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Helicobacter pylori and microRNAs - relation with innate immunity and progression of preneoplastic conditions

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Abstract

The accepted paradigm for intestinal-type gastric cancer pathogenesis is a multistep progression from chronic gastritis induced by *Helicobacter pylori* to gastric atrophy, intestinal metaplasia, dysplasia and ultimately gastric cancer. The genetic and molecular mechanisms underlying disease progression are still not completely understood as only a fraction of colonized individuals ever develop neoplasia suggesting that bacterial, host and environmental factors are involved. MicroRNAs are noncoding RNAs that may influence *Helicobacter pylori*-related pathology through the regulation of the transcription and expression of various genes, playing an important role in inflammation, cell proliferation, apoptosis and differentiation. Indeed, *Helicobacter pylori* has been shown to modify microRNA expression in the gastric mucosa and microRNAs are involved in the immune host response to the bacteria and in the regulation of the inflammatory response. MicroRNAs have a key role in the regulation of inflammatory pathways and *Helicobacter pylori* may influence inflammation-mediated gastric carcinogenesis possibly through DNA methylation and epigenetic silencing of tumor suppressor microRNAs. Furthermore, microRNAs influenced by *Helicobacter pylori* also

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