

BA compared with HV. De novo lipogenesis index (DNL) (C16:0/C18:2n-6) was significantly elevated in UC compared with both HV and CD. The ELOVL6 elongase activity index (C18:0/C16:0) and the StA/IA ratio (C18:0/C18:2n-6) in UC were significantly increased compared with HV. AA/EDA ratio (C20:4n-6/C20:2n-6) was increased in both UC and CD. Oral butyrate plus inulin significantly enhanced fecal BPB, reduced elevated *B. fragilis*/*F. prausnitzii* ratio, lowered serum pro-inflammatory SA and 2-HIVA and restored the initially lowered LA and EDA. 85% of UC patients in A1 (butyrate) group demonstrated significant improvement in rectal bleeding and stool frequency by day 14, compared with 55% in A2 group.

Conclusions: The changes in serum metabolome, reflecting metabolic pathways disturbances (glycolysis, TCA cycle, fatty acid metabolism, ketone body metabolism, phenylalanine, tyrosine and tryptophan metabolism, microbial metabolism) are observed in both UC and CD. Some of metabolites and a new metabolomic index (AA/EDA ratio) may be considered as candidate biomarkers of CII. Oral butyrate plus inulin has a prebiotic (butyrogenic) effect, restoring BPB.

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Immunoglobulin G selectively binds pathobionts in the terminal ileum of paediatric IBD patients

H. Armstrong, M. Alipour, R. Valcheva, P. Shah, D. Zaidi, J. Jovel, G. Wong, M. Carroll, H. Huynh, L. Dieleman, E. Wine*
University of Alberta, Edmonton, Canada

Background: Gut microorganisms have been associated with the development of Crohn disease (CD) and ulcerative colitis (UC) in a number of studies to date; however, most studies focused on identifying microbial changes have examined only samples from stool or inflamed areas of the intestine, limiting the ability to differentiate between cause and effect. Therefore, we focused on bacteria from non-inflamed areas and sought to develop a method to specifically identify pathobionts. Our hypothesis was that immunoglobulin (Ig) G, an antibody naturally formed in response to invasive microbes, can be used as a novel marker of pathobionts in IBD patients.

Methods: Building on our recent work that demonstrated altered composition and diversity of bacteria from the uninflamed terminal ileum (TI) in paediatric UC patients, we focused on microbes proximal to diseased areas as we believe they may drive inflammation distally. Intestinal washes were collected during endoscopy from the TI of paediatric IBD patients and non-IBD controls. Using fluorescence-activated cell sorting (FACS) we separated IgG-bound (IgG+) from unbound (IgG-) bacteria, extracted their DNA, and analysed composition by 16S and metagenomic sequencing using the Illumina MiSeq platform. We then confirmed virulence of specific IgG-bound bacteria in-vitro.

Results: FACS was efficient in separating IgG+ from IgG- bacteria; the method was validated by Image Cytometry. Greater numbers of IgG-bound microbes were observed in CD (2-fold) and UC (1.5-fold) patients, compared with non-IBD. Interestingly, while there was relatively little difference in species abundance between IBD and non-IBD patients, IgG binding favoured specific Bacteroidetes, Firmicutes, and Proteobacteria in CD, and specific Bacteroidetes and Proteobacteria in UC. Many of these changes were more prominent in moderate/severe disease than in cases that were mild/in remission. When examined in-vitro, selective IgG+ species displayed pro-inflammatory effects and invasive potential, supporting their pathobiont potential.

Conclusions: Using FACS to isolate IgG-bound bacteria collected from luminal washes in children with IBD we selectively identified previously unrecognised mucosa-associated microbes with apparent pathobiont qualities, associated with IBD. Further characterizing the role of specific bacterial species bound by IgG may provide insight into IBD pathogenesis and could assist in directing therapies to those patients most likely to respond, including by use of microbe-altering treatments.

