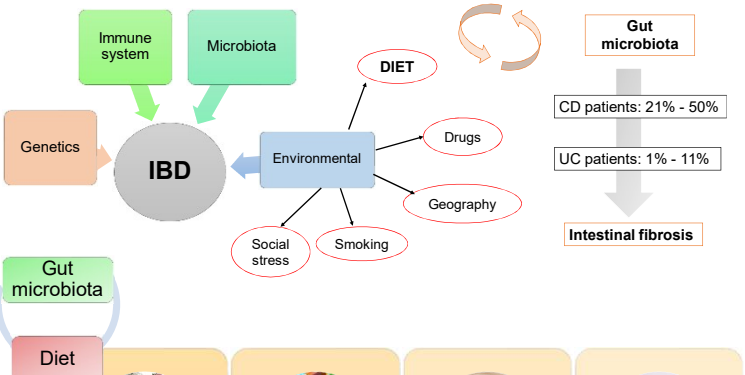
Sucralose and maltodextrin affect differently the gut microbiota of healthy individuals and IBD patients

I. Gonza*[1], E. Goya – Jorge[1], C. Douny[2], M.L. Scippo[2], E. Louis[3] and V. Delcenserie[1]

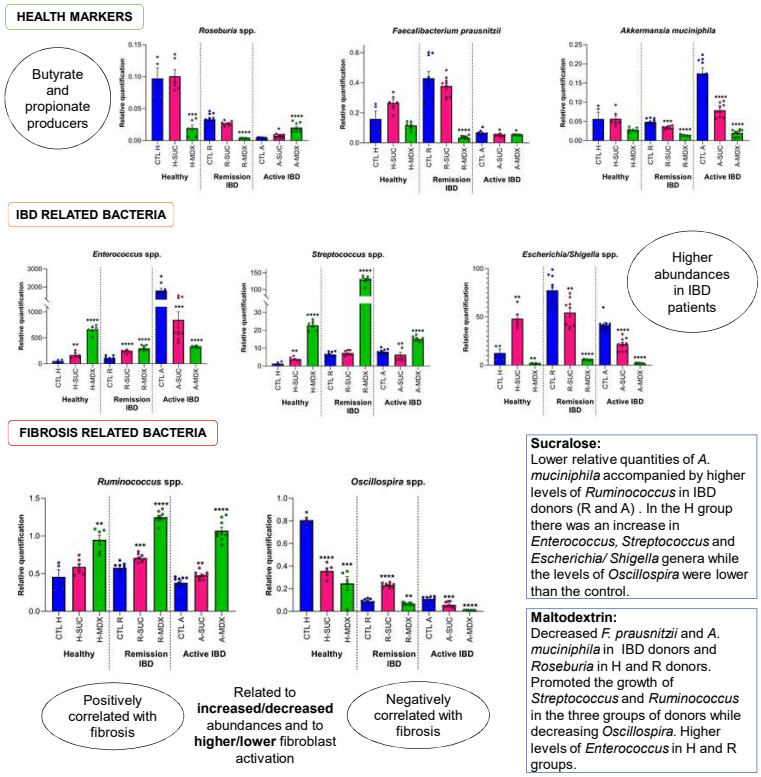
1: Food Quality Management, Food Science Department, FARAH, University of Liège , 2: Food Analysis, Food Science Department, FARAH, University of Liège, 3: Department of Gastroenterology, University and CHU of Liège

***Contact:** iegonza@uliege.be*

CONTEXT



RESULTS – MICROBIAL CHANGES

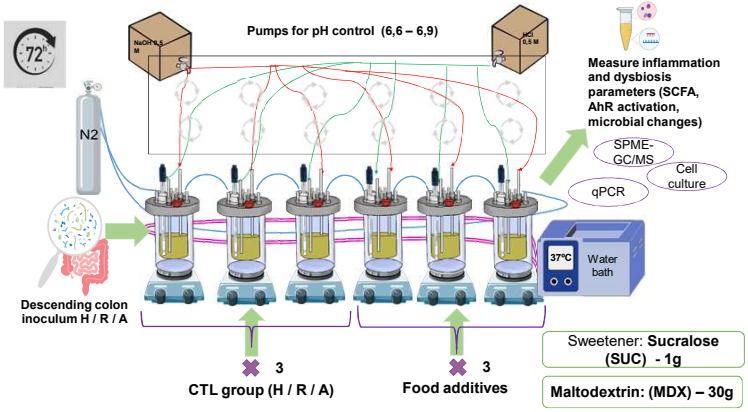


Sucralose:
Lower relative quantities of *A. muciniphila* accompanied by higher levels of *Ruminococcus* in IBD donors (R and A) . In the H group there was an increase in *Enterococcus*, *Streptococcus* and *Escherichia/ Shigella* genera while the levels of *Oscillospira* were lower than the control.

