MICROBIOTA Mag

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| OVERVIEW |

Functional dyspepsia and the gut microbiota

BIOCODEX Microbiota Institute

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SCIENCEALERT - How Long Poop Stays in Your Body Could Be Quietly Shaping Your Gut Microbiome – And Disease Risk

In this article, ScienceAlert explores how gut transit time may influence the composition and activity of the gut microbiota, and how these changes could potentially be linked to disease risk.

8M reach/100 engagements



CALORIES.HUB - Sourdough feed your gut microbiome

Sourdough bread is presented as a healthier alternative to conventional bread, with claims that its fermentation process may help regulate blood sugar, improve mineral absorption, and support the gut microbiota through prebiotic compounds.

1,5M reach/47k engagements



BIG THINK CLIPS - Microbiome expert: How to reset your gut overnight | Tim Spector

Epidemiologist Tim Spector shares 8 practical gut-health shifts, from eating 30 plants a week to embracing fermented foods and reconsidering breakfast.

510,149 views/13k likes

EDITO



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Dear readers,

Have you ever stopped to consider these questions: how many patients with persistent upper gastrointestinal symptoms do you see each week? And how many of them remain undiagnosed with functional dyspepsia (FD)?

Functional dyspepsia affects around 10% of adults worldwide, yet it can still be challenging to diagnose. Postprandial fullness, early satiety, epigastric pain or burning… its symptoms can overlap with those of gastroesophageal reflux disease, gastroparesis, or irritable bowel syndrome. This clinical overlap can, quite simply, make the diagnosis difficult to pin down.

But functional dyspepsia also illustrates how our understanding of gastrointestinal disorders is evolving. Now recognized as a disorder of gut–brain interaction, it can no longer be reduced to a single dysfunction of the stomach. Altered motility, visceral hypersensitivity, impaired duodenal barrier and immune function, psychosocial factors—and, increasingly, the microbiota: the mechanisms involved reveal a complex condition at the crossroads of the gastrointestinal tract, the immune system, and the brain.

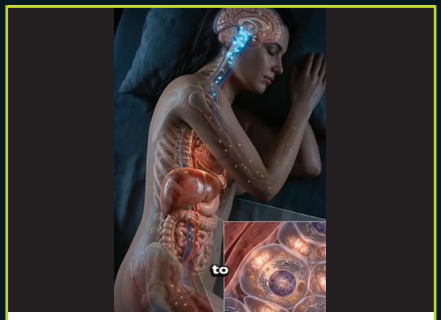
In this issue's review, Professor Lucas Wauters helps us unravel this complexity and better understand how emerging evidence on the duodenum and its microbiota is reshaping our understanding of the pathophysiology of functional dyspepsia and opening up new therapeutic perspectives. He also examines the recent changes introduced by the Rome V criteria, particularly in the definition and classification of different patient profiles.

Because changes in diagnostic criteria can sometimes represent far more than a shift in terminology: they can reflect a new way of recognizing symptoms, characterizing patients more accurately and, potentially, guiding their management more effectively.

That is precisely the purpose of *Microbiota Mag*: to identify, amid a constantly evolving scientific landscape, the advances that challenge established thinking, sharpen diagnosis and, ultimately, improve the care of your patients..

Enjoy your reading!

Functional dyspepsia affects around 10% of adults worldwide, yet it can still be challenging to diagnose.



BODY PROTOCOLS - What Happens If You Stop Eating Sugar for 30 Days?

What really happens inside your body? Explore the surprising effects on your brain, gut microbiome, skin, insulin, inflammation and metabolism.

971,426 views



MEDSCAPE - Coffee and the *Gut*: What's Brewing in the *Microbiome*?

Emerging evidence suggests coffee influences gut microbiota through bioactive compounds beyond caffeine, with potential effects on digestive and metabolic health.

919k impressions



NEUROLOGIA E PSIQUIATRIA - Cansaço extremo e tristeza profunda?

The video focuses on the connection between gut health, the microbiome, and mental health—specifically with depression and chronic fatigue.

7k engagements/115k views



By Prof. Lucas Wauters

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Functional dyspepsia and the gut microbiota

Functional dyspepsia (FD) refers to chronic epigastric symptoms, with postprandial distress syndrome and epigastric pain syndrome as the major subgroups. It is part of the disorders of gut-brain interaction, with visceral signaling (gut-brain) via duodenal hyperpermeability and immune activation, potentially triggered by an altered microbiome composition or dysbiosis. Interestingly, first-line therapy with proton pump inhibitors (PPI) may improve both symptoms and duodenal alterations in the short term but adversely influence the duodenal environment in the long-term. In addition, other treatments, including prokinetics and neuromodulators, have limited efficacy or raise safety issues. Therefore, alternative treatments have been studied, including the spore-forming *Bacillus coagulans* MY01 and *Bacillus subtilis* MY02. Besides the clear efficacy and safety in a randomized, double-blind, placebo-controlled trial, beneficial immune and microbial changes were found and more pronounced in FD patients on PPIs, including a reduction in small intestinal bacterial overgrowth. These and other studies have provided insights into potential underlying mechanisms of FD, including the role of gut microbiota.



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Definition and diagnosis

Dyspepsia refers to chronic upper abdominal symptoms originating from the gastroduodenal region with a significant impact on patients' quality of life. According to the Rome criteria, functional dyspepsia (FD) is characterized by bothersome epigastric symptoms that remain unexplained after routine investigations [1-3]. Disorders of gut-brain interaction (DGBI), including FD, are related to any combination of dysmotility, visceral hypersensitivity, altered luminal or central factors. In contrast, organic dyspepsia may result from erosive esophagitis, peptic ulcer disease or cancer in a minority of patients [1-3].

Functional dyspepsia is characterized by bothersome epigastric symptoms that remain unexplained after routine investigations.

Two subgroups of FD were proposed by the Rome III consensus and reiterated in the Rome IV version:

- postprandial distress syndrome (PDS) with postprandial fullness or early satiation,
- epigastric pain syndrome (EPS) with epigastric pain or burning [1].

The recent Rome V version specifies that symptoms must be severe enough to affect usual activities with a minimal fre-

TABLE 1. ROME V CRITERIA FOR FUNCTIONAL DYSPEPSIA.

	• Must include	• Associated symptoms	• Comments
FD	Bothersome postprandial fullness, early satiation, epigastric pain and/or burning	Heartburn is not a gastroduodenal symptom but often coexists	Predominant nausea and/or persisting vomiting excludes the diagnosis
PDS	Bothersome postprandial fullness and/or early satiation at least 2 days per week	Postprandial epigastric pain or burning, nausea, upper abdominal bloating or excessive belching	Postprandial: symptoms are triggered or worsened within 2 hours of meal intake
EPS	Bothersome epigastric pain and/or epigastric burning at least 1 day per week	EPS can be induced or worsened postprandially, or occur independently from meal ingestion	Meal-related EPS: EPS starts or gets worse after meals in ≥ 50% of episodes. Meal-unrelated EPS: EPS starts or gets worse after meals in < 50% of episodes

Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
EPS, epigastric pain syndrome; FD, functional dyspepsia; PDS, postprandial distress syndrome.

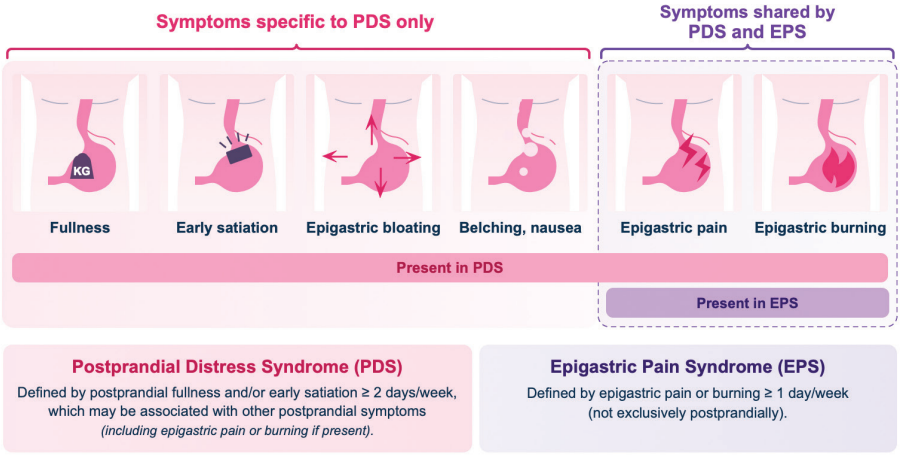
quency of 1 (EPS) or 2 (PDS) days per week [2], which is lower than the previous Rome IV criteria for PDS (3 days per week) (**table 1**).

As PDS is characterized by meal-related symptoms (occurring < 2 hours after meal ingestion), postprandial epigastric pain in the presence of PDS symptoms should also be characterized as PDS and not EPS according to Rome V [2]. In the absence of PDS, epigastric pain symptoms could be further subdivided into meal-related or meal-unrelated EPS, which requires further studies (**figure 1**). Nausea (especially postprandially) and vomiting can coexist in PDS and EPS, but predominant nausea or recurrent vomiting is more common in chronic nausea and vomiting syndrome or gastroparesis [2].

Using the Rome IV criteria, PDS was consistently more prevalent (61% *vs.* 18% for EPS and 21% for overlap) with a similar distribution across the USA, UK and Canada [4]. In addition, FD often coexisted with gastroesophageal reflux (GER) and/or irritable bowel syndrome (IBS). Of note, bowel disorders should be considered when epigastric symptoms are consistently relieved by bowel movements [1-3].

While the exclusion of structural, systemic, or metabolic diseases is necessary for confirmation of FD, the yield of routine endoscopy is low in (young) patients with dyspeptic symptoms and no alarm or risk factors (see below). The prevalence of dyspepsia was higher in women, smokers, users of nonsteroidal anti-inflammatory drugs (NSAIDs) and after *Helicobacter pylori* (*H. pylori*) infection [1-3]. Thus, a non-