

response. Previous studies showed that *E coli* laden (*E coli* positive) macrophages produce less TNF α compared to those not infected with bacteria (*E coli* negative). T cells are seen clustering around these infected macrophages suggesting T cell activation is altered between the innate and adaptive immune systems.

Aims To use co-localisation immunofluorescent techniques to investigate the immunological profile of T cells associated with *E coli* positive and negative macrophages, and those from control mucosa.

Methods Snap frozen mucosal biopsies were taken at routine colonoscopy from patients with CD and controls with normal colorectal mucosa and cryostat section made. *E coli* positive/negative macrophages were identified using CD-68 staining and a polyclonal anti *E coli* antibody and the T cells were identified using an anti CD3 antibody. In serial sections the T cells were labelled with anti IL10, IL17, TNF α , TGF β , FoxP3 or IL23 receptor and the results analysed using confocal microscopy.

Results The profile of T cells surrounding *E coli* positive macrophages compared to *E coli* negative macrophages demonstrated an elevated number of cells with a regulatory phenotype. More of these T cells expressed IL-10 (67% vs 11%; $p=0.001$) and FoxP3 (14% vs 2%; $p=0.001$). In contrast, increased numbers of pro-inflammatory T cells were seen surrounding *E coli* negative macrophages as measured by cells staining positive for TNF α (22%), IL-17 (26%) and IL23 (30%), which were not seen in association with *E coli* positive macrophages.

Conclusion There is an increased number and specific distribution of regulatory or inflammatory T cells surrounding lamina propria macrophages in CD according to intracellular persistence of *E coli*. The results suggest that despite *E coli* persistence, an appropriate immune containment occurs while it is the macrophages and T cells free of *E coli* which make the main contribution to active inflammation. Further studies are needed to determine the immune interplay between these populations and whether other microbial factors are present in *E coli* negative macrophages.

Competing interests None.

Keywords *E coli*, Lamina propria, Macrophages, T cells.

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IMMUNE PROFILE OF GUT MUCOSA T CELLS ASSOCIATED WITH *E COLI* LADEN MACROPHAGES IN CROHN'S DISEASE

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Introduction There is increasing evidence that a defect in the handling of intracellular bacteria is involved in the pathogenesis of Crohn's disease (CD). In CD, we have previously demonstrated a population of intestinal macrophages harbouring viable *E coli* and it is therefore possible that this failure of bacterial killing contributes to the observed inflammatory