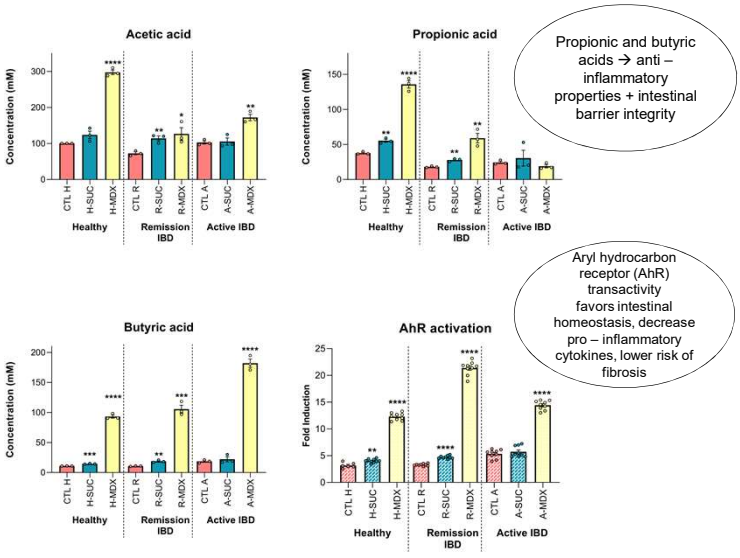
Maltodextrin:
Decreased *F. prausnitzii* and *A. muciniphila* in IBD donors and *Roseburia* in H and R donors. Promoted the growth of *Streptococcus* and *Ruminococcus* in the three groups of donors while decreasing *Oscillospira*. Higher levels of *Enterococcus* in H and R groups.

EXPERIMENTAL DESIGN

Healthy (n = 5) | **Remission of IBD** (n = 5) | **Active IBD** (n = 5)



RESULTS – MICROBIAL ACTIVITY



Sucralose:
Increased propionic and butyric acid production in H and R groups as well as higher AhR transactivity were observed. Acetic acid was also increased in the R group.

Maltodextrin:
Increased acetic and butyric acid production in the three groups of donors and propionic acid in H and R donors. A higher AhR transactivity was observed in the three groups of donors.

CONCLUDING REMARKS

The microbiota from IBD patients showed higher susceptibility to the addition of food additives than healthy individuals. The depletion of *F. prausnitzii* and *Roseburia* is a common characteristic in IBD patients [1–4]. However, the deleterious effect observed on butyrate – producing bacteria such as *F. prausnitzii* and *Roseburia* in the presence of SUC and MDX was stronger when food additives were tested on the microbiota from patients in remission of IBD. The positive association between the abundance of facultative anaerobes as *Enterococcus* and *Streptococcus* and IBD (both CD and UC) has been largely reported [1,5–7]. The increase observed in these bacterial groups with SUC and MDX in our donors, could mean that they are at higher risk to develop or exacerbate IBD. This was specially remarkable in the H group. The negative impact of food additives on *A. muciniphila*, especially in the R and A donors, is of great interest as this bacterium has been negatively associated with inflammation biomarkers[8], so food additives could aggravate the inflammatory response in IBD patients. The behavior of *Ruminococcus* and *Oscillospira* face to SUC and MDX in the H group could lead to facilitate fibrosis induction. The mucolytic bacteria as *Ruminococcus* and *M. schaedleri* were found directly and positively associated to fibrosis induction as they were capable to modulate the fibroblast function while *Oscillospira* mitigate fibroblast activation[9].

This study raise the importance of analyzing the effects of SUC and MDX on the gut microbiota from healthy individuals and donors suffering from IBD (active and remission periods) over longer time periods in order to identify their specific role in the onset or exacerbation of dysbiosis as well as the assessment of other inflammation markers taking into account the digestion process.

REFERENCES:

1. Zhu, S. *et al.* Composition and diverse differences of intestinal microbiota in ulcerative colitis patients. *Front. Cell. Infect. Microbiol.* **12**, 1–13 (2022).
2. Ma, Y. *et al.* Metagenome Analysis of Intestinal Bacteria in Healthy People, Patients With Inflammatory Bowel Disease and Colorectal Cancer. *Front. Cell. Infect. Microbiol.* **11**, 1–12 (2021).
3. Clooney, A. G. *et al.* Ranking microbiome variance in inflammatory bowel disease: A large longitudinal intercontinental study. *Gut* **70**, 499–510 (2021).
4. Pisani, A. *et al.* Dysbiosis in the Gut Microbiota in Patients with In fl ammatory. *Microbiol. Spectr.* **10**, (2022).
5. Mancabelli, L. *et al.* Identification of universal gut microbial biomarkers of common human intestinal diseases by meta-analysis. *FEMS Microbiol. Ecol.* **93**, 1–10 (2017).
6. Abdel-Rahman, L. I. H. & Morgan, X. C. Searching for a Consensus Among Inflammatory Bowel Disease Studies: A Systematic Meta-Analysis. *Inflamm. Bowel Dis.* **29**, 125–139 (2023).
7. Chang, T. E. *et al.* Fecal microbiota profile in patients with inflammatory bowel disease in Taiwan. *J. Chinese Med. Assoc.* **84**, 580–587 (2021).
8. Chen, L., Reynolds, C., David, R. & Peace Brewer, A. Development of an Index Score for Intestinal Inflammation-Associated Dysbiosis Using Real-World Stool Test Results. *Dig. Dis. Sci.* **65**, 1111–1124 (2020).
9. Jacob, N. *et al.* Inflammation-independent TL1A-mediated intestinal fibrosis is dependent on the gut microbiome. *Mucosal Immunol.* **11**, 1466–1476 (2018).