H. felis infected mice is likely to result in increased expressions of genes which control the influx and efflux of iron in gastrointestinal cells. Divalent metal transporter 1 (DMT1) which is negatively controlled by hepcidin is expressed on the apical membrane of gastrointestinal cells and absorbs reduced ferrous iron (Fe^{2+}) from the lumen. There was a significant increase ($p < 0.02$) in *Dmt1* gastric transcript levels in *H. felis*-infected INS-GAS compared to uninfected INS-GAS mice or FVB/N controls. Other iron metabolism gene expression levels were also significantly increased with gastric *H. felis* infection, such as Ferroportin 1 ($p < 0.05$), Transferrin receptor 1 ($p < 0.003$) and Lipocalin 2 ($p < 0.001$). Serum ferritin levels were significantly reduced ($p < 0.001$) in *H. felis*-infected INS-GAS mice compared to uninfected controls at 9 months.

Conclusion This study demonstrates the interplay between hepcidin transcript levels and the expression of host iron metabolism genes. Previous studies have shown that chronic gastric *H. felis* infection in INS-GAS mice results in decreased serum transferrin saturation. This study investigates the molecular basis for previous observations and finds that chronic gastric *Helicobacter* infection results in a significant reduction in gastric hepcidin gene expression levels which coincides with significant increases in gene expression of several proteins implicated in host iron metabolism.

Competing interests None.

Keywords *Helicobacter*, hepcidin, INS-GAS mouse, iron metabolism.

PTU-096 ★ **HOST IRON METABOLISM GENE EXPRESSION IN INS-GAS MOUSE MODEL OF *HELICOBACTER* INFECTION**

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Introduction Iron deficiency is the most common nutritional disorder globally. There is increasing evidence from clinical and population studies for a role of *H. pylori* infection in the aetiology of iron deficiency. Rodent models of *Helicobacter* infection are needed to investigate causal links to iron deficiency in the host. The aim of this study was to investigate the effects of gastric *Helicobacter* infection on host iron metabolism gene expression in hypergastrinaemic INS-GAS mice.

Methods Male transgenic INS-GAS mice were inoculated with *H. felis* or broth only by oral gavage. Uninfected genetic background FVB/N mice were used as controls. Infected mice and uninfected controls were sacrificed at 9 months post-inoculation. Gastric corpus mucosa was stored for total RNA extraction and analysis by quantitative real time PCR. The level of expression of target genes was assessed as relative abundance compared to *Gapdh* control gene by the $\Delta\Delta\text{Ct}$ method. Serum ferritin protein levels were measured by ELISA.

Results *H. felis*-infected INS-GAS mice had a significant reduction in hepcidin transcript levels in the gastric mucosa compared to uninfected INS-GAS mice and FVB/N controls ($p < 0.03$). The reduction in hepcidin hormone expression in