CORRESPONDENCE

RE: Effects of Helicobacter pylori Treatment on Gastric Cancer Incidence and Mortality in Subgroups

We read with interest the recent follow-up of the Shangdong Intervention Trial (1). In that study, volunteers received what is now recognized as a regimen with low eradication success and have been followed for more than 15 years without additional treatment. A reduction in cancer risk was confirmed over time. During the interval, the study has been in progress and it has become clear that Helicobacter pylori infection is responsible for the vast majority of gastric cancer and that cancer can be prevented if H. pylori eradication is accomplished before atrophic damage occurs (2). H. pylori eradication at later times also has a benefit in that it stops the progression of damage and the age-related increase in cancer incidence. Even in those at the highest risk (ie, those who have experienced an early gastric cancer), H. pylori eradication reduces the risk of metachronous cancer (3,4). A number of studies have shown that H. pylori–host cell interactions can in themselves cause genetic instability, including double-stranded DNA breakage, providing an additional rational for H. pylori eradication (2,5). In 2013, the Japanese government instituted a program of H. pylori eradication and surveillance to eliminate gastric cancer from that country (6). Nowhere can we find a statement that all the participants in the study have been or are being offered effective anti–H. pylori therapy and confirmation of cure. Effective regimens have been identified in China, and this is now easily accomplished (7). US government–sponsored research carries the legacy of the Tuskegee and the Guatemala syphilis experiments. Continued failure to end this experiment by providing H. pylori eradication, in our opinion, can not be justified and is long overdue.

DAVID Y. GRAHAM
MASAHIRO ASAKA

References

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Notes
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Affiliations of authors: Department of Medicine, Michael E. DeBakey Veterans Affairs Medical Center and Baylor College of Medicine, Houston, TX (DYG); Cancer Preventive Medicine, Hokkaido University Graduate School of Medicine, Hokkaido, Japan (MA).

Correspondence to: David Y. Graham, MD, Department of Medicine, Michael E. DeBakey Veterans Affairs Medical Center and Baylor College of Medicine, Houston, TX (DYG); Cancer Preventive Medicine, Hokkaido University Graduate School of Medicine, Hokkaido, Japan (MA).

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