LETTER TO THE EDITOR

CagA status of *Helicobacter pylori* infection and *p53* gene mutations in gastric adenocarcinoma

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Dear Sir,

We read with great interest the research findings of Dr Shibata et al. (1) regarding the association between *p53* mutation and *CagA*⁺/*H. pylori* infection. The authors searched for *p53* gene mutations by SSCP and, when necessary, by direct sequencing of DNA samples obtained from tumor specimens of patients with gastric adenocarcinoma. *Helicobacter pylori* infection and CagA status were investigated by ELISA for serum IgG antibodies. Undoubtedly, the use of sera obtained prior to cancer diagnosis greatly improves the sensitivity of *H. pylori* detection and CagA status investigation. However, it should be pointed out that the interval between serum collection and cancer diagnosis was extremely variable, from 2 years to more than three decades, which makes possible that some patients have already had premalignant lesions, such as atrophy, an adverse condition for *H. pylori* survival, when serum was obtained. The fact that some samples were drawn many years before carcinoma diagnosis increases the chance of acquiring infection with other *H. pylori* strains, including CagA⁺ ones. Also, it must be mentioned the possibility of changing the bacterium CagA status, what may be achieved by selective loss of parts of the *cag* pathogenicity island (2). It may be added that the combination of *H. pylori*⁺ and CagA⁺/*H. pylori* groups for statistical analysis could lead to a biased result.

We have been investigating *p53* somatic mutations in exons 5–8 by direct sequencing of DNA isolated from distal gastric carcinoma patients. In contrast to the results reported by Shibata et al., we observed that only six of 43 (13.9%) *H. pylori*⁺ patients harbored mutations, one of them in exons 5 and 6. Also, all mutations we detected were substitutions. We cultured *H. pylori* strains from all patients and investigated their *cagA* status by PCR. The frequency of *cagA⁺* strains we found (42/43, 97.7%) was higher than that observed by Shibata et al. (61/74, 82.4%, for distal gastric cancer). It should be highlighted that the high prevalence we detected is meaningful as *cagA⁺* strains were found in ~65% of Brazilian patients with gastritis only (3).

The differences observed between our findings and those reported by Shibata et al. can be due to particular characteristics of each population studied. Although *cagA⁺* *H. pylori* infection has been related to distal gastric carcinoma, we think that there is no clear biological evidence for associating CagA⁺ strains with higher prevalence of *p53* gene mutations in gastric adenocarcinoma yet, at least in some geographical areas.

References