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Effects of helicobacter pylori infection on iron metabolism genes in patients with iron deficiency anaemia

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Abstract

Introduction There is a strong association between *Helicobacter pylori* (HP) infection and iron deficiency anaemia (IDA). The balance between iron uptake, storage and reutilisation is maintained by the liver hormone hepcidin. Paediatric studies have shown that HP disrupts iron function by inducing hepcidin. However, the effects of HP on iron metabolism and tissue hepcidin levels in adults remain controversial. Previously, we have shown by immunohistochemistry that the iron transport protein, ferroportin, was localised to the cytoplasm in the duodenal tissues from the HP-IDA group compared with the IDA group (70% versus 40% respectively) whereas in the control group ferroportin immunoreactivity was mainly localised to the membrane borders.¹ Hence, in this study, we aimed to investigate the tissue expression of hepcidin at the mRNA level and the effects of HP on iron metabolism in duodenal and gastric tissues from IDA patients and controls.

Methods Patients with HP-IDA, IDA and control groups (n=14/group) participated in this study, with ethics approval and informed consent. Duodenal and gastric biopsies were obtained and evaluated by RT-PCR to determine the mRNA expression of hepcidin and iron regulators, including the iron import protein transferrin (TFR-1), divalent metal transporter1 (DMT1), and iron storage protein ferritin.

Results In the duodenal and gastric tissues, DMT-1 and ferritin expression levels were significantly higher in the HP-IDA group compared with IDA and control groups ($p < 0.01$ and $p < 0.001$ respectively). In the duodenal tissues, ferritin expression levels were also significantly higher in the IDA group than the control group ($p < 0.001$). In the gastric tissues from the IDA group, TFR-1 was significantly lower compared with the control group ($p < 0.01$). There were no significant differences between the tissue groups with respect to hepcidin or TFR-1 expression levels in the duodenal tissues.

Conclusions *Helicobacter pylori* infection increases internalisation and storage of iron, and blocks iron transport by altering ferroportin expression independently of hepcidin in IDA patients. It is likely that inflammatory mediators associated with HP infection play a role in disrupting iron function which could represent therapeutic targets.² Therefore, further studies are ongoing in our laboratories to investigate the mechanisms by which HP modulates iron metabolism proteins.

Reference

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