# Stomach bacterium damages human DNA

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The stomach bacterium *Helicobacter pylori* is one of the biggest risk factors for the development of gastric cancer, the third most common cause of cancer-related deaths in the world. Molecular biologists from the University of Zurich have now identified a mechanism of Helicobacter pylori that damages the DNA of cells in the gastric mucosa and sets them up for malignant transformation.

Gastric cancer is one of the most common and often fatal cancers: Every third cancer death is due to gastric carcinoma. The main risk factor for the development of gastric cancer is the chronic infection of the gastric mucosa with the bacterium *Helicobacter pylori*. Since this bacterium was discovered in 1983, scientists have been puzzling over the molecular mechanisms triggering carcinogenesis. Now, several research teams headed by Prof. Anne Müller and Prof. Massimo Lopes from the Institute of Molecular Cancer Research at the University of Zurich have demonstrated how Helicobacter pylori harms human and animal host cells in in vitro experiments. The study published in the science journal *PNAS* shows that infection of host cells leads to breaks in both strands of the DNA double helix.

## Degree of damage depends on duration of infection

Müller, Lopes and their teams also show that the frequency of the double-strand breaks depends on the intensity and duration of the infection.

The DNA breaks induced by *H. pylori* trigger the cell's natural DNA damage signaling and repair mechanisms. If the bacterium is killed off

with antibiotics within a few hours of infection, most of the breaks can be repaired successfully. Prolonged infections that imitate the conditions in the chronically infected host, however, exhaust the cell's repair response and the dangerous double-strand breaks can no longer -or only imprecisely- be repaired, causing genetic mutations or the death of the cell. The new findings are in agreement with the previously described genomic instability of gastric cancer cells and will be important for a better understanding of the pathological mechanisms promoting gastric carcinogenesis.

Provided by University of Zurich