Helicobacter pylori as an oncogenic pathogen, revisited

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Abstract:

Gastric cancer is an inflammation-associated malignancy aetiologically related to infection with the bacterium, Helicobacter pylori, which is considered a necessary but insufficient cause. Unless treated, H. pylori causes life-long acute and chronic gastric inflammation resulting in progressive gastric mucosal damage that may result in gastric cancer. The rate of progression from superficial gastritis, to an atrophic metaplastic mucosa, and ultimately to cancer relates to the virulence of the infecting H. pylori as well as host and environmental factors. H. pylori virulence is a reflection of its propensity to cause severe gastric inflammation. Both mucosal inflammation and H. pylori can cause host genomic instability, including dysregulation of DNA mismatch repair, stimulation of expression of activation-induced cytidine deaminase, abnormal DNA methylation and dysregulation of micro RNAs, which may result in an accumulation of mutations and loss of normal regulation of cell growth. The difference in cancer risk between the most and least virulent H. pylori strain is only approximately 2-fold. Overall, none of the putative virulence factors identified to date have proved to be disease-specific. The presence, severity, extent and duration of inflammation appear to be the most important factors and current evidence suggests that any host, environmental or bacterial factor that reliably enhances the inflammatory response to the H. pylori infection increases the risk of gastric cancer.

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