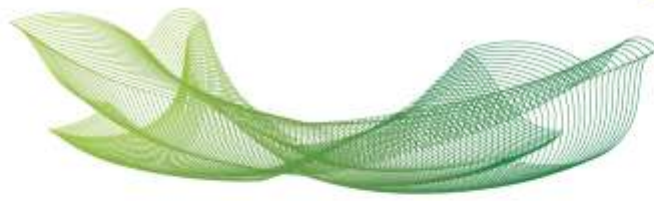




Tipo	Periódico
Título	Effect of <i>Helicobacter pylori</i> Infection on <i>GATA-5</i> and <i>TFF1</i> Regulation, Comparison Between Pediatric and Adult Patients
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Resumo	<p>Background: <i>GATA</i> factors, which constitute a family of transcription regulatory proteins, participate in gastrointestinal development. Trefoil factor 1 (<i>TFF1</i>) plays a crucial role in mucosal defense and healing, and evidence suggests that <i>GATA-5</i> mediated its regulation. Gastric cancer is a multiple-step process triggered by <i>Helicobacter pylori</i> and is characterized by accumulation of molecular and epigenetic alteration. The aim of this study was to evaluate the effect of <i>H. pylori</i> infection on the regulation of <i>GATA-5</i> and <i>TFF1</i> in vitro and in vivo.</p> <p>Results: Infected cells exhibited upregulation of <i>GATA-5</i> and <i>TFF1</i> after 48 h. An increase in <i>GATA-5</i> and <i>TFF1</i> mRNA levels was also found in mice samples after 6 and 12 months of infection, respectively. In human samples, we found an association between <i>H. pylori</i> infection and <i>GATA-5</i> upregulation. In fact, among <i>H. pylori</i>-infected patients, hypermethylation was observed in 45.5% of pediatric samples, in 62.6% of chronic gastritis samples, and in 63% of gastric cancer samples. Regarding <i>TFF1</i>, the expression levels were similar in pediatrics and adults patients, and were independent of <i>H. pylori</i> infection, and the expression of these factors was downregulated in gastric cancer samples. <i>GATA-5</i> promoter methylation was associated with a decrease in <i>TFF1</i> mRNA levels.</p> <p>Conclusions: Our results suggest that the upregulation of <i>GATA-5</i> and <i>TFF1</i> observed in vitro and in vivo may be correlated with a protective effect of the mucosa in response to infection. The epigenetic inactivation of <i>GATA-5</i> observed in human biopsies from</p>



	infected patients may suggest that this alteration is an early event occurring in association with <i>H. pylori</i> infection.
Fomento	