strongly associated with GC progression than the complete type [5].

Hp-induced inflammation, demonstrated in both mice (Houghton's theory) and humans, triggers the migration of bone-marrow–derived stem cells to the gastric mucosa. There, these cells undergo metaplastic and dysplastic changes that can lead to GC, in line with Correa's cascade [6].

Areas exhibiting severe inflammation, IM, atrophy, and GC also show increased mast cell density, correlated with *Hp*-induced gastritis [7]. The CagA and VacA cytotoxins of *Hp* play central roles in promoting oncogenesis, consistently with Correa's model.

The absence of such features in cases of IM suggests a more favorable prognosis. For example, Hwang *et al* [8] reported that IM disappeared ≥5 years after *Hp* eradication. *Hp* eradication protects against GC in patients with IM or dysplasia (follow-up range: 2-26.5 years), and may reverse these conditions Furthermore, incomplete IM regresses within 10 years following *Hp* eradication [5].

Notably, radiofrequency ablation can eradicate incomplete IM [9], and endoscopic grading of IM, as a valuable surveillance tool, reduces the need for routine biopsy sampling [10].

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Authors' reply

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The comments raised in the letter by Prof. Kountouras *et al* [1] are fully consistent with the findings of our study [2]. Nevertheless, several issues discussed in that letter were beyond the scope of our investigation. In this context, 3 points merit attention:

1. In addition to the inflammation induced by *Helicobacter pylori* (*H. pylori*), other bacteria—such as *Porphyromonas gingivalis* (Pg)—have been reported to accelerate the progression of the Correa cascade. In our study, the presence

of Pg in the gastric mucosa was not investigated. However, this possible coexistence does not diminish the primary role of *H. pylori* as an etiopathogenic factor in the evolution of the Correa cascade. In our cohort, a strong correlation was observed between *H. pylori* infection and the development of gastric intestinal metaplasia (GIM), both complete and incomplete types (P<0.001). Our data also support the presence of bile salts in the gastric lumen as an additional pathogenic mechanism contributing to GIM [2-4].

2. Both the CagA and VacA cytotoxins of *H. pylori* play a central role in driving the inflammatory process that promotes gastric carcinogenesis, in accordance with Correa's model [5]. However, the aim of our study was to assess the prevalence of *H. pylori* infection and GIM, rather than to determine the proportion of *H. pylori* strains expressing CagA or VacA.
3. Several studies have shown regression of GIM in the gastric body and antrum 3 to 5 years after successful eradication therapy [6]. The objective of our study, however, was to evaluate the prevalence of GIM in our population. The impact of *H. pylori* eradication on the epidemiology of GIM was not examined.

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