EDITORIAL

Working towards a better understanding of Helicobacter pylori-related gastric cancer’s biology

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Gastric cancer is a significant cause of cancer-related mortality and morbidity. In terms of disease etiology, it ranks as the fourth and fifth most common contributor of male and female cancer-related death, respectively[1]. A marked geographical variability has been observed for gastric cancer incidence, with the majority of cases reported in Eastern Europe, South America, and Asia[2]. This has been ascribed to environmental (e.g., H. pylori infection), dietary, as well as genetic factors[3].

H. pylori is considered as a definite carcinogen by the International Agency for Research on Cancer, and intestinal metaplasia has been suggested as the intermediate event in the development of H. pylori-related gastric cancer[4]. However, the molecular mechanism of this event needs to be further elucidated. In this issue of AMOR, a report from Ukraine’s Vinnitsa National Pirogov Memorial Medical University by Sergii Vernygorodskyi attempts to improve our current understanding of the molecular mechanism associated with H. pylori-related gastric carcinogenesis[5]. The investigator found that the activation of CDX2, along with the simultaneous inactivation and a decreased number of genes (e.g., SHH, SOX2, and RUNX3) responsible for gastric differentiation, is a probable cause that leads to the appearance of intestinal metaplasia.

Therefore, this study serves as an important catalyst to promote further research on the molecular mechanism and programming of gastric cancer cells, in hopes of gaining new knowledge in the prevention and treatment of gastric cancer.

Conflict of interest

The author declares no potential conflict of interest with respect to the research, authorship, and/or publication of this article.

References


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