

## Letter to the editor

## ***Faecalibacterium prausnitzii* is not decreased in symptomatic uncomplicated diverticular disease of the colon**

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Received June 21, 2022; Accepted August 13, 2022; Published online in J-STAGE August 30, 2022

**In this letter, assessment of the amount of fecal *Faecalibacterium prausnitzii* in symptomatic uncomplicated diverticular disease (SUDD) is described. Among 44 consecutive patients, comprising 15 SUDD patients, 13 patients with asymptomatic diverticulosis (AD), and 16 healthy controls (HC), the fecal amount of *Faecalibacterium prausnitzii* was not found to be significantly different between HC, AD and SUDD subjects ( $p=0.871$ ). Moreover, its count in the HC microbiota ( $-4.57 \pm 2.15$ ) was lower compared with those in the AD ( $-4.11 \pm 1.03$ ) and SUDD subjects ( $-4.03 \pm 1.299$ ). This behavior seems to be different from that occurring in inflammatory bowel disease (IBD) and similar to that of other mucin-degrading species in a SUDD setting.**

**Key words:** diverticular disease, fecal microbiota, *Faecalibacterium prausnitzii*

### TO THE EDITOR:

About one-fifth of patients harboring colonic diverticula may develop symptomatic uncomplicated diverticular disease (SUDD) [1]. It is generally thought that microbial imbalance is a milestone for the occurrence of symptoms [1]. However, knowledge about the microbial imbalance in these patients is still under debate, since most of the current knowledge on gut microbiota is linked only to the classical inflammatory bowel disease (IBD). In particular, mucin-degrading species (including *Faecalibacterium prausnitzii*) are decreased in IBD [2–4], whereas some of them (such as *Akkermansia muciniphila* and *Roseburia hominis*) have been found to increase in SUDD [5, 6]. Since the behavior of *F. prausnitzii* is not well known [6], we assessed whether *F. prausnitzii* is also decreased in SUDD patients.

Forty-four consecutive women, comprising 15 SUDD patients, 13 patients with asymptomatic diverticulosis (AD), and 16 healthy controls (HC), who lived in the same geographical area, had not used antibiotics in the three months before enrolment, and

did not have ongoing or past acute complicated or uncomplicated diverticulitis were enrolled. SUDD was diagnosed according to well-defined criteria [1].

Bacterial DNA from fecal samples was extracted using real-time polymerase chain reaction (PCR) with appropriate primers to quantify *F. prausnitzii* species. Standard curves were created using serial tenfold dilutions of bacterial DNA extracted from *F. prausnitzii*. All samples were analyzed in duplicate in two independent real-time PCR assays. A  $p$ -value  $<0.05$  was considered significant. The protocol was approved by the Ethics Committee of “S. Eugenio” Hospital.

The amount of *F. prausnitzii* was not significantly different among HC, AD, and SUDD subjects ( $p=0.271$ ). In particular, its count in the HC microbiota ( $-4.57 \pm 2.15$ ) was lower compared with those in the AD ( $-4.11 \pm 1.03$ ) and SUDD subjects ( $-4.03 \pm 1.299$ ; Fig. 1).

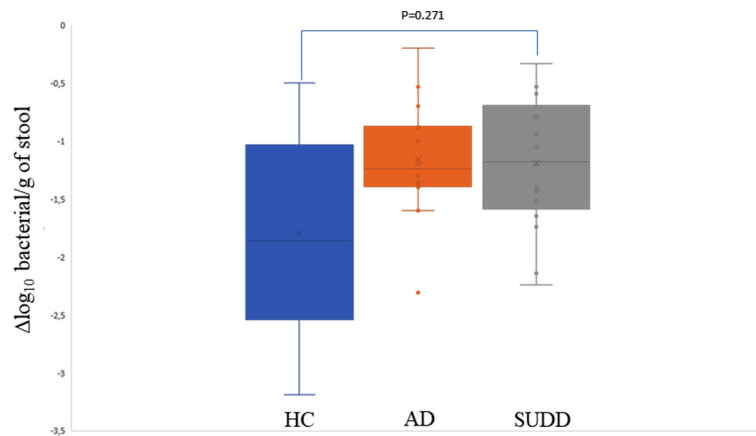
Our results revealed for the first time that relative abundance of *F. prausnitzii* was not decreased in SUDD. On the contrary, we found that it was increased in fecal samples of both AD and

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**Fig. 1.** Quantification of *Faecalibacterium prausnitzii* in healthy control (HC), asymptomatic diverticulosis (AD), and symptomatic uncomplicated diverticular disease (SUDD) subjects. The qPCR results were plotted as a box and whisker plot. Boxes (containing 50% of all values) indicate the median (horizontal line across the middle of the box) and interquartile range.

SUDD patients in comparison to HC patients. This behavior seems to be opposite to that occurring in IBD [4]. Why this occurs is unknown, and the metabolic impact of this behavior of these mucin-degrading and short-chain fatty acid (SCFA)-producer bacterial species in SUDD is also unknown. In the IBD field, it has been hypothesized that the reduction of these species is associated with a reduction in SCFAs, with a significant metabolic imbalance in the intestinal cells [4]. However, a reduction in several SCFAs has been identified that paralleled a significant increase in the abundance of *A. muciniphila* in SUDD patients [5], an imbalance that was restored by adequate treatment [7]. If the mucin-degrading and SCFA-producing bacterial species are not decreased and if SCFAs can be decreased, these bacterial species may cause higher mucus degradation in SUDD patients, with microbial imbalance and low production of SCFAs. This hypothesis seems to be confirmed by the observation that SCFA production from peptides and organic acids, mediated by saccharides, appears to be the most important metabolic parameter for metabolome restoration during the treatment of SUDD and to parallel the reduction in the amount of *A. muciniphila* [7]. Further studies are therefore warranted to confirm these findings.

### AUTHOR CONTRIBUTIONS

Antonio TURSI planned and conducted the study; Paola MASTROMARINO, Daniela CAPOBIANCO, Walter ELISEI, GianMarco GIORGETTI, Federica FABIOCCHI, and Giovanni BRANDIMARTE collected data; Antonio TURSI, Paola MASTROMARINO, Daniela CAPOBIANCO, Giuseppe CAMPAGNA, Marcello PICCHIO and Walter ELISEI interpreted data; Antonio TURSI drafted the manuscript; and Antonio

TURSI, Paola MASTROMARINO, Daniela CAPOBIANCO, Walter ELISEI, Giuseppe CAMPAGNA, Marcello PICCHIO, GianMarco GIORGETTI, Federica FABIOCCHI, and Giovanni BRANDIMARTE approved the final draft submitted.

### CONFLICT OF INTEREST

The authors declare they do not have any conflict of interest.

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