The Authors Reply: The Influence of *Helicobacter pylori* Eradication on Soluble CD40 Ligand and Oxidative Stress: A Step Forward to Prevent Atherosclerosis

Key words: *H. pylori*, endoscopy, leptin, atherosclerosis

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We thank Eshraghian et al for their interest and valuable comments on our recent publication (1). There are several methods which can be used to diagnose whether a patient is infected with *H. pylori*. Although invasive method remains a better test, noninvasive techniques are widely used in the clinical setting as these methods offer a safe and accurate means of detecting of *H. pylori* infection. No single test is universally accepted as the “gold standard” for the diagnosis of *H. pylori* infection. Endoscopy is an accurate test for diagnosing *H. pylori* as well as the inflammation and ulcers that it causes. Direct comparisons between 14C urea breath test and serology have shown serology to be less accurate in the diagnosis of *H. pylori* infection (2). The Asia-Pacific Consensus Conference on the management of *Helicobacter pylori* infection (3) recommended that in countries with a high incidence of gastric cancer, patients with uninvestigated dyspepsia should be examined by endoscopy. Indeed, Turkey has a relatively high rate of gastric cancer. Gastric cancer is the second leading cause of cancer deaths in men and the third in women. Therefore, it is important to use invasive endoscopic observation and direct histological evaluation of *H. pylori* status to detect gastric cancer (4). According to our current view, if the patient is found to be positive for *H. pylori* infection, endoscopy should be performed.

On the other hand, serum leptin concentrations are reported to be correlated with the percentage of body fat before and after *H. pylori* eradication. Although, leptin levels are also decreased after eradication, weight loss does not significantly alter the amount of circulating leptin relative to total fat stores. Leptin and body fat remains highly correlated after weight loss. Because leptin is produced and secreted by adipose tissue, a decrease in leptin levels with loss of body fat is expected. When obese humans lose fat mass, leptin levels decline; however, the decline in circulating leptin does not always correlate with the amount of weight lost (5). In fact, not all subjects who lost weight showed a decrease in leptin level and in some cases, the leptin levels even increased with weight loss (6). This suggests that there are other factors that influence changes in leptin with weight loss. As mentioned in our manuscript, subjects with diabetes mellitus were excluded. The levels of fasting glucose did not differ between pre and post treatment periods (88.1±8.0 vs 84.9±9.0; p=0.1).

In conclusion, treatment of *H. pylori* infection may not only improve ulcer healing, but it also reduces the presumed atherosclerosis risk by decreasing traditional risk factors such as oxidative stress, fat accumulation and soluble CD40L levels in these patients.

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References