s estimated that two-thirds of all people today have H. pylori living in their guts. But in most cases, the bug is just a harmless part of the stomach’s normal bacterial population. The stomach is a highly acidic environment, and an uncomfortable setting for the bacteria, so H. pylori makes itself at home by secreting an enzyme that reduces acidity.

In some people, however, the bug’s presence provokes the immune system, and the body’s inflammatory response can lead to ulcers and even stomach cancer. This makes H. pylori sound like a houseguest from hell. And for the most part, we’ve eliminated it. Improved hygiene, dependable refrigeration, and the increased use of antibiotics in industrialized nations have drastically reduced the number of people whose bodies harbor H. pylori. In the West, as infections have decreased, so have the rates of peptic ulcers and stomach cancers.

But the story doesn’t end there. Although fewer people are developing peptic ulcers and stomach cancer, more people than ever are suffering from esophageal cancer. According to epidemiologist Farin Kamangar, incidences among white American males have increased 350 percent during the past two decades. The problem highlights our complex relationship with H. pylori. While the bug does cause ulcers, it also lowers acidity levels in the stomach. Without H. pylori, acid can start creeping up the esophagus, causing acid reflux and esophageal cancer.

Gary Huffnagle, a University of Michigan microbiologist, points out that the mixed effects of H. pylori are turning out to be common among microbes. “As scientists, we sometimes like to call this large collection of bacteria ‘potentially pathogenic organisms.’ They serve some good roles, but in the wrong situation, they can cause problems.”

The range of current experiments involving H. pylori also reflects just how confusing our relationship with the bacteria is. On the one hand, the National Institute of Health is funding research to understand how H. pylori are transmitted and how their presence can lead to stomach cancer. On the other hand, the organization has also spent time and money trying to determine why the absence of H. pylori is connected to esophageal cancer. This one little bug has shaken up our ideas about what bacteria do in our bodies. Sometimes, we can’t live with them, and sometimes, we can’t live without them.