Correspondence

Is there a low prevalence of *Helicobacter pylori* infection in Jordanian patients with gastric cancer?

To the Editor

Awad et al[1] have explored the epidemiologic features of Jordanian patients with diagnosis of gastric cancer (GC) in the period January 2006 to May 2016. A total of 165 patients were included. Regarding tumor types, most of gastric malignancies were primary adenocarcinomas (62%). Regarding *Helicobacter pylori (H.pylori)* infection, in 129 cases, the presence of the bacterium could be assessed, with a prevalence of 49.6%. Intestinal-type and signet ring adenocarcinomas showed a prevalence of *H. pylori* infection of 55.6% and 48.8%, respectively.[1] *Helicobacter pylori* is a slow-growing, micro-aerophilic, Gram-negative bacterium, usually acquired during childhood, whose natural habitat is the luminal surface of the gastric epithelium.[2] Although in 1994 the International Agency for Research on Cancer classified *H. pylori* as a group I carcinogen, namely, a definite cause of GC, to date the bacterium is recognized as a necessary but insufficient cause of GC. This is due to the fact that such a malignancy is a complex, multifactorial disease caused by initiators and other continuator agents. *Helicobacter pylori* causes chronic gastric inflammation that may progress to the precancerous changes of atrophic gastritis and intestinal metaplasia.[2]

Several epidemiologic studies have approached the study of the relationship between *H. pylori* and GC. In a multicentre study, conducted in Northern Italy, we have shown that 82.3% of patients with GC versus 56.5% of controls ($p<0.0001$; odds ratio, 3.58; 95% confidence interval: 2.53-5.07) were seropositive for anti-*H. pylori*. There was no difference between intestinal-type and diffuse-type carcinoma.[3] This high prevalence rate is similar to those reported by several studies.[4] Considering that in Jordan previous studies have found a prevalence of *H. pylori* of approximately 80% in patients with dyspepsia and gastritis or peptic ulcer[5] it is unexpected the low prevalence described by Awad et al in case of GC.[1] It would be interesting to know how the authors could explain these differences. This would enrich their interesting findings.

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Reply from the Author

I would like to thank Dr Pellicano for his interesting and valuable comments and queries. Our study demonstrated that 55.6% of intestinal type and 48.8% of signet ring gastric adenocarcinomas contained *Helicobacter pylori (H. pylori)*.[1] As Pellicano et al pointed out this prevalence is less than the 80% rate of *H. pylori* infection in Jordanian patients reported by Bani Hani et al.[6] Our rate is also less than the prevalence reported by Latif et al[7] who found that 70% of Jordanian patients with acute gastritis and 73% with chronic gastritis had *H. pylori*. One reason for this discrepancy is the method used to detect *H. pylori*. We relied on hematoxylin and eosin stained slides for their detection, whereas Bani Hani et al[6] used modified Giemsa stain and Latif et al[7] used bacteriology as well as histological studies; these 2 methods would have increased the detection rate. However, we believe that the main reason for the discrepancy is that the population in these studies are different. Our patients had gastric cancer, some with advanced disease, whereas none of the patients in the above mentioned studies had gastric carcinoma; they suffered from gastritis or gastric or duodenal ulceration. *Helicobacter pylori* associated gastritis predisposes to gastric cancer through a multistep process that includes gastric atrophy and intestinal metaplasia but it is well known that during this process *H. pylori* disappear because atrophy and intestinal metaplasia create an unfavorable environment for *H. pylori*.[8] Within the population of our study the prevalence of intestinal metaplasia was 34.6%, this is possibly an underestimate because sampling of the patients' specimens was not aimed at detecting metaplasia but at detecting cancer and its stage. The 34.6% rate quoted in the study is the rate of incidentally found intestinal metaplasia foci.

In summary we do not believe that the prevalence of *H. pylori* infection in gastric carcinoma among Jordanian patients is low. There are limitations in our study regarding detecting the actual prevalence of *H. pylori* due to the methodology used but also it is important to recognize that the demonstration of *H. pylori* after cancer development does not reflect the actual prevalence as this bacterium is expected to disappear once cancer has fully developed.

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