

Comment On The Study Of Wong et al.

Wong¹ et al tried to determine whether treatment of H pylori infection reduces the incidence of gastric cancer. A prospective, *randomised, placebo-controlled*, population-based primary prevention study tried to discover the relationship between treatment of H pylori infection and the reduction of the incidence of gastric cancer.

Design

Patients were randomly assigned and have received H pylori eradication treatment (omeprazole, 20 mg, amoxicillin and clavulanate potassium 750 mg, and Metronidazole, 400 mg, all twice daily for 2-weeks). Besides of all, **there are still some questions about this study.**

- 1) Has the intake of the drugs by the patient been controlled and ensured to a necessary extent?
- 2) In so far, let us *assume*, the drugs were taken by the patients, how can the study group be sure, that Helicobacter pylori was not resistant against applied drugs before the treatment? Was this tested before the eradication therapy the right way? Was the H. p. treatment successful?
- 3) On the other hand, patients with precancerous lesions and with no precancerous lesions were treated under same conditions. Are there no medical differences between precancerous lesions and no precancerous lesions. Are precancerous lesions not already the first step on the way to gastric cancer? If yes, does it make sense to treat (pre) cancer with antibiotics?

Besides of all this concerns, Wong's study has achieved better results then published one.

Reanalysis.

Under the condition above, just about 18 new cases of gastric cancers developed. Thus we obtain the following 2 x 2 table. We set $\alpha = 0.05$, one sided, thus $Z = 1.64485363$.

		Gastric cancer		
		1	0	
Helico- bacter pylori inf.	1	7	810	817
	0	11	802	813
		18	1612	1630

Firstly.

$$p(\text{H pylori eradication treatment} \mid \text{gastric cancer}) = (11 + 810) / 1630 = \mathbf{0,995705521472393}$$

$$p(\text{H pylori eradication treatment} \mid \text{gastric cancer})_{\text{lower}} = 1 - (1.64485363 / \sqrt{4 * 1630}) = \mathbf{0,97962941694485}$$

We accept our Ho: H pylori eradication treatment excludes gastric cancer, because

$$p(\text{H pylori eradication treatment} \mid \text{gastric cancer}) > p(\text{H pylori eradication treatment} \mid \text{gastric cancer})_{\text{lower}}$$

1 **Wong** BC, Lam SK, Wong WM, Chen J S, Zheng TT, Feng RE, Lai KC, Hu WH, Yuen ST, Leung SY, Fong DY, Ho J, Ching CK, Chen JS; China Gastric Cancer Study Group, "Helicobacter pylori eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial," *JAMA*, 291(2) (2004 Jan 14), 187-94.

Result 1.

Even under the conditions above, Wong et al found that H pylori eradication treatment **excludes** gastric cancer, because $p > p(H \text{ pylori eradication treatment} \mid \text{gastric cancer})_{\text{lower}}$. This is Wong's first proof, that **H pylori eradication treatment excludes gastric cancer!**

Secondly.

Wong et al. investigated a **subgroup** of patients with no precancerous lesions on presentation. No patient of this subgroup developed gastric cancer during a follow-up of 7.5 years after H pylori eradication treatment compared with those who received placebo (**0 vs. 6; p = 0.02**). Why not?

Result 2.

The probability of the Exclusion relationship is $p(H \text{ pylori eradication treatment} \mid \text{gastric cancer}) = 1$ and thus highly significant! This is Wong's second proof, that **H pylori eradication treatment excludes gastric cancer!**

Conclusions

Wong's study (Department of Medicine, University of Hong Kong, Hong Kong, China. bcywong@hku.hk) has definitely proofed, that H pylori eradication treatment excludes gastric cancer in both, in patients with precancerous lesions and in patients with no precancerous lesions. The study above is only mal-analysed.

In so far, on the one hand, based on the data of Naomi Uemura ¹ et al. (Dr. Uemura at the Department of Gastroenterology, Kure Kyosai Hospital, 2-3-28 Nishichuo, Kure City, Japan, or at n-uemura@mua.biglobe.ne.jp) we know that H pylori is the cause of gastric cancer. On the other hand, Wong's prospective ², randomised ³, placebo ⁴ -controlled, population-based primary prevention study has definitely proofed, that H pylori eradication treatment excludes gastric cancer. In so far, as I have already published, it is definitely proofed, that

Helicobacter pylori is the cause of human gastric cancer.

Ilija Barukcic, Jever, Germany.

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- 1 Naomi **Uemura** et al., Helicobacter pylori infection and the development of gastric cancer. N Engl. J Med. 345 (2001) 11, 784.
 - 2 A **prospective study** is slow and looks forward in time. It is a study in which people are divided into groups. This people are exposed or not exposed to the risk factor of interest before the outcomes have occurred. Example. Let us select a group of subjects. Let watch them for a decade. In contrast, a retrospective study is fast and looks backwards in time. Case control studies never are randomised controlled trials.
 - 3 **Randomised controlled clinical trial** (RCT) avoid any possibility of selection bias in a trial. A group of patients is randomised analogous to tossing a coin into an experimental group and a control group. These groups are followed up for the variables/outcomes of interest.
 - 4 **Placebo** is an inactive intervention received by the participants of the control group, which is as such indistinguishable form the active intervention received by the participants in the experimental group.