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LETTER TO THE EDITOR CONCERNING "DECREASED SERUM GHRELIN FOLLOWING *HELICOBACTER PYLORI* ERADICATION"

H. Mori[1], H. Suzuki[2]

[1]Division of Gastroenterology and Hepatology, Department of Internal Medicine, Keio University School of Medicine, Tokyo, Japan
[2]Division of Gastroenterology and Hepatology, Department of Internal Medicine, Tokai University School of Medicine, Isehara, Japan

Corresponding Author: Hideki Mori, MD; email: mori-d@keio.jp

Dear Editor,

We read with great interest the paper by Mantero et al[1], which reported decreased ghrelin levels after 12 weeks or later of *Helicobacter pylori* eradication. We previously reported results up to 48 weeks post-eradication[2]; although we found no significant differences over time, ghrelin levels tended to decrease in closed-type atrophic gastritis, which features less pronounced atrophic changes than open-type atrophic gastritis. In contrast, ghrelin levels remained unchanged in patients with open-type atrophic gastritis and were consistently lower than those of patients with closed-type atrophic gastritis[2].

It is notable that Mantero et al[1] confirmed our earlier findings, demonstrating that ghrelin levels in patients with closed-type atrophic gastritis tended to decrease following *H. pylori* eradication[2]. In the Mantero et al's study[1], histological examinations revealed no cases of atrophy extending to the corpus, and all cases were considered closed-type atrophic gastritis, findings that are consistent with our results.

Ghrelin protects the gastric mucosa through its antioxidant effect[3]; the most likely reason for its decreased levels in closed-type atrophic gastritis is the elimination of oxidative stress caused by the chronic inflammation associated with *H. pylori* infection.

We previously showed that *H. pylori* infection increases plasma ghrelin levels in a Mongolian gerbil model[4,5]; our findings suggested that oxidative stress due to persistent infection may affect ghrelin levels. However, it is conceivable that the loss of inflammatory cell infiltration may restore sonic hedgehog- and ghrelin-producing cells; therefore, a future study with a longer follow-up is needed to confirm the effect of *H. pylori* eradication on plasma ghrelin levels[6-10].

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Conflict of Interest

The authors declare they have no conflict of interest.

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