

Tummy troubles -- gastrin key in bacterial-induced stomach cancer

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Current research suggests that levels of gastrin play a key role in the development of *Helicobacter*-induced stomach cancer. The related report by Takaishi et al, "Gastrin is an essential cofactor for *Helicobacter*-associated gastric corpus carcinogenesis in C57BL/6 mice," appears in the July 2009 issue of *The American Journal of Pathology*.

More than 50% of the world's population is infected with *Helicobacter pylori*, which causes chronic inflammation of the stomach lining and is strongly linked to the development of gastric ulcers and [stomach cancer](#). Stomach cancer is the second leading cause of cancer-related deaths world-wide.

Helicobacter infection results in increased expression of gastrin, a hormone that stimulates secretion of gastric acid; however, the role of gastrin in cancer development remains unclear. High levels of gastrin lead to the development of stomach cancer, but absence of gastrin has been shown to increase the numbers of tumors in the gastric antrum, the lower section of the stomach that empties into the small intestine.

To reconcile this apparent disparity, a group led by Dr. Timothy Wang at the Columbia University Medical Center in New York, NY examined the contribution of *Helicobacter* infection to [gastric cancer](#) in animal models with either high expression of gastrin or no gastrin at all. They found that *Helicobacter* infection in mice with high levels of gastrin resulted in cancer of the gastric corpus (main body of the stomach), whereas infection in gastrin-deficient mice developed cancer in a

different part of the stomach, the gastric antrum. Gastrin, therefore, plays a key role in the development of *Helicobacter*-induced stomach cancer, but may have distinct effects on carcinogenesis in different parts of the stomach.

Takaishi et al suggest that "gastrin may serve as a rheostat for the stomach. Gastrin likely plays a central role in the safety network for the protection from mucosal damages caused by gastric acid secretion induced by gastrin itself, and thus either too much or too little gastrin can predispose to carcinogenesis. Therefore, clinicians in the future may need to be more circumspect when prescribing with acid suppressive drugs, such as proton-pump inhibitors, for a long-term use in patients infected with [Helicobacter pylori](#)." In future studies, Dr. Wang and colleagues plan to study "host factors other than gastrin [that] are also ... important for *Helicobacter*-associated gastric carcinogenesis. These include specific cytokines and chemokines induced by *Helicobacter* infection, and modulated by gastrin, that link inflammation and cancer. In addition, they plan to study the role of other non-*Helicobacter* bacteria that colonize the stomach when acid secretion is suppressed, since bacterial overgrowth likely contributes to gastric carcinogenesis".

More information: Takaishi S, Tu S, Dubeykovskaya ZA, Whary MT, Muthupalani S, Rickman BH, Rogers AB, Lertkowitz N, Varro A, Fox JG, Wang TC: Gastrin is an essential cofactor for *Helicobacter*-associated gastric corpus [carcinogenesis](#) in C57BL/6 mice. *Am J Pathol* 2009 175: 2775-2786

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