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# Up-regulated Th17 cell function is associated with increased Peptic ulcer disease in *Helicobacter pylori*-infection

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## Abstract

**Background:** During *Helicobacter pylori* (*H. pylori*) infection CD4<sup>+</sup> T cells in the gastric lamina propria are hyporesponsive and polarized by Th1/Th17 cell responses controlled by Treg cells. The objective of this study was to determine the number of Th17 cells in gastric mucosa of patients with gastritis and peptic ulcer and determined the relationship between main virulence factor of *H. pylori* and Th17 cells.

**Methods and materials:** A total of 89 *H. pylori*-infected gastritis patients, 63 *H. pylori*-infected peptic ulcer patients and 48 *H. pylori*-negative non-ulcer dysplasia patients were enrolled in this study. The number of Th17 was determined by immunohistochemistry. IL-8 and IL-17A expressions were determined by real-time polymerase chain reaction (qPCR). Also, the grade of chronic and active inflammation was investigated for involvement according to the density of neutrophils and mononuclear in gastric mucosal crypts, from one to all crypts.

**Results:** The number of Th17 cells and the expression of IL-8 and IL-17A in infected patients were significantly higher than uninfected subjects. The number of Th17 cells and the expression of IL-8 and IL-17A in infected patients with peptic ulcer were significantly higher than patients with gastritis. Additionally, the numbers of Th17 cells as well as the expression of IL-8 and IL-17A were positively correlated with the degree of *H. pylori* density in infected patients with peptic ulcer, while this correlation was negative in infected patients with gastritis. The numbers of Th17 cells as well as the expression of IL-8 and IL-17A were positively correlated with the degree of chronic inflammation.

**Conclusion:** The predominant Th17 cell responses may play a role in the pathogenesis of peptic ulcers disease in infected patients.

**Keywords:** *Helicobacter pylori*; Th17; Gastritis; Peptic ulcer disease; Virulence factor

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