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Helicobacter pylori inhibits intercellular communication of cultured gastric cells

The formation of a cancer is proven to be a multi-stage, multi-mechanism process by animal and human studies. As a definite carcinogen, the role of *Helicobacter pylori* (*H pylori*) in the formation of gastric cancer has been unclear. An article to be published on November 7 in the *World Journal of Gastroenterology* reveals a new mechanism explaining the promotional effect of *H pylori* on gastric cancer.

Gap junctions are fundamental structures necessary for cell differentiation, tissular physiology and normal functions of the organs of the body. The loss of functional gap junctions has been described in cancer cells and led to the hypothesis that such type of intercellular communication is involved in the carcinogenesis process. Up to now, a lot of data has been accumulated, confirming that gap junctional intercellular communication (GJIC) is frequently decreased or absent in cancers such as liver cancer, skin cancer, bladder cancer, breast cancer, lung cancer, and so on. However, the change of GJIC in *H pylori*-associated gastric cancer has been little exploited.

In this article, the researchers treated a human gastric cell line *in vitro* with intact bacteria and sonicated extracts of two *H pylori* strains with the virulence protein CagA. These were positive (CagA+) and negative (CagA-), respectively. After overnight treatment, the GJIC of the cells was measured by a technique named fluorescence redistribution after photobleaching (FRAP). The authors found that both CagA+ and CagA- *H pylori* strains could inhibit the GJIC of gastric cells when compared with a blank group. In addition, the inhibitory effect on the GJIC of gastric cells of CagA+ *H pylori* was more significant than that of CagA-.

The authors' conclusion emphasized the close relationship between *H pylori* and gastric cancer. The authors indicate that *H pylori* may make initiated cells of gastric cancer escape from the control of the neighboring ones by inhibiting GJIC. Consequently, *H pylori*, especially CagA+ strains, play an important role in the developmental process of gastric cancer.

The results of this article provide an innovative direction to develop new drugs for curing gastric cancer. That means drugs able to restore GJIC in cells with deficient gap junction may be used in the prevention and/or treatment of human gastric cancer.

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Reference: Tao R, Hu MF, Lou JT, Lei YL. Effects of H pylori infection on gap-junctional intercellular communication and proliferation of gastric epithelial cells in vitro. World J Gastroenterol 2007; 13(41): 5497-5500

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