

propria that expressed CD74. Specific staining using anti-CD74 clone LN-2 antibodies showed that the highest density of CD74 expression occurred along the apical side of the cells and faint staining was detected at the basolateral side. These results indicate a correlation between *H. pylori* infection and CD74 expression. There was a highly significant association between the high expression of CD74 in infected and uninfected patients. These results could be explained in the light of other studies, Beswick and colleagues<sup>(8, 9)</sup> investigated the interaction of *H. pylori* with the class II major histocompatibility complex (MHC)-associated invariant chain (CD74), which found to be highly expressed by gastric epithelial cells, and they suggested a role for CD74 in gastric epithelial cell interaction with *H. pylori* leading to NF- $\kappa$ B signaling that results in IL-8 secretion. Moreover, Bacterial binding was increased when CD74 surface expression was increased by IFN- $\gamma$  treatment or by fibroblast cells transfected with CD74, while binding was decreased by CD74 blocking antibodies, enzyme cleavage of CD74, and CD74-coated bacteria. *H. pylori* was also shown to bind directly to affinity-purified CD74 in the absence of class II MHC. Increased CD74 expression by cells that showed increased IL-8 production in response to *H. pylori*, and agents that block CD74 decreased these responses. Therefore, adherence of the bacteria to the gastric mucosa is one of the initial steps of *H. pylori* infection and is an important virulence factor. Many different *H. pylori* adhesins have been identified<sup>(15)</sup> implying that adherence is a multifactorial process.

In conclusion, the present results show that in *H. pylori* positive biopsy specimens an overexpression of CD74 were observed in infected patients.

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