An etiological role for H. pylori in autoimmune gastritis

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Autoimmune type atrophic gastritis is a severe gastric atrophy associated with vitamin B12 deficiency. The reason for the disappearance of acid secreting cells from gastric mucosa is not fully understood, but the role of Helicobacter pylori in initiating the mucosal damage is suspected in animal studies. A study found signs of previous H. pylori infection in patients with autoimmune type atrophic gastritis.

Experimental animal studies have shown that *H. pylori* shares several antigenic regions in common with acid secreting cells in gastric mucosa. Antibodies triggered by *H. pylori* destroy acid secreting cells due this antigenic mimicry. *H. pylori* infection is very common in humans, and about half of the infected patients develop atrophic changes over the years. In end stage severe atrophy, *H. pylori* disappears and signs of a previous infection are difficult to detect.

This research, lead by Dr. L Veijola and her colleagues in the University of Helsinki, Finland, has recently been published on January 7, 2010 in World Journal of Gastroenterology. This study also confirmed the findings of previous studies that the serum markers of autoimmune gastritis appear with increasing frequency in patients with *H. pylori* infection, when the acid secreting capacity vanishes.

The prevention of autoimmune atrophic gastritis, and thus pernicious anaemia, by eradicating the *H. pylori* would make the lifelong vitamin B12 substitution therapy unnecessary.