



Risk of gastric and oesophageal adenocarcinomas following discontinuation of long-term proton-pump inhibitor therapy

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Received: 2 December 2022 / Accepted: 7 December 2022 / Published online: 17 December 2022
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Dear Editor,

Holmberg et al. described reduction of gastric and oesophageal adenocarcinomas following discontinuation of proton-pump inhibitor therapy [1]. While PPIs have for long been suspected to cause gastric adenocarcinomas originating from the enterochromaffin-like (ECL) cell via hypergastrinemia [2, 3], a role by PPIs in oesophageal adeno-carcinogenesis has not been accepted. Gastrin, a peptide hormone, affects cell function only via the CCK-2 (gastrin) receptor, which is localized on the ECL cell, and possibly on some cells in Barrett's mucosa [4]. The CCK-2 receptor in Barrett's mucosa was postulated to mediate the oesophageal adeno-carcinogenic effect [1]. However, previous studies have indicated that PPI treatment reduces the risk of oesophageal carcinomas, and the gastrin antagonist netazepide failed to affect Barrett's oesophagus [5]. Furthermore, in just published AGA guidelines on Barrett's oesophagus PPI treatment was still advocated. Therefore, the role of PPIs in oesophageal adeno-carcinogenesis is not clear, and future studies are required.

Data availability Not applicable for this letter.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This comment refers to the article available online at <https://doi.org/10.1007/s00535-022-01930-3>.

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