Medical Xpress

Understanding how the stomach responds to injury could help target therapy against gastric damage

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A better understanding of the stomach's immune response to *Helicobater pylori* (*H. pylori*) infection could lead to new therapies targeting damage in the stomach, report researchers in the March issue of *Cellular and Molecular Gastroenterology and Hepatology*, the basic science journal of the American Gastroenterological Association.

When *H. pylori* infection is present, the alarmin Interleukin (IL)-33 is a critical messenger that triggers changes necessary for coping with the injuries caused by the infection. Specifically, it actives an inflammatory immune response that begins the process of cell loss that can lead to the onset of metaplasia.

"These (IL-33) immune cell drivers of proliferation and expansion of metaplasia may be a critical target for intervention and further research," said lead study author Jon N. Buzzelli, PhD, Murdoch Children's Research Institute, Department of Pediatrics, Royal Children's Hospital.

Researchers found that IL-3 is elevated during acute response to infection. It declines in chronic infection. This may account for alterations observed in patients with chronic infection.

This investigation used a number of mouse models to define patterns of immune regulation of gastric pathology.

"Since other studies have noted that IL-33 may be elevated in association with metastatic gastric cancer, further investigations will be necessary to determine the changing influences of IL-33 at different stages of cancer development," notes James R. Goldenring, MD, PhD, AGAF, associate editor, *Cellular and Molecular Gastroenterology and Hepatology*.

More information: Buzzelli, Jon N., et al., IL33 Is a Stomach Alarmin That Initiates a Skewed Th2 Response to Injury and Infection, *Cellular and Molecular Gastroenterology and Hepatology* 2015: 1(2): 203-221.e3, www.cmghjournal.org/article/S2 … (14)00013-7/abstract

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