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Resumo	Gastric cancer (GC) is the second leading cause of cancer-related mortality worldwide. The disease develops from the accumulation of several genetic and epigenetic changes. Among other risk factors, Helicobacter pylori infection is considered the main driving factor of GC development. H. pylori infection increases DNA damage levels and leads to epigenetic dysregulation, which may favor gastric carcinogenesis. An early step in double-strand break repair is the recruitment of ataxia-telangiectasia mutated serine/threonine kinase (ATM) to the damaged site, where it plays a key role in advancing the DNA damage checkpoint process. H. pylori infection has been associated with the introduction of double-strand breaks in epithelial cells, triggering damage signaling and repair response involving ATM. Thus, the current study analyzed the effect of H. pylori infection on the DNA damage response sensor, ATM, in gastric epithelial cells and in biopsy specimens from patients with GC. In this study, we identified that H. pylori infection stimulated DNA damage, and therefore induced ATM in a virulence factoredependent manner. In addition, we found that H. pylori might activate ATM through histone H3 and H4 hyperacetylation and DNA promoter hypomethylation. Our findings show a mechanism associating ATM signaling induction with H. pylori infection
Fomento	