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Quantitative microbiome profiling changes the described dysbiotic state in inflammatory bowel disease

C. Caenepeel^{1,2*}, S. Vieira-Silva³, J. Sabino^{1,2}, K. Machiels¹, G. Falony³, M. Ferrante^{1,2}, G. Van Assche^{1,2}, J. Raes³, S. Vermeire^{1,2}

¹KU Leuven, The Department of Chronic Diseases, Metabolism and Ageing (CHROMETA), Leuven, Belgium, ²UZ Leuven, Department of Gastroenterology and Hepatology, Leuven, Belgium, ³KU Leuven, Rega Institute, Department of Microbiology and Immunology, Leuven, Belgium

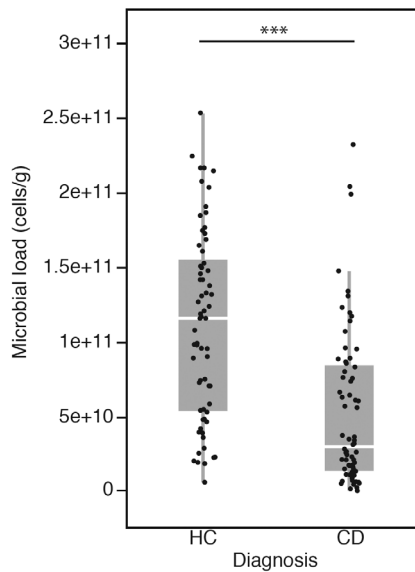
Background: Gut microbiota play a crucial role in inflammatory bowel disease (IBD). The usage of culture-independent techniques lead to the identification of dysbiosis in IBD, but generate only relative microbiome profiles (RMP) with no ability to provide information on the extent or directionality of changes in taxa abundances. Quantitative microbiome profiling (QMP) combines microbiome sequencing with flow cytometric counting of microbial cells to quantitatively assess microbiota variation (Vandeputte *et al.*, 2017). We aimed to investigate the differences in microbial load in patients with active Crohn's disease (CD). We compared the distribution of the different enterotypes and hypothesised that microbial load may be associated with the inflammatory status in IBD.

Methods: Fecal samples of 69 CD patients with endoscopically active disease were collected prior to biological therapy. Fecal samples of 66 healthy controls (HC) from the Flemish Gut Flora Project (FGFP) were used as comparison. Microbiota phylogenetic profiling was conducted by using 16S rRNA gene amplicon sequencing, and microbial loads of frozen fecal samples were measured using flow cytometry. These cell counts were used to transform the sequencing data into an absolute microbiome abundance matrix that allowed QMP by modifying sequencing depth rarefying procedures and generated QMP expressed as number of cells per gram faces.

Results: Our flow cytometric analysis data confirm a significant lower microbial load (Wilcoxon $r = -0.49$; $p < 0.001$) in active CD, up to a hundred-fold lower, compared with HC (Figure 1). Enterotypes distribution varies between the active CD and HC. Notably, 10.6% of the FGFP samples were typed as Bacteroides2, compared with a much higher prevalence of 88.2% in our patients with active CD. Furthermore, we compared the microbial load between clinical responders (defined as HBI score of ≤ 4 points) and non-responders (no decrease from study baseline HBI score of at least four points) prior to biological therapy. Baseline microbial density was not significantly different between these clinical responders ($n = 54$) and non-responders ($n = 15$) (Wilcoxon $p = 0.48$).

Conclusions: The introduction of QMP, as recently published, leads to a revisiting of the known dysbiosis in IBD microbiome research. Using QMP, we confirm previous observations of lower microbial loads and a high Bacteroides2 prevalence in CD, even more

pronounced due to the active disease state. No difference in microbial density is seen between clinical responders and non-responders, but further investigation is needed.



Microbial Load in healthy controls vs. Crohn's disease

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Vegetarian and gluten-free diet in patients with IBD—associated with a different microbiota compared with omnivore IBD patients

P. Schreiner^{1*}, B. Yilmaz², Y. Franc³, J.-B. Rossel³, B. Misselwitz¹, M. Scharl¹, J. Zeitz⁴, P. Frei⁵, T. Greuter¹, S. Vavricka⁶, V. Pittet³, A. Siebenhüner⁷, P. Juillerat⁸, R. von Känel⁹, A. Macpherson², G. Rogler¹, L. Biedermann¹

