Certain strains of stomach bacteria contribute to gastric carcinogenesis

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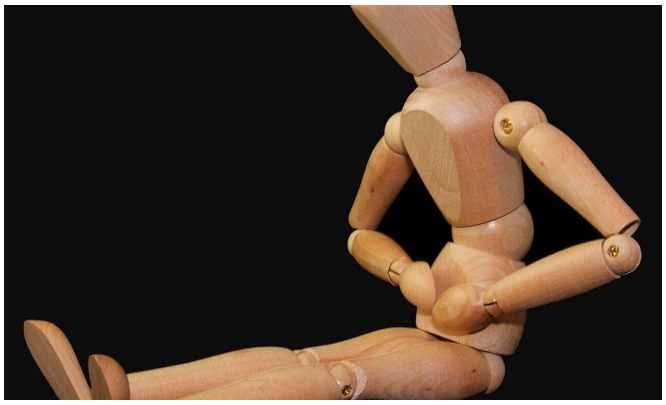

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The bacterium Helicobacter pylori colonizes the stomach in half of the world's population and increases the risk of gastric cancer.

Strains of H. pylori that contain the "cag PAI" gene cluster are associated with higher cancer risk. These strains synthesize the Cag type IV secretion system (T4SS), which injects the oncoprotein CagA and other bacterial products into stomach cells.

Aung Soe Lin, Mark McClain, Ph.D., Timothy Cover, MD, and colleagues developed methods for controlling Cag T4SS activity in an animal model of H. pylori-induced gastric cancer. They reported in the journal *mBio* that animals exposed to Cag T4SS activity had increased gastric inflammation and developed premalignant lesions and/or invasive gastric adenocarcinoma, demonstrating that Cag T4SS activity contributes to gastric carcinogenesis.

The researchers also showed that turning on Cag T4SS activity only during the early stage of infection is sufficient to trigger cellular changes that lead to gastric inflammation and cancer at later time points, consistent with a "hit-and-run" model of carcinogenesis.

More information: Aung Soe Lin et al. Temporal Control of the Helicobacter pylori Cag Type IV Secretion System in a Mongolian Gerbil Model of Gastric Carcinogenesis, *mBio* (2020). DOI: 10.1128/mBio.01296-20

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