

***H. pylori* and gastric malignancies**

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Objectives

At the end of this presentation, attendees should be able:

- 1) to identify *Helicobacter pylori* as a risk factor for gastric malignancies, namely for intestinal subtype of gastric adenocarcinoma¹;
- 2) to consider areas of controversy on causal relation between *H. pylori* infection and stomach cancer leading to consider other risk factors and management attitudes for individuals with this infection.

Summary

Helicobacter pylori infection prevalence varies worldwide but it still represents the most frequent in humans. Its role in diverse gastroduodenal diseases has been largely suggested by innumerable studies, and in 1994, it has been considered by the W.H.O. as type I carcinogen. Nevertheless, when both classic causality criteria and currently used clinical evidence levels for recommendation are reviewed, there is, at least, place for controversy [Eslick GD]. Gastric adenocarcinoma is still a major health problem and, at least, for intestinal subtype, a carcinogenesis cascade from normal mucosa leading to gastric adenocarcinoma has been described [Correa P]. Animal models were able to describe diverse mechanisms by which *H. pylori* may induce cells changes leading to cancer [Tsukamoto T]. Furthermore, both case-controls studies and cohort studies support a statistical association and a temporal relationship between *H. pylori* infection and gastric cancer. Moreover, non-controlled trials showed that individuals treated for *H. pylori* showed a decreased risk for developing gastric cancer. However, controlled trials did not consistently show that reduction and precancerous lesions were not reversible as well in most studies [Eslick GD, Fuccio L and Lee Y-C]. Also, other factors such as diet and, more specifically, host genetic variation have also be involved in carcinogenesis representing as well factors that may change observations at clinical studies and are potentially useful for management strategies definitions [Correa P, Eslick GD and Machado JC]. Thus, *H. pylori* seems to be the single most easily identifiable and relevant risk factor for gastric adenocarcinoma; and when diagnosed should be treated because it may prevent cancer to occur. However, a better understanding of gastric carcinogenesis modeling should proceed as correct and integrative assessment of bacterial, host factors and phenotype may conduct to more (cost-) effective strategies [Dinis-Ribeiro M].

Recommended reading

- 1) Correa P and Houghton J. Carcinogenesis of *Helicobacter pylori*. *Gastroenterology* 2007; 133: 659-672.
- 2) Dinis-Ribeiro M e tal. Feasibility and cost-effectiveness of using magnification chromoendoscopy and pepsinogen serum levels for the follow-up of patients with atrophic chronic gastritis and intestinal metaplasia. 2007;22:1594-604.
- 3) Eslick GD. *Helicobacter pylori* infection causes gastric cancer? A review of the epidemiological, meta-analytic and experimental evidence. *World J Gastroenterol* 2006; 12: 2991-2999.
- 4) Figueiredo C et al. *Helicobacter pylori* and interleukin 1 genotyping: an opportunity to identify high-risk individuals for gastric carcinoma. *J Natl Cancer Inst.* 2002;94: 1680-7.

¹ The lecture will be focused on existing evidence of the role of *H. pylori* on gastric adenocarcinoma. M.A.L.T. lymphoma will not be extensively assessed.

- 5) Fuccio L et al. Systematic review: Helicobacter pylori eradication for the prevention of gastric cancer. *Aliment Pharmacol ther* 2007; 25: 133-141.
- 6) Lee Y-C et al. Is eradication of Helicobacter pylori the feasible way to prevent gastric cancer? New evidence and progress but still a long way to go. *J Formos Med Assoc* 2008; 107: 591-599.
- 7) Tsukamoto T et al. Animal Models in stomach carcinogenesis. *Toxicologic Pathology* 2007; 35: 636-648