

Gut Microbiota May Predict the Short-Term Recurrence of Symptomatic Uncomplicated Diverticular Disease

Antonio Tursi^{1,2*}, Giorgia Procaccianti^{3*}, Silvia Turrone^{3,4}, Rudi De Bastiani⁵, Federica D'Amico³, Leonardo Allegretta⁶, Natale Antonino⁷, Elisabetta Baldi⁵, Carlo Casamassima⁸, Giovanni Casella⁵, Mario Ciuffi⁵, Marco De Bastiani⁵, Lorenzo Lazzarotto⁵, Claudio Licci⁹, Maurizio Mancuso⁵, Antonio Penna¹⁰, Giuseppe Pranzo¹¹, Guido Sanna⁵, Cesare Tosetti⁵, Maria Zamparella⁵, Marcello Picchio¹²

1) Territorial Gastroenterology Service, Barletta-Andria-Trani Local Health Agency, Andria; 2) Dept. of Medical and Surgical Sciences, School of Medicine, Catholic University, Rome; 3) Unit of Microbiome Science and Biotechnology, Department of Pharmacy and Biotechnology, University of Bologna, Bologna; 4) IRCCS Azienda Ospedaliero-Universitaria di Bologna, Bologna; 5) GIGA-CP Italian Association for Primary Care Gastroenterology, Feltre; 6) Division of Gastroenterology, Santa Caterina Novella Hospital, Galatina; 7) General Practitioner, Private Practice Gastroenterologist, Bisceglie; 8) General Practitioner, Private Practice Gastroenterologist, San Ferdinando di Puglia; 9) Private Practice Gastroenterologist, Monopoli; 10) Private Practice Gastroenterologist, Bari; 11) Ambulatory for IBD Treatment, Valle D'Itria Hospital, Martina Franca; 12) Division of Surgery, P. Colombo Hospital, Velletri, Italy

Address for correspondence:

Antonio Tursi, MD
Via Torino, 49 - 76123 Andria (BT), Italy
antotursi@tiscali.it

Received: 07.09.2025

Accepted: 01.12.2025

*contributed equally

ABSTRACT

Background & Aims: Symptomatic Uncomplicated Diverticular Disease (SUDD) may recur, but it is still unknown whether the gut microbiota (GM) may play a role. We aimed to analyze whether GM plays a role in short-term (6 months) relapse in SUDD patients for whom GM data were retrospectively available.

Methods: GM was profiled using 16S rRNA amplicon sequencing in SUDD patients who did or did not relapse within 6 months of diagnosis, as well as in patients with asymptomatic diverticulosis (AD) who served as controls.

Results: Of 66 SUDD patients, 5 experienced SUDD recurrence within 6 months of diagnosis. Relapsers and non-relapsers did not differ in GM alpha and beta diversity, either between each other or compared to controls. However, taxonomically the phylum Verrucomicrobiota, the family Erysipelotrichaceae and the genera Prevotellaceae NK3B31 group, Roseburia and Holdemanella were enriched in SUDD relapsers, while Ruminococcaceae and Sutterella were enriched in non-relapsers ($p < 0.05$).

Conclusions: This study suggests an association between recurrent SUDD and pro-inflammatory taxa that have previously been implicated in severe SUDD and other gut disturbances. Larger and longer cohort studies are warranted to further investigate the potential predictive role of GM in short-term recurrence of SUDD and to inform precise intervention strategies.

Key words: symptomatic uncomplicated diverticular disease – SUDD – disease recurrence – gut microbiota.

Abbreviations: AD: asymptomatic diverticulosis; FDR: false discovery rate; GM: gut microbiota; IBD: inflammatory bowel disease; SUDD: symptomatic uncomplicated diverticular disease.

INTRODUCTION

The so-called symptomatic uncomplicated diverticular disease (SUDD) is the main clinical syndrome affecting patients with diverticulosis of the colon [1]. Its prevalence is limited to 10-15% of patients having diverticulosis in both the Western and Eastern worlds [2, 3], and its diagnosis is sometimes delayed due to overlapping with irritable bowel syndrome [4]. Symptomatic uncomplicated diverticular disease often recurs, with more than 91% of patients experiencing a relapse within 12 months, and up to 8% of these

patients may develop acute diverticulitis in the long term [5]. Until now, it is not well understood why patients having SUDD may often recur.

