
LETTER TO THE EDITOR

***Helicobacter pylori* Infection as an Essential Factor for Stomach Cancer**

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To the Editors,

A recent article (Wu et al., 2005) provided state-of-the-art information on the relationship between *Helicobacter pylori* (*H. pylori*) and stomach cancer. It is particularly useful for understanding the biology and mechanisms regarding the virulence of *H. pylori* (Covacci et al., 1999; Hatakeyama and Higashi, 2005) and host genetic polymorphisms (El-Omar et al., 2000; Graham and Graham, 2002) which impact on defence against the bacterium, which may of course play a crucial role in gastric carcinogenesis.

In this context we need to stress the very low gastric cancer incidence rates observed in Yogyakarta and Semarang which appear to be due to the rarity of appreciable *H. pylori* infection (Tokudome et al., 2005a, b). The bacterium seems to be an egg, without which nothing can happen. This is in direct line with earlier findings suggestive that *H. pylori* is essential and necessary for gastric carcinogenesis, at least, for non-cardia gastric adenocarcinoma (Uemura et al., 2001; Brenner et al., 2004).

References

- Brenner H, Arndt V, Stegmaler C, et al (2004). Is *Helicobacter pylori* infection a necessary condition for noncardia gastric cancer? *Am J Epidemiol*, 159, 252-8.
- Covacci A, Telford JL, Del Giudice G, et al (1999). *Helicobacter pylori*, virulence and genetic geography. *Science*, 284, 1328-33.
- El-Omar EM, Carrington M, Chow WH, et al (2000). Interleukin-1 polymorphisms associated with increased risk of gastric cancer. *Nature*, 404, 398-402.
- Graham KS, Graham DY (2002). *H. pylori*-associated Gastrointestinal Diseases. 2nd Ed. Handbooks in Health Care, Newton.
- Hatakeyama M, Higashi H (2005). *Helicobacter pylori* CagA: a new paradigm for bacterial carcinogenesis. *Cancer Sci*, 96, 835-43.
- IARC (2004). Monographs on the Evaluation of Carcinogenic Risks to Humans. Tobacco Smoke and Involuntary Smoking. Vol. 83. IARC, Lyon.

From the etiological standpoint of stomach cancer, we therefore need to make a radical paradigm shift away from the general emphasis on lifestyle-related cancer, with risk factors including smoking, consumption of salt, and low intake of vegetables and fruit (World Cancer Research Fund/American Institute for Cancer Research, 1997; IARC, 2004) towards infectious disease. *H. pylori* should be placed at the top of the environmental factors, not parallel with them (Wu et al., 2005).

Thus, for the practical prevention of gastric cancer, the infection route/vehicle of *H. pylori* must be explored and pinpointed for infection control. Development of a vaccine to be applied during early childhood is also urgently required. An effective eradication strategy for infected people must be put in place. Periodic screening using gastroscopy should be launched along with consultation on modulations of the related environmental/behavioral/lifestyle factors for patients for whom eradication treatment proves unsuccessful or who have already developed chronic atrophic gastritis.

- Tokudome S, Soeripto, Triningsih FXE, et al (2005a). Rare *Helicobacter pylori* infection as a factor for the very low stomach cancer incidence in Yogyakarta, Indonesia. *Cancer Lett*, 219, 57-61.
- Tokudome S, Samsuria WD, Soeripto, et al (2005b). *Helicobacter pylori* infection appears essential for stomach carcinogenesis - observations in Semarang, Indonesia. *Cancer Sci*, 96, 873-5.
- Uemura N, Okamoto S, Yamamoto S, et al (2001). *Helicobacter pylori* infection and the development of gastric cancer. *N Engl J Med*, 345, 784-9.
- World Cancer Research Fund/American Institute for Cancer Research (1997). Food, Nutrition and the Prevention of Cancer: a Global Perspective. American Institute for Cancer Research, Washington, DC.
- Wu M-S, Chen C-J, Lin J-T (2005). Host-environment interactions: their impact on progression from gastric inflammation to carcinogenesis and on development of new approaches to prevent and treat gastric cancer. *Cancer Epidemiol Biomarkers Prev*, 14, 1878-82.

Shinkan Tokudome^{1*}, Akihiro Hosono¹, Sadao Suzuki¹, Reza Ghadimi¹, Tsutomu Tanaka¹, Hiromitsu Ichikawa¹, Machiko Miyata¹, Mitsuhiro Marumoto¹, Hiroyuki Agawa¹, Kazuyuki Arakawa¹, Ryosuke Ando¹, Nami Hattori¹, Kiyoshi Shibata^{1,2}, Zhao Yang¹

1 Department of Health Promotion and Preventive Medicine, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan, *2* Kasugai City Health Care Center, Kasugai, Japan, *Correspondence: E-mail: tokudome@med.nagoya-cu.ac.jp