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Gastric cancer: pathogenesis, risks, and prevention

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Intestinal (IGCA) and diffuse (DGCA) gastric adenocarcinomas, the two main microscopic subtypes, are dissimilar regarding their epidemiological and demographic characteristics. Both tumor types comprise approximately 40% of all gastric adenocarcinomas. The DGCAs more often occur in young age groups, more often affect the corpus, and are less infrequently associated with atrophic gastritis and intestinal metaplasia than the IGCAs. The risk of both DGCA and IGCA is increased in the presence of *Helicobacter pylori* infection, and the risk rises with increases in grade and extent of atrophic gastritis and intestinal metaplasia. It is likely that the development of up to 80% of the DGCAs and IGCAs can be prevented with early eradication of the *H. pylori* infection. The pathogenesis and morphogenesis of DGCAs are unknown, but the morphogenesis of IGCAs includes identifiable precancerous conditions such as atrophic gastritis and intestinal metaplasia as well as identifiable precancerous lesions (adenomas, dysplasias). Atrophic gastritis is a direct result of the *H. pylori* infestation. Atrophic gastritis, for unknown reasons, appears in more than half of the infected subjects during their lifetime. *H. pylori* gastritis triggers a variety of reactions, with the reaction cascades resulting in errors of the cell genome and ending up as neoplastic tumors.

Key words: gastric cancer, chronic gastritis, *Helicobacter pylori*, atrophic gastritis, carcinogenesis

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