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Resumo	Genetic and epigenetic inactivation of DNA mismatch repair (MMR) genes might lead to modifications in cancer-related gene expression and cancer development. Recently, it has been shown that the infection by *Helicobacter pylori*, the major causative agent of gastric cancer, induces DNA damage and inhibits MMR DNA repair. Also, it has been reported that microRNAs (miRs) have an important role in regulating genomic stability and MMR DNA repair. Thus, the aim of this study was to identify miRs regulating MMR pathway in *H. pylori*-associated gastric carcinogenesis. To address this question, a gastric epithelial cell line and AGS cancer gastric cells were infected with several *H. pylori* strains. MMR gene expression and miRs correlating with *H. pylori* strain infection were evaluated. The results showed that *H. pylori* infection significantly down-regulated the expression of all selected MMR genes. Also, *H. pylori* infection modulated the expression of several miRs (including miR-150-5p, miR-155-5p, and miR-3163), after 4, 8, and 12 h of infection. Computational prediction of candidate miRs and their predicted MMR targeting sites were obtained from TargetScan, mirDB, and MetaCore. The generated data indicated that the selected miRs (miR-150-5p, miR-155-5p, and miR-3163) could possibly target and modulate MMR genes (*POLD3*, *MSH2*, and *MSH3*, respectively). The target validation was performed using mimics and luciferase gene reporter assays. Briefly, this study shows that *H. pylori* impairs MMR DNA repair pathway and identifies miRs that regulate MMR gene expression in gastric cancer. © 2016 Wiley Periodicals, Inc.
Fomento	