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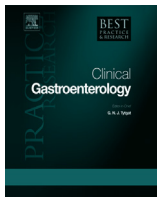

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Chronic gastritis – An update

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A B S T R A C T

Helicobacter pylori is the main aetiologic factor for chronic gastritis worldwide. The degree of inflammation and the evolution of this form of chronic gastritis can vary largely depending on bacterial virulence factors, host susceptibility factors and environmental conditions. Autoimmune gastritis is another cause of chronic inflammation in the stomach, which can occur in all age groups. This disease presents typically with vitamin B12 deficiency and pernicious anaemia. The presence of anti-parietal cell antibodies is highly specific for the diagnosis. The role of *H. pylori* as a trigger for autoimmune gastritis remains uncertain.
Other rare conditions for chronic gastritis are chronic inflammatory conditions such as Crohn's disease or on the background of lymphocytic or collagenous gastroenteropathies.

Introduction

Helicobacter pylori (*H. pylori*) infection is the most common cause of chronic inflammation of the stomach worldwide. The bacterium discovered by Warren and Marshall in 1982 colonizes around half

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of the world population. All *H. pylori* infected individuals develop chronic gastritis; the degree of mucosal inflammation results from the interplay of bacterial virulence factors, host susceptibility genes and environmental factors. Corpuspredominant gastritis, severe gastric atrophy and intestinal metaplasia confine a risk for the development of gastric cancer as shown in large prospective cohorts [1–3].

Autoimmune gastritis (AIG) is another cause of chronic inflammation of the stomach. The term AIG refers to a variety of definitions such as atrophic body gastritis, pernicious anaemia and Morbus Biermer. The clinical presentation can be variable, most typically with vitamin B12 deficiency and manifest pernicious anaemia (PA), but iron deficiency may also be a consequence. At the time of diagnosis, the oxyntic mucosa is usually transformed and shows severe glandular atrophy.

The traditional concept of AIG needs to be revisited because of the role of *H. pylori* infection in the development of AIG through mimicry of the proton pump of parietal cells (H^+/K^+-ATPase). The risk for type 1 gastric neuroendocrine neoplasm, as well as the co-occurrence with various autoimmune diseases is reviewed in this article.

Besides *H. pylori* gastritis and AIG, several other chronic inflammatory conditions of the gastric mucosa are discussed. They include collagenous gastritis, lymphocytic gastritis, M. Menetrier and M. Crohn and are rare forms.

H pylori gastritis

Epidemiology

The excusive permanent reservoir of *H. pylori* is the human stomach. Local prevalence of *H. pylori* varies greatly from 8% (North America) to 90% (Siberia) depending on geographic region and socioeconomic conditions [4]. A selection of *H. pylori* prevalence in the adult population from different continents is shown in Table 1. The bacterium is found in feces and the oral cavity of infected individuals [5] but exclusively colonizes and persists in the gastric mucosa. Since the wide-spread use of antibiotic treatment and the improvement of hygiene in industrialized countries, prevalence of *H. pylori* infection of children and young individuals gradually decreases [6], and lies below 10% in children in western populations [7,8].

Pathogenesis

Bacterial factors

H. pylori is an obligate pathogen in the human stomach. The bacterium possesses various virulence factors which facilitate survival, cell adhesion, cell damage and evasion from immune response. Table 2 shows a list of the best known virulence factors and their functions, excellently reviewed recently by Posselt et al. [9]. The pathogenicity island cytotoxin-associated gene a (cagA) in particular is expressed by a large number of strains and is considered a potent inductor of inflammation [10]. The cagA gene exhibits strong variations in different geographic locations correlated with incidence of gastric cancer [11,12]. Attention was drawn on variations in the conserved sequence of the *cagA* gene Glu-Pro-Ile-Tyr-Ala (EPIYA) motifs. Particularly the number of C tyrosine phosphorylations seems to play a decisive role in virulence as reviewed in another chapter of this edition. Two or more EPIYA C motifs point to an

Table 1
Reports of *H. pylori* prevalence in the largest studies in adults from different continents published between 2010 and 2011 using tests for active infection. Modified from Goh et al [6].

Continent	Country	N	Test	Prevalence
Asia	Korea	10,102	RUT	51%
Africa	Morocco	755	Histology	69%
South America	Chile	5664	RUT	78%
North America	USA	78,985	Histology	8%
Europe	Albania,	101 Albanian	Histology	54%
	Greece	101 Greek		34%