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Other stomach microbiota modulate resistance to H. pylori-driven ulcers

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Mice with different naturally occurring stomach bacteria have distinct susceptibilities to disease caused by *Helicobacter pylori*, the well-known cause of ulcers in humans, according to a study published online ahead of print in the journal *Infection and Immunity*. This is the first study to document (in mice) that the presence of certain bacteria in the stomach microbiota can prevent pathology from *H. pylori*.

The gastro-intestinal tract is a veritable ecosystem packed with microbes, and over the last decade, investigators have been discovering that the species composition of that ecosystem can have a profound effect on human health. But the eureka moment that led to this study came "when we realized that mice from different vendors mount different responses to *H. pylori* infection," says principal investigator Karen Ottemann of the University of California, Santa Cruz.

Following this discovery, the researchers divided mice from the vendor, Taconic Farms, into three groups: mice treated with antibiotics in order to kill some of the resident bacteria, mice that were fed normal stomach bacteria after antibiotic treatment, and mice that were not treated. They then infected each group with *H. pylori*, and assayed the animals' stomachs for immune system cells.

"The antibiotic-treated mice had small quantities of particular inflammatory cells, called Th1 T helper cells," says Ottemann. Both the untreated mice, and the treated mice that were then fed normal stomach bacteria had normal (higher) levels of Th1 T helper cells. These results

suggested that the normal stomach microbes contribute to disease caused by *H. pylori*, says Ottemann.

The researchers then determined that around 4,000 species of bacteria were different in the high- and low-inflammation (no antibiotics, and antibiotic-treated, respectively) mice. Notably, the mice with low inflammation "had elevated amounts of Clostridia, bacteria known to prevent inflammation in the intestine," says Ottemann. Thus, the Clostridia may be key to dampening *H. pylori* pathology, although that remains to be determined, she says.

Ottemann says that this research may lead to predicting future *H. pylori* disease, including ulcers and gastric cancer—which has few treatment options and high mortality—based on stomach microbiota.

"After we determine which microbes underlie *H. pylori* disease outcomes, we could test whether *H. pylori*-infected people harbor those particular bacteria, and target them for curing," says Ottemann. Alternatively, such people could receive the protective bacteria as probiotics. The latter might be a superior option, because while prone to ulcers in middle and advanced age, people who harbor *H. pylori* are less likely to get esophageal cancer and asthma.

More information: A.S. Rolig, C. Cech, E. Ahler, J.E. Carter, and K.M. Ottemann, 2013. The degree of Helicobacter pylori inflammation is manipulated by the pre-infection host microbiota. Infect. Immun. Online ahead of print 19 February 2013, doi:10.1128/IAI.00044-13

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