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 COMMENTS AND  
 RESPONSES
 

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**Comment on: Jeon et al. *Helicobacter pylori* Infection Is Associated With an Increased Rate of Diabetes. Diabetes Care 2012;35:520–525**

We read with great interest the article by Jeon et al. (1) who, in a prospective cohort study (Sacramento Area Latino Study on Aging), showed that *Helicobacter pylori* (*H. pylori*) infection was associated with increased incidence of diabetes. Although these findings are very interesting, they should be interpreted with caution, and some points should be discussed. The first issue is that the study participants were elderly with a mean age of 68.7 years. It is well known that both incidence of diabetes and *H. pylori* seropositivity increase in older individuals (2). Thus, this association may be due to an incidental finding secondary to the age of the cohort. The second point is that type 2 diabetes is a multifaceted disease, and it is unlikely that it can be explained by one cause. This notion is strengthened by the fact that the results of the study did not show any relation

between *H. pylori* infection and homeostatic model assessment for insulin resistance or other diabetes risk factors such as obesity or central obesity. Insulin resistance has a central role in diabetes and has been reported to be associated with *H. pylori* infection in previous studies (3). The third point is the methods for assessing *H. pylori* infection. The evaluation of anti-*H. pylori* titer (indirect method) has the drawback that despite the bacterial eradication or the fact that it could disappear with the presence of gastric atrophy, the serological positivity can persist. The urea breath test (direct method) appears to have a higher sensitivity and specificity than other methods (2). Therefore, patients who might have a lifelong seropositivity may be negative if studied by a direct method. This resulting misclassification would cause a decrease in the observed association between *H. pylori* and diabetes. The results would be more interesting if authors provide a time table showing us the onset of diabetes in each patient and the *H. pylori* status at the onset of diabetes.

Despite being limited by the above cited points, the relevance of such a study is its originality: being the first to focalize on the effect of *H. pylori* infection on the incidence of type 2 diabetes. If *H. pylori* itself is responsible for the association, this would be of great potential importance for public health as the infection is treatable with a single course of antibiotics, and reinfection rarely occurs (4). Thus, there is still need for large interventional randomized trials in order to

prove a causal association between *H. pylori* infection and diabetes.

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