comparable to calprotectin. Microscopic colitis (MC) is a common cause of chronic, watery diarrhoea and represents an inflammatory bowel disease with unknown aetiology and pathogenesis. Diagnosis depends on histological evaluation of colonic biopsies. There is a need for non-invasive diagnostic tools, and this study examines the potential of NGAL as a biomarker in collagenous colitis (CC) as one of the two main histological forms of MC.

Methods: Gene expression of colonic biopsies (n = 9/group) from active CC, budesonide treated CC in clinical remission and healthy controls (HC) was explored by RNA-sequencing analysis. An extended set of colonic biopsies (n = 17–29/group) was also examined by immunohistochemistry (IHC) and *in situ* hybridisation (ISH). Faecal samples from the same patient groups, in addition to samples from patients with irritable bowel disease diarrhoea (IBS-D), were assayed for NGAL and calprotectin (Calpro) by ELISA.

Results: The NGAL gene, *LCN2*, was significantly upregulated in active CC vs. HC (log2 2.694, fold change 6.471) adjusted p-value 0.005) and in pairs of active CC vs. treated CC *LCN2* was significantly downregulated (log2 0.345, fold change –1.536), adjusted p-value 0.04). Both immunohistochemical staining and in situ hybridisation identified increased NGAL expression localised to the mucosal epithelial cells in active CC, compared with an almost absent and scarce expression in HC and treated CC, respectively. There were great individual differences in faecal concentrations of NGAL particularly in the active CC group, but the NGAL concentrations were significantly increased compared with HC, IBS-D and treated CC.

Conclusion: NGAL is upregulated and located mainly to the colonic epithelium of active CC and reduced in clinical remission after budesonide treatment. This is also reflected in the faecal concentrations. We propose NGAL as a valuable biomarker in evaluating the inflammatory activity related to CC and a potential faecal biomarker discriminating CC from IBS-D.

P100

CD161 levels are reduced in all subpopulations of T-cell colonic mucosal lymphocytes in inflammatory bowel disease

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Background: CD161 is a type C lectin expressed in NKs cells and peripheral T cells (TCRγδ and αβ, NKTs), enriched in intestinal populations. Its expression can be modulated by infections and inflammation. MAIT cells are a subset of innate antimicrobial T-cells abundant in the mucosa but their role in immunological regulation is still unknown.

Aim: To measure CD161 levels in subtypes of T-lymphocytes of intestinal mucosa: $CD4^+$, $CD8^+$, double positive (DP,$CD4^+CD8^+$), double negative (DN,$CD4^-CD8^-$), MAIT cells ($CD161^+TCRV\alpha7.2^+$) and intraepithelial cells ($CD103^+$)

Methods: Twenty-six patients with active inflammatory bowel disease (IBD) without immunosuppressive treatment (n = 9 Crohn's disease -CD- colon, 9 CD ileum, n = 8 ulcerative colitis -UC- and 10 healthy controls (paired biopsies of ileum, right and left colon) were included. Lymphocyte subpopulations were analysed with LSRFortessa cytometer. Non-parametric Kruskal–Wallis test was applied. Results are expressed as % of median (25–75%IQI).

Results: In healthy mucosa, we did not find differences related to location in any of CD161 subpopulations except for increase of $CD3^+CD161^+CD103^+$ and decrease of $CD3^+CD161^+CD103^-$ in left colon compared with right colon and ileum. Regarding MAIT cells, a progressive decrease was observed in distal parts of intestine for $CD3^+MAIT^+CD103^+$ while $CD3^+MAIT^+CD103^-$ subpopulation has a specular behaviour; $CD3^+CD8^+MAIT^+$ was increased in ileum compared with colon (Table 1).

Conclusion: There is a regional specialisation for the subset CD103+ of both CD161+CD103+ and MAIT_CD103+ cells in healthy intestine. CD3+CD161+ T cells are reduced in IBD colonic inflammation and could serve as a marker of active IBD but not to sort between CD and UC.

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Biomarkers of elastin degradation are associated with clinical and biochemically active disease in Crohn's disease

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Healthy intestine	Ileum	Right colon	Left colon	p-value
$CD3^+CD161^+CD103^-$	43.2 (33.3-63.0)	41.5 (26.4-55.8)	26.0 (18.7-35.2)	0.034
$CD3^+CD161^+CD103^+$	56.8 (42.2-66.6)	58.4 (44.1-73.5)	73.9 (64.7-81.1)	0.029
$CD3^+MAIT^+CD103^+$	57.1 (41.2-63.5)	42.7 (24.7-52.2)	32.4 (22.4-43.7)	0.042
$CD3^+MAIT^+CD103^-$	45.6 (36.5-58.7)	57.2 (47.7-75.2)	67.5 (57.8-77.5)	0.037
$CD3^+CD8^+MAIT^+$	3.2 (2.2-5.4)	1.7 (1.2-2.5)	1.8 (1.5-2.2)	0.019

In ileal CD vs. healthy ileal mucosa, no differences were observed regarding $CD3^+CD161^+$ and $CD3^+MAIT^+$. A reduction of $CD3^+CD161^+$ was observed in colonic IBD compared with controls due to a reduction of $CD3^+CD4^+CD161^+$, $CD3^+CD8^+CD161^+$ and $CD3^+DN\_CD161^+$ (Table 2).

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Left colon	UC	CD	Healthy	p-value
CD3_CD161	35.9 (28.1-42.7)	39.8 (33.0-55.1)	61.2 (57.7-67.5)	0.004
$CD3^+CD4^+CD161^+$	39.1 (37.2-56.0)	47.0 (38.3-62.5)	71.8 (68.3-77.0)	0.001
$CD3^+CD8^+CD161^+$	14.0 (12.3-25.3)	21.5 (15.6-45.7)	49.1 (33.3-56.9)	0.013
$CD3^+DN\_CD161^+$	16.0 (12.2-24.3)	18.4 (14.3-49.3)	45.6 (23.4-57.8)	0.008