¹UniversityHospital of Zurich, Gastroenterology and Hepatology, Zurich, Switzerland, ²University of Bern, Department of Clinical Research Gastroenterology and Mucosal Immunology, Bern, Switzerland, ³Lausanne University Hospital, Institute of Social and Preventive Medicine, Lausanne, Switzerland, ⁴Klinik Hirslanden, Center of Gastroenterology, Zurich, Switzerland, ⁵Gastroenterology Bethanien, Zurich, Switzerland, ⁶Center of Gastroenterology and Hepatology, Zurich, Switzerland, ⁷UniversityHospital of Zurich, Hematology and Oncology, Zurich, Switzerland, ⁸Inselspital Bern University Hospital, Department of Visceral Surgery and Medicine, Bern, Switzerland, ⁹Clinic Barmelweid, Department of Psychosomatic Medicine, Barmelweid, Switzerland

Background: Several studies demonstrate a lower diversity in the gut microbiota of patients with inflammatory bowel disease (IBD). Microbial alterations induced by dietary changes are amongst the key suspected responsible environmental factors to promote an increase in the incidence of IBD, and may adversely impact the course of established disease. Subsequent to our already presented results of lower psychosocial wellbeing in vegetarian diet (VD) and gluten-free diet (GFD) IBD patients (Poster 711, ECCO 2017) we investigated comparative microbial composition of IBD patients according to diet.

Methods: Dietary pattern was analysed in a total of 1656 IBD patients from the Swiss Inflammatory Bowel Disease Cohort Study

between 2006 and 2015. Microbiota composition was analysed in 149 patients, including 12 vegetarian patients and 14 patients following a GFD by means of high-throughput sequencing. Within the majority of meat-eating patients, we further compared the microbiota of the low vs. high-meat-intake patients (i.e. ≤ 4 vs. >4 days per week).

Results: In the alpha diversity analysis (Shannon) we observed a significant difference between GFD and meat-eating Crohn's disease (CD) patients with lower species richness in meat-eating patients ($p = 0.026$). In the ulcerative colitis (UC) group no significant difference in the alpha diversity was seen. Both CD and UC revealed significantly different β diversity in meat-eating patients compared with their VD and GFD counterparts. Bacterial taxa did not differ according to diet types in CD, whereas within meat-eating CD patients the following significant differences in taxa were found: Faecalibacterium, Bilophila and Butyricimonas taxa were found to be less abundant in the high-meat-intake CD group. On the other hand, there was a higher relative abundance of Eubacterium (family Erysipelotrichaceae), Enterococcus, Lactobacillus, Lactococcus, Fusobacterium, and Tepidimonas in the high-meat-intake CD group. In UC meat-eating patients there was a significantly higher relative abundance of Ruminococcus compared with GFD and VD patients. The high-meat-intake UC patients had a higher relative abundance of Lachnospira, Ruminococcus, and Parabacteroides.

Conclusions: The gut microbiota composition in meat-eating IBD patients is significantly different compared with those following a VD or GFD. The potentially anti-inflammatory taxa Faecalibacterium and Butyricimonas were reduced and the inflammatory taxa Erysipelotrichaceae and Enterococcus were increased in the high-meat-intake CD patients. Our results demonstrate several bacterial changes in regularly meat-eating IBD patients compared with VD or GFD, specifically lower species richness with a dose-response effect in meat-eating CD patients.

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The gut microbiota composition and metabolic activity of HLA-B27 transgenic rats with gut inflammation resembles the dysbiotic characteristics of human inflammatory bowel disease

V. Svolos^{1*}, B. Nichols¹, D. Bolognini², C. Quince³, U.Z. Ijaz⁴, R.T. Papadopoulou¹, C. Ansalone⁵, J. Salmond⁶, R. Klopffleisch⁷, D.R. Gaya⁸, R.K. Russell⁹, R. Hansen⁹, K. Gerasimidis¹, S. Milling⁵
¹Human Nutrition, School of Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow Royal Infirmary, Glasgow, UK, ²Centre for Translational Pharmacology, Institute of Molecular, Cell and Systems Biology, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK, ³Warwick Medical School, University of Warwick, Warwick, UK, ⁴School of Engineering, University of Glasgow, Glasgow, UK, ⁵Institute of Infection, Immunity and Inflammation, School of Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK, ⁶Queen Elizabeth University Hospital, Glasgow, UK, ⁷Institute of Veterinary Pathology, Freie Universitaet, Berlin, Germany, ⁸Department of Gastroenterology, Glasgow Royal Infirmary, Glasgow, UK, ⁹Department of Paediatric Gastroenterology, Hepatology and Nutrition, Royal Hospital for Children, Glasgow, UK