Chronic *Trypanosoma brucei* infection disrupts intestinal health

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Trypanosoma brucei is a protozoan parasite known to colonise multiple tissue niches during chronic infection. The most severe pathology is associated with brain colonisation which results in behavioural disturbances. However, to date, little is known about the impact of chronic *T. brucei* infection on the gastrointestinal (GI) tract. Identification of *T. brucei* in the GI tract has implications for disease progression as a disturbed gut-brain communication axis is now recognised as a key driver of brain pathology in other medical conditions.

Using a murine model of chronic *T. brucei* infection, we identified parasites in various regions along the GI tract. This accumulation resulted in a significant increase in extravascular T-bet$^+$ CD4$^+$ T cells and RORγt$^+$ CD8$^+$ T cells, accompanied by a depletion of B cells in the large intestine lamina propria. These immunological perturbations were accompanied by changes in the microbiome and the repertoire of metabolites generated in the GI tract, along with dysfunction to the GI tract as determined by faecal output, potentially due to impairment of the enteric nervous system.

Interestingly, the immunological changes were also found in the brain parenchyma, potentially mirroring the findings in the GI tract. Our data support the conclusion that chronic *T. brucei* infection alters GI health and could potentially result in modifications in the gut-brain axis. Future work in the lab will focus on addressing whether the lymphocytes identified in the GI and brain are clonally related, and whether the changes in the GI tract are communicated to the brain via the enteric nervous system.