intestinal inflammation in Crohn's disease (CD) patients. Our aim is to identify the cellular subsets present in healthy and inflamed colon, and to describe how individual subsets are regulated in active CD.

Methods: Colon biopsies from a healthy control (HC) and two patients with endoscopically active colonic CD were collected and processed by enzymatic digestion and cell sorting to discard dead cells. Single-cell suspensions were processed by droplet-based protocol 10× chromium system followed by library preparation and sequencing. Sequencing data were aligned and quantified using Cell Ranger. Downstream analysis was performed using the R package Seurat.

Results: scRNA-seq analysis identified 14 transcriptionally different cell subsets in the healthy colon, including diverse types of T cells, plasma cells, mesenchymal cells, epithelial cells and myeloid populations, as well as a subset of MHCII+ CD20+ B cells. Overall, the number of transcriptionally differentiated cellular subsets was greater (16 and 17 subsets per sample) in the two CD colonic samples revealing higher cellular heterogeneity in active CD. In particular, in the HC we identified three T-cell subsets including a CD4+, CD8+ and a smaller subset of intraepithelial lymphocytes. In contrast, samples from CD patients presented four to six clearly differentiated T-cell clusters depending on the patient, with a marked enrichment of cytotoxicity genes (GZMA, GZMB, NKG7, CCL5 and AOAH) and T-regulatory genes (FOXP3, CTLA4, BATF and IL2RA). In addition, the HC sample presented three differentiated subsets of antibody-secreting plasma cells with predominant IgA and IgM production. In contrast, a clear expansion of IgG-producing plasma cells was observed in both CD patients. Moreover, the profiles of specific subsets significantly changed revealing transcriptional regulation within the individual subsets. Specifically, macrophages within the HC mucosa expressed the M2 markers CD163 and CD209 together with a whole signature of 163 additional genes, while in CD, they lost expression of these markers and upregulated CXCL8 and IL1B which were co-expressed with 268 additional genes common to macrophage populations in both CD patients.

Conclusion: We show that sc-RNAseq can be applied to study at a high cellular resolution human intestinal biopsies. Understanding the transcriptomic profile of the CD-related subsets will help dissect disease pathophysiology and provide knowledge for therapeutic targets.

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Regional distribution of TCR Vδ1 and TCR Vδ2 cells in healthy and inflamed mucosa of inflammatory bowel disease

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Background: Vδ1+ and Vδ2+ are TCRγδ lymphocytes of the gut intraepithelial compartment that recognise proteins without help of antigen-presenting cells. Its relevance in inflammatory bowel disease (IBD) is unknown.

Aim: To measure Vδ1+ and Vδ2+ in T-cell subtypes [CD4+, CD8+, double positive (DP,CD4+CD8+), double negative (DN,CD4−CD8−) and CD103+] of healthy and IBD inflammed gut mucosa.

Methods: Twenty-six active IBD patients without immunosuppressants (n = 18 Crohn's disease (CD), eight ulcerative colitis (UC)) and 10 healthy controls (paired biopsies of ileum, right and left colon) were included. Lymphocytes were analysed with LSRFortessa cytometer. Results expressed as % median (25–75%IQI).

Results: Healthy mucosa: reduction in ileum of total CD3+Vδ1+ due to CD3+CD8+Vδ1+ and CD3+DN_Vδ1+

Healthy gut	Ileum	Right colon	Left colon	p-value
CD3+Vδ1+	2,6 (1.2–5.7)	11.0 (7.9–16.1)	7.1 (5.1–9.6)	0.001
CD3+CD8+Vδ1+	2.7 (1.3–5.6)	13.4 (7.4–18.1)	10.6 (6.3–15.6)	0.005
CD3+DN_Vδ1+	29.8 (17.8–39.4)	55.5 (37.7–72.4)	51.2 (45.5–67.7)	0.003

Ileal CD vs. ileal control: CD3+DN_Vδ2+ reduction and increase of Vδ1 and Vδ2 CD4+.

Ileum	CD	Healthy	p-value
CD3+DN_Vδ2+	5.5 (1.9–9.7)	15.9 (9.9–36)	0.021
CD3+Vδ2+DN	44.9 (27.1–56.9)	69.60 (52.9–73.5)	0.027
CD3+Vδ1+CD4+	6.1 (2.7–16.8)	1.8 (0.5–4.0)	0.043
CD3+Vδ2+CD4+	20.8 (6.6–45.3)	2.5 (0–5.6)	0.003

Colonic CD, UC vs. control: CD3+Vδ1+ decrease in CD and UC, and decrease of their subsets CD3+CD8+Vδ1+ and CD3+DN_Vδ1+. CD103+ and CD103− showed specular behaviour with CD3+DN_Vδ1+CD103+ and CD3+CD8+Vδ1+CD103+ decrease and CD3+DN_Vδ1+CD103− and CD3+CD8+Vδ1+CD103− increase both in UC and CD. A similar effect was observed for CD103+ and CD103− of CD3+DN_Vδ2+. CD3+Vδ1+CD4+ was increased in UC and ileal CD.

Conclusion: Reduction of Vδ1+ and Vδ2+, mainly of CD103+, may play a role in IBD pathophysiology by perpetuating inflammation. Increase of CD3+Vδ1+CD4+ in both Ileal CD and UC may compensate this decrease with a selective increase in 'helper' function.

	UC	CD	Healthy	p-value
CD3+Vδ1+	2.1 (1.6–3.0)	2.8 (1.9–20.6)	7.1 (5.0–9.6)	0.002
CD3+CD8+Vδ1+	2.3 (1.1–5.8)	3.7 (2.8–23.7)	10.6 (6.4–15.6)	0.013
CD3+DN_Vδ1	18.9 (16.2–27.5)	28.4 (20.5–61.2)	51.2 (45.5–67.8)	0.001
CD3+CD8+_Vδ1+CD103+	49.9 (17.2–60.9)	45.0 (13.3–84.5)	93.8 (87.7–98.4)	0.002
CD3+CD8+Vδ1+CD103−	50.0 (32.1–82.8)	55.0 (15.5–86.7)	6.1 (1.6–12.3)	0.003
CD3+DN_Vδ1+CD103+	7.4 (2.9–14.9)	48.5 (16.4–82.3)	91.3 (76.3–96.1)	0.001
CD3+DN_Vδ1+CD103−	92.5 (85.1–97.1)	51.5 (17.7–83.6)	8.7 (3.8–23.6)	0.001
CD3+DN_Vδ2+CD103+	6.5 (0.9–8.8)	4.2 (0–30.0)	59.8 (18.5–67.2)	0.011
CD3+DN_Vδ2+CD103−	93.5 (91.2–99.03)	72.2 (33.8–97.9)	37.5 (27.5–52.2)	0.004
CD3+Vδ1+CD4+	4.6 (2.2–15.3)	1.4 (0.7–5.8)	0.6 (0.3–1.5)	0.006