In recent years, some studies identified gut microbiota (GM) perturbations in SUDD [6]. Although limited by the small sample size, these studies were the first to focus on the potential role of GM perturbation in the pathogenesis of the disease [6]. More recently, we found that GM changes are linked to the severity of abdominal pain, which is the main symptom characterizing SUDD [1]. In particular, potentially harmful taxa (such as *Proteobacteria*, *Streptococcaceae*, and *Megasphaera*) were overrepresented in patients experiencing more severe abdominal pain, and may contribute to inflammation and symptom exacerbation [7]. Interestingly, GM changes in these patients (particularly, an increase in *Enterobacteriaceae* and *Megasphaera*) have also been found to be closely associated with increasing endoscopic severity of the disease [8]. Despite these interesting findings, it is still unclear whether GM perturbation may predict the clinical outcomes of SUDD, including recurrence.

Aim of this study was therefore to assess potential differences in GM between SUDD patients with and without disease recurrence, as compared to patients with asymptomatic diverticulosis (AD) as controls.

METHODS

We retrospectively assessed SUDD and AD patients, who had stool samples collected by fecal swab for microbiota profiling and stored at the Unit of Microbiome Science and Biotechnology, Department of Pharmacy and Biotechnology, University of Bologna (Bologna, Italy). All fecal swabs were collected using the eNAT® System (Copan, Brescia, Italy) and stored at -80°C. We identified patients who had experienced a recurrence of SUDD after diagnosis.

The study was conducted in accordance with clinical practice guidelines and the principles of the [Declaration of Helsinki](#). All patients gave written informed consent before undergoing endoscopy and/or CT scan and/or fecal sampling. Ethics committee approval for this retrospective study was obtained from the Azienda Ospedaliero-Universitaria “Ospedali Riuniti”, Foggia, Italy (PROT. 164/CE/2023, October 23, 2023).

Inclusion criteria were: males and females >18 years; colonic diverticulosis diagnosed by colonoscopy or imaging (abdominal computed tomography and/or ultrasonography); diagnosis of SUDD (defined as left-lower and long-lasting quadrant pain in patients with diverticulosis) [9, 10], during the 6 months prior to fecal collection; possibility of retrospectively reconstructing the symptoms and clinical history of patients with SUDD; GM assessment performed at the Unit of Microbiome Science and Biotechnology, Department of Pharmacy and Biotechnology, University of Bologna (Bologna, Italy). SUDD short-term recurrence was defined as recurrence of left-lower and long-lasting quadrant pain in patients with prior diagnosis of SUDD, in whom acute diverticulitis occurrence was excluded by abdominal CT scan or intestinal ultrasound.

Exclusion criteria were: radiological signs (by abdominal computed tomography and/or ultrasonography) of acute diverticulitis (defined as inflammation of the colonic wall harboring diverticula with fat stranding, and with or without complications such as abscesses, stenosis or fistulas, namely uncomplicated or complicated diverticulitis) [10]; inflammatory bowel disease (IBD); irritable bowel syndrome; ischemic colitis; prior colonic resection; patients with a history of cancer, of any origin, at the time of SUDD diagnosis and/or under treatment with chemotherapy and/or radiotherapy; a history of alcohol, drug, or chemical abuse; patients with a current or recent (≤ 3 months) episode of COVID-19 [11]. Furthermore, patients who had received treatment with GM manipulation tools (such as probiotics and antibiotics, including those not absorbed), and/or non-steroidal anti-inflammatory drugs (except for acetyl-salicylic acid ≤ 100 mg/day) < 4 weeks prior to fecal collection, were excluded. This was done in light of the well-known impact of probiotics [12], antibiotics (including the non-absorbable rifaximin, widely used in SUDD) [13], and mesalazine (an anti-inflammatory drug used to relieve SUDD symptoms and prevent diverticulitis) [14] on the GM, which could potentially introduce bias.

The primary endpoint was to compare the GM of SUDD patients who did or did not relapse within a short term (6 months) of SUDD diagnosis.

Microbial DNA was extracted using the repeated bead-beating plus column method, with minor adaptations [7]. Indexed libraries of the V3-V4 hypervariable regions of the 16S rRNA gene were prepared according to the Illumina protocol “16S Metagenomic Sequencing Library Preparation” (Illumina, San Diego, CA, USA). The final pool was obtained by mixing all samples to an equimolar concentration of 4 nM, denaturing and diluting to 5 pM prior to sequencing on an Illumina MiSeq platform using a 2×250 bp paired-end protocol according to the manufacturer’s instructions. Raw sequencing reads are available for download from the National Center for Biotechnology Information Sequence Read Archive (NCBI SRA; BioProject ID: PRJNA1216941).

Bioinformatics and Statistical Analysis

Raw sequences were processed using PANDASeq (15) and QIIME 2 [16]. Reads were filtered for length and quality and then binned into amplicon sequence variants (ASVs) using DADA2 [17]. Taxonomic assignment was performed using the VSEARCH algorithm [18] and the SILVA database (August 2020 release) [19]. Alpha diversity was calculated using several metrics, such as the Shannon index, the number of observed ASVs and Faith’s phylogenetic diversity. Beta diversity was calculated as weighted UniFrac distances and visualized using Principal Coordinates Analysis (PCoA) plots.

Statistical analysis was performed using R software and the “vegan” (<https://cran.r-project.org/package=vegan>) and “Made4” (20) packages. Data separation in PCoA plots was tested using a PERMANOVA test (“Adonis” function in “vegan”). Group differences in alpha diversity and relative taxon abundance were assessed using the Kruskal-Wallis test followed by post-hoc Wilcoxon tests. P-values were adjusted using the Benjamini-Hochberg method. A false discovery rate (FDR) ≤ 0.05 was considered statistically significant, and FDR ≤ 0.1 was considered to indicate a trend.

RESULTS

According to the inclusion and exclusion criteria, 66 patients with SUDD and 30 patients with AD were identified. Of the SUDD patients, 5 had a recurrence of SUDD and 61 had no recurrence within 6 months of SUDD diagnosis. The demographic and clinical characteristics of SUDD and AD patients are reported in Table I. No differences were found between SUDD relapsers and not relapsers, but both groups differed from AD patients in terms of the diet followed (a vegetarian diet was mainly recorded in the AD group) and abdominal pain, which was only recorded in the SUDD groups ($p=0.0059$).

The GM of SUDD patients who did or did not relapse within 6 months of diagnosis did not differ in alpha or beta diversity, either between each other or compared to AD controls ($p>0.05$) (Fig. 1A). However, taxonomically the phylum Verrucomicrobiota, the family *Erysipelotrichaceae* and the genera *Prevotellaceae NK3B31 group*, *Roseburia* and *Holdemanella* showed higher relative abundances in the SUDD-relapsing group than in the non-relapsing group (Wilcoxon test, $p<0.05$) (Fig. 1B-D).

Table I. Demographic and clinical characteristics of SUDD patients who relapsed (rSUDD) or did not relapse (SUDD) within 6 months of diagnosis, and patients with asymptomatic diverticulosis (AD) as controls

	SUDD (n=61)	rSUDD (n=5)	AD (n=30)	p ^a
Male gender, n (%)	35 (57.4)	3 (60.0)	11 (36.7)	0.15
Median (IQR) age, years	63.8 (36-81)	62 (52-70)	62.5 (55-71)	0.457b
Smoking, n (%)	9 (14.7)	1 (20.0)	4 (13.3)	0.793
Presence of comorbidities, n (%)	42 (68.8)	5 (100)	24 (80.0)	0.242
Previous appendectomy, n (%)	8 (13.1)	1 (20.0)	7 (23.3)	0.389
Diagnostic tool, n (%)				
Colonoscopy	48 (78.6)	4 (80.0)	22 (73.3)	0.771c
Computed tomography	12 (19.7)	-	4 (13.3)	
Ultrasonography	5 (8.2)	-	3 (10.0)	
Diet, n (%)				<0.0001 ^c
Mediterranean	33 (54.1)	1 (54.1)	16 (53.3)	0.394
Prevalence of meat	5 (8.2)	-	-	0.36
Prevalence of fish	-	1 (20.0)	-	0.05
Vegetarian	8 (13.1)	1 (20.0)	12 (40.0)	0.015
Vegan	-	-	2 (6.7)	0.197
Abdominal pain, median (IQR) VAS score	5 (3-10)	5 (3-10)	0 (0-3)	0.0059 ^b
Bristol stool form scale, median (IQR)	4 (3-5)	3 (3-5)	4 (3-5)	0.263b

IQR: interquartile range; VAS: visual analogue scale; ^aFisher's exact test; ^bKruskal-Wallis test; ^cChi2 test.

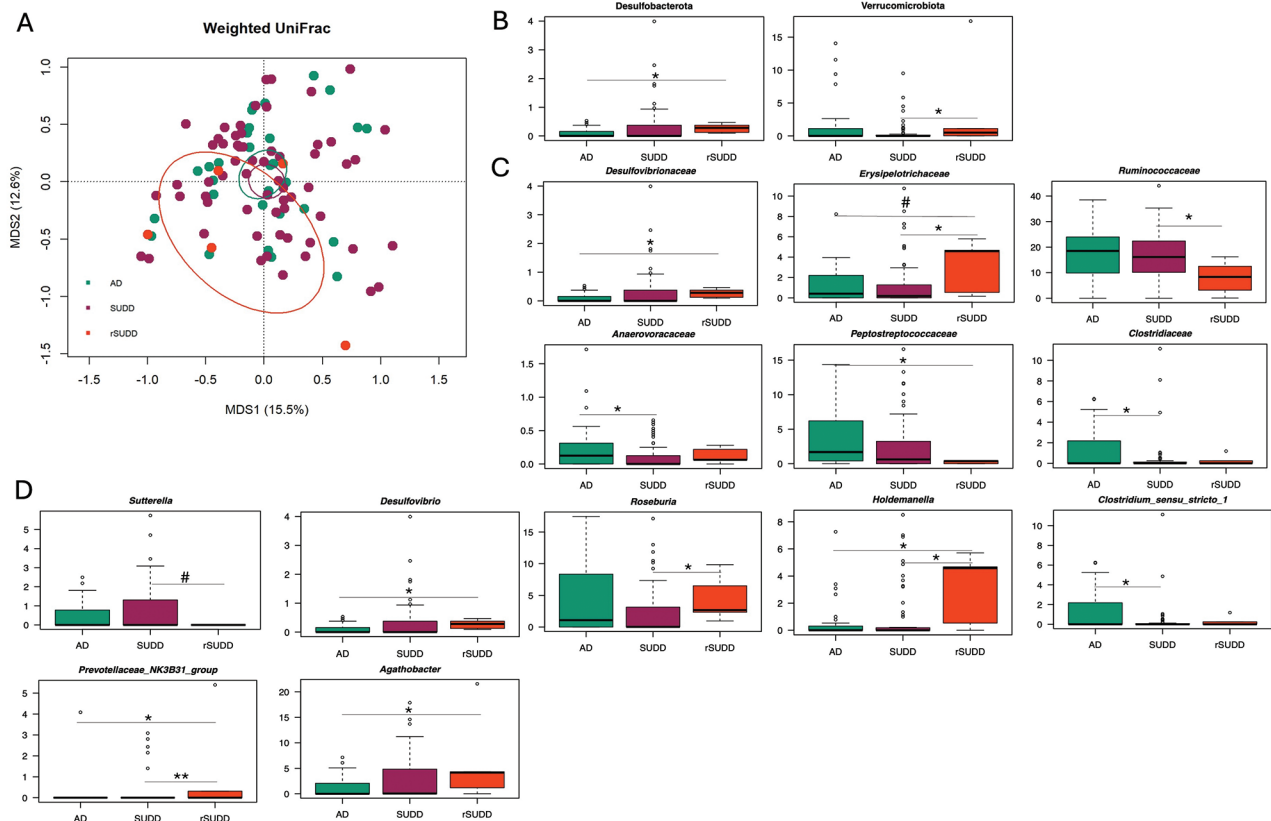


Fig. 1. Gut microbiota profile of SUDD patients who relapsed or did not relapse within 6 months of diagnosis compared to patients with asymptomatic diverticulosis. (A) Principal Coordinates Analysis (PCoA) based on weighted UniFrac distances between gut microbiota profiles of SUDD patients who did (rSUDD) or did not (SUDD) relapse within 6 months of SUDD diagnosis, and patients with asymptomatic diverticulosis (AD). Ellipses include 95% confidence area based on the standard error of the weighted average of sample coordinates. No significant segregation was found (Adonis, $p > 0.05$). Boxplots showing the relative abundance distribution of phyla (B), families (C) and genera (D) differentially represented between study groups. Wilcoxon test; * $p \leq 0.05$, ** $p \leq 0.01$, # $p \leq 0.1$.

Notably, *Erysipelotrichaceae*, *Prevotellaceae* NK3B31 group and *Holdemanella* were also overrepresented compared to AD patients ($p \leq 0.06$). Conversely, *Ruminococcaceae* and *Sutterella* were significantly enriched in non-relapsers compared to relapsers ($p < 0.05$). Further differences were observed between SUDD groups and AD patients. In particular, compared to AD patients, SUDD relapsers were characterized by higher proportions of Desulfobacterota, *Desulfovibrionaceae*, *Desulfovibrio* and *Agathobacter*, and lower proportions of *Peptostreptococcaceae* ($p < 0.05$). SUDD non-relapsers were characterized by comparatively lower proportions of *Clostridiaceae*, *Anaerovoracaceae* and *Clostridium sensu stricto 1* ($p \leq 0.04$).

DISCUSSION

This study investigated differences in GM between SUDD patients who experienced disease recurrence and those who did not, in order to assess the potential role of GM in SUDD recurrence. While no differences were observed in alpha or beta diversity, the GM of SUDD-relapsing patients differed from that of non-relapsing patients in the proportions of many taxa. In particular, SUDD relapsers were enriched in the phylum Verrucomicrobiota (including mucin-degrading bacteria such as *Akkermansia*), the family *Erysipelotrichaceae* and the genera *Holdemanella*, *Prevotellaceae* NK3B31 group, and *Roseburia*. *Erysipelotrichaceae* members, particularly *Holdemanella*, have previously been found to be overrepresented in the GM of SUDD patients and have been associated with IBD severity [21, 22]. *Prevotellaceae* NK3B31 is an opportunistic pathogen associated with stress and inflammation [23], and its family (*Prevotellaceae*) has been found to be enriched in severe SUDD [7]. Notably, the differences in *Erysipelotrichaceae*, *Holdemanella* and *Prevotellaceae* NK3B31 group were also significant compared to AD patients, further strengthening their potential role in SUDD recurrence. Conversely, the overabundance of *Roseburia*, a butyrate-producing genus, in SUDD relapsers is counterintuitive and requires further investigation. Compared to non-relapsers, SUDD relapsers also showed lower proportions of *Ruminococcaceae* and *Sutterella*. The underrepresentation of *Ruminococcaceae*, one of the most abundant families in a healthy adult GM, may further contribute to an unbalanced GM prone to favoring SUDD recurrence. Indeed, a decrease in *Ruminococcaceae* has so far been linked to multiple gastrointestinal disorders [24]. It should be noted that the *Ruminococcaceae* family includes known fiber degraders and short-chain fatty acid producers, suggesting a potential relationship with dietary habits. Although it was not possible to identify differences in diet between SUDD relapsers and non-relapsers in our cohort, fewer patients in both groups followed a vegetarian diet compared to AD patients. On the other hand, *Sutterella* has been reported to be depleted in IBD patients (25), although some species, such as *Sutterella wadsworthensis*, have been implicated in bacteremia in patients with gastrointestinal disorders (26). It is also worth noting that, compared to AD patients, SUDD relapsers were characterized by higher proportions of sulphate-reducing bacteria (Desulfobacterota, *Desulfovibrionaceae* and *Desulfovibrio*), known pathobionts capable of inducing

inflammation, also through impaired barrier function driven by excess production of hydrogen sulphide [27]. Once again, such differences could be related to diet, as sulphate-reducing bacteria are typically associated with western diets, low in fiber while high in protein and fat [28]. Other compositional differences were found between SUDD relapsers or non-relapsers and AD patients, matching previous observations [7].

As expected, this study has some limitations: i) the retrospective design, which limits observation of the GM to a strict timeframe (it cannot be ruled out that acute diverticulitis could occur outside of the 6-month period); ii) the small cohort size, particularly of SUDD relapsers (which also prevented further investigation into the possible relationship between diet, GM profile and SUDD recurrence); iii) the short-term analysis (six months) to assess recurrence, that could limit the collection of potential relapsers though a longer follow-up; iv) the use of 16S rRNA amplicon sequencing, which, while a standard method for microbiota profiling, lacks the resolution for species-level taxonomic classification and functional inference; and v) the absence of mechanistic insights, which would necessitate further investigation, including the use of *in vitro* and animal models.

Despite these limits, this study reveals an association between recurrent SUDD and GM taxa previously been implicated in severe SUDD and IBD. This suggests a more dysbiotic profile than in SUDD patients with no short-term disease recurrence, and hints at a potential predictive role of the GM. Larger and longer cohort studies are warranted to further investigate the presence and potential biomarker role of these taxa in recurrent SUDD. Similarly, these studies will need to investigate the relationship with dietary habits. However, a recent article of ours [29] suggests that diet may have little impact on GM in cases of severe SUDD. It is therefore reasonable to speculate that the same applies in cases with a high probability of recurrence, and that other GM modulation tools should consequently be employed to address dysbiosis and prevent recurrence.

CONCLUSIONS

This is the first time that a difference was found in the GM of SUDD patients with versus without a recurrence of the disease within 6 months. These findings may explain why some SUDD patients experience short-term disease relapse. However, further prospective studies are needed to confirm these data, in order to plan adequate GM-based interventional treatment.

Conflicts of interest: None to declare.

Authors' contribution: A.T. conceived the study and designed the methodology. All authors collected, analyzed and interpreted the data. A.T., G.P., S.T. and F.D.A. drafted the manuscript and revised it critically for important intellectual content. All the authors read and approved the final version of the article.

Data Availability Statement: The raw sequencing data that support the findings of this study were deposited in the National Center for Biotechnology Information Sequence Read Archive (BioProject ID: PRJNA1216941).

Supplementary material: To access the supplementary material visit the online version of the *J Gastrointestin Liver Dis* at <http://dx.doi.org/10.15403/jgld-6535>.

REFERENCES

- Tursi A, Scarpignato C, Strate LL, et al. Colonic diverticular disease. *Nat Rev Dis Primers*. 2020;6:20. doi:10.1038/s41572-020-0143-6
- Tursi A, Elisei W, Franceschi M, Picchio M, Di Mario F, Brandimarte G. The prevalence of symptomatic uncomplicated diverticular disease could be lower than expected: a single-center colonoscopy-based cohort study. *Eur J Gastroenterol Hepatol*. 2021;33(Suppl 1). doi:10.1097/MEG.0000000000002253
- Jono T, Kasai Y, Kessoku T, et al. The prevalence and characteristics of symptomatic uncomplicated diverticular disease among Asian patients with unexplained abdominal symptoms. *J Neurogastroenterol Motil*. 2024;30:87-96. doi:10.5056/jnm23132
- Santacroce G, Lenti MV, Abruzzese GM, et al. Diagnostic delay in symptomatic uncomplicated diverticular disease: an Italian tertiary referral centre study. *Intern Emerg Med*. 2024;19:99-106. doi:10.1007/s11739-023-03241-7
- Tursi A, Franceschi M, Elisei W, Picchio M, Di Mario F, Brandimarte G. The natural history of symptomatic uncomplicated diverticular disease: a long-term follow-up study. *Ann Gastroenterol*. 2021;34:208-213. doi:10.20524/aog.2021.0581
- Tursi A, Papa A. The role of gut microbiota in the pathogenesis of diverticular disease: where are we now? *Genome Med*. 2024;16:153. doi:10.1186/s13073-024-01211-9
- Tursi A, Turrioni S, De Bastiani R, et al. Gut microbiota in symptomatic uncomplicated diverticular disease stratifies by severity of abdominal pain. *Eur J Gastroenterol Hepatol*. 2025;37:147-153. doi:10.1097/MEG.0000000000002656
- Tursi A, Procaccianti G, Turrioni S, et al. Gut microbiota perturbations are linked to endoscopic severity of diverticular disease. *J Gastrointest Liver Dis*. 2025;34:317-322. doi:10.15403/jgld-5215
- Tursi A, Elisei W, Picchio M, Giorgetti GM, Brandimarte G. Moderate to severe and prolonged left lower-abdominal pain is the best symptom characterizing symptomatic uncomplicated diverticular disease of the colon: a comparison with fecal calprotectin in clinical setting. *J Clin Gastroenterol*. 2015;49:218-221. doi:10.1097/MCG.000000000000109
- Tursi A, Brandimarte G, Di Mario F, et al. International consensus on diverticulosis and diverticular disease: statements from the 3rd International Symposium on Diverticular Disease. *J Gastrointest Liver Dis*. 2019;28:57-66. doi:10.15403/jgld.2014.1121.282.tur
- Freedberg DE, Chang L. Gastrointestinal symptoms in COVID-19: the long and the short of it. *Curr Opin Gastroenterol*. 2022;38:555-561. doi:10.1097/MOG.0000000000000871. doi:10.1097/MOG.0000000000000871
- Tursi A, Papa V, Lopetuso LR, Settanni CR, Gasbarrini A, Papa A. Microbiota composition in diverticular disease: implications for therapy. *Int J Mol Sci*. 2022;23:14799. doi:10.3390/ijms232314799. doi:10.3390/ijms232314799
- De Vincentis A, Santonico M, Del Chierico F, et al. Gut microbiota and related electronic multisensorial system changes in subjects with symptomatic uncomplicated diverticular disease undergoing rifaximin therapy. *Front Med (Lausanne)*. 2021;8:655474. doi:10.3389/fmed.2021.655474
- Dai L, Tang Y, Zhou W, et al. Gut microbiota and related metabolites were disturbed in ulcerative colitis and partly restored after mesalamine treatment. *Front Pharmacol*. 2021;11:620724. doi:10.3389/fphar.2020.620724
- Masella AP, Bartram AK, Truszkowski JM, Brown DG, Neufeld JD. PANDAseq: paired-end assembler for illumina sequences. *BMC Bioinformatics*. 2012;13:31. doi:10.1186/1471-2105-13-31
- Bolyen E, Rideout JR, Dillon MR, et al. Reproducible, interactive, scalable and extensible microbiome data science using QIIME 2. *Nat Biotechnol*. 2019;37:852-857. doi:10.1038/s41587-019-0209-9
- Callahan BJ, McMurdie PJ, Rosen MJ, Han AW, Johnson AJ, Holmes SP. DADA2: high-resolution sample inference from Illumina amplicon data. *Nat Methods*. 2016;13:581-583. doi:10.1038/nmeth.3869
- Rognes T, Flouri T, Nichols B, Quince C, Mahé F. VSEARCH: a versatile open source tool for metagenomics. *PeerJ*. 2016;4. doi:10.7717/peerj.2584
- Quast C, Pruesse E, Yilmaz P, et al. The SILVA ribosomal RNA gene database project: improved data processing and web-based tools. *Nucleic Acids Res*. 2013;41(Database issue). doi:10.1093/nar/gks1219
- Culhane AC, Thioulouse J, Perrière G, Higgins DG. MADE4: an R package for multivariate analysis of gene expression data. *Bioinformatics*. 2005;21:2789-2790. doi:10.1093/bioinformatics/bti394
- Hou K, Wu ZX, Chen XY, et al. Microbiota in health and diseases. *Signal Transduct Target Ther*. 2022;7:135. doi:10.1038/s41392-022-00998-y
- Vestergaard MV, Allin KH, Eriksen C, et al. Gut microbiota signatures in inflammatory bowel disease. *United European Gastroenterol J*. 2024;12:22-33. doi:10.1002/ueg2.12410
- Liu Y, Chen H, Feng L, Zhang J. Interactions between gut microbiota and metabolites modulate cytokine network imbalances in women with unexplained miscarriage. *NPJ Biofilms Microbiomes*. 2021;7:24. doi:10.1038/s41522-021-00216-4
- Biddle AS, Stewart LC, Blanchard JL, Leschine S. Untangling the genetic basis of fibrolytic specialization by Lachnospiraceae and Ruminococcaceae in diverse gut communities. *Diversity*. 2021;5:627-640. doi:10.3390/d13120627
- Su Q, Tun HM, Liu Q, et al. Gut microbiome signatures reflect different subtypes of irritable bowel syndrome. *Gut Microbes*. 2023;15:2157697. doi:10.1080/19490976.2022.2157697
- Kirk KF, Andersen KL, Tarpgaard IH, Nielsen HL. Three cases of *Sutterella wadsworthensis* bacteremia secondary to abdominal infections. *Anaerobe*. 2021;72:102460. doi:10.1016/j.anaerobe.2021.102460
- Singh SS, Carrol-Portillo A, Lin HC. *Desulfovibrio* in the gut: the enemy within? *Microorganisms*. 2023;11:1772. doi:10.3390/microorganisms11101772
- David LA, Maurice CF, Carmody RN, et al. Diet rapidly and reproducibly alters the human gut microbiome. *Nature*. 2014;505:559-563. doi:10.1038/nature12820
- Tursi A, Procaccianti G, D'Amico F, et al. Impact of diet on gut microbiota in diverticular disease of the colon: an exploratory retrospective study. *Microorganisms*. 2025;13:2428. doi:10.3390/microorganisms13112428.