

# The Expression of CD74 Molecule in *H.pylori* Infected Gastric Mucosal Tissue

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## **Abstract**

**Background:** *Helicobacter pylori* cause gastric inflammation. Recent interest has been focused on the role of CD74 (the class II MHC-associated invariant chain expressed on the surface of gastric epithelial cells) as an adhesion molecules used by *H.pylori* that may contribute to the proinflammatory immune response seen during infection.

**Objective:** The aim of this study was to detect the CD74 mucosal expression in *H.pylori* infected patients and compare it with uninfected patients.

**Patients and Methods:** Sixty-four patients' age mean (34± 1.7) years (14-66 years) who underwent upper gastrointestinal endoscopy because of gastrointestinal complaints, were studied.

A number of both invasive and non-invasive diagnostic tests were used for the diagnosis of *H. pylori* infection, as well as immunohistochemical study of biopsy

specimens to detect the CD74 mucosal expression.

**Results:** After the diagnosis of *H.pylori* infection, patients were grouped as *H. pylori* positive, (n=47) and *H. pylori* negative (n=17). According to immunohistochemical study of biopsy specimens, the expression of CD74 was observed in infected subjects, and there was a significant difference in the CD74 expression ( $p= 0.005$ ) between infected and uninfected patients.

**Conclusion:** According to immunohistochemical study of biopsy specimens an overexpression of CD74 was observed in infected subjects

**Keywords:** *Helicobacter pylori*; CD74; gastric epithelial cells; Immunohistochemistry (IHC)

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## **Introduction**

*Helicobacter pylori* infection provokes a vigorous humoral and cellular immune response in humans, but the organism is rarely eliminated from the gastric mucosa and infection persists lifelong in the absence of treatment<sup>(1)</sup>. *H. pylori* colonize the human stomach and is usually found either as an extracellular pathogen in the gastric mucosa or tightly attached to the cells of the gastric epithelium. Colonization by *Helicobacter pylori* always causes chronic gastritis and leads to the development of severe gastroduodenal diseases such as peptic

ulcers, gastric adenocarcinoma, or Lymphoma of the mucosa associated lymphoid tissue (MALT)<sup>(2,3)</sup>. Although the mucosal colonization of *H. pylori* induces a mixed Th1/2-mediated mucosal cytokine milieu<sup>(4,5)</sup> and the generation of *H. pylori*-specific T- and B-cell clones, the inflammatory response is not sufficient to eradicate the organism from its host<sup>(5,6)</sup>. The chronic immune response induced could afford a colonization advantage for the bacteria by providing improved availability of adhesion places. An example of this is the resulting increase in class II major histocompatibility complex (MHC) and CD74, induced by IFN- $\gamma$  and IL-8 that are used as receptors by *H. pylori*<sup>(7,8,9)</sup>. The CD74 chain was thought to function mainly as an MHC class II chaperone, which promotes an endoplasmic reticulum (ER) exit of MHC class II molecules, directs them

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