



REVIEW ARTICLE

Clinical consequences of *Helicobacter pylori* infection in children and its relation with the response of the gastric mucosa to the infection

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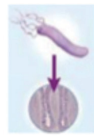
Abstract

Helicobacter pylori is usually acquired during childhood and remains in the gastric mucosa for years, often lifelong if untreated. It can be concluded that the gastric mucosa of children actively responds to the presence of *H. pylori*. Current evidences suggest that whereas *H. pylori* infection rarely causes peptic ulcers or gastric atrophy in children, it seems to be associated with iron deficiency and iron deficiency anemia; the evidence also suggests the infection may cause growth retardation. In contrast, *H. pylori* infection has been associated with a reduced risk of asthma and allergy in children and adults; also, epidemiological studies suggest that there is an inverse association between *H. pylori* infection and risk for esophageal adenocarcinoma. The gastric mucosa of children elicits a significant inflammatory response in the site of infection, with increased expression of toll-like receptors (TLRs) and cytokines, and increased epithelial proliferation. This response may partly be responsible for the required “immune training” needed to protect for the development of esophageal cancer, asthma, allergy or even diabetes later in life. The response may as well be associated with growth retardation, iron deficiency and increased risk for enteric infections. It then seems that our co-evolution with *H. pylori* has rendered benefits for human health making clear that this relationship is complex and the decision to eradicate the infection should be taken with caution.

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NORMAL
GASTRIC
MUCOSA



ACUTE
GASTRITIS

CHRONIC
GASTRITIS



INFLAMMATION →

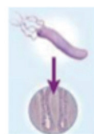
Altered epithelial
functions and altered
non-heme iron
solubility:

- Iron deficiency
- Anemia
- Malnutrition



➤ Growth
retardation?

CHILDHOOD



NORMAL
GASTRIC
MUCOSA



ACUTE
GASTRITIS

CHRONIC
GASTRITIS



Early priming of
epithelial immune
cells;
tolerance in
dendritic cells and
differentiation of
Tregs



Reduced incidence
of autoimmune
diseases later in
life?

CHILDHOOD

ADVANCE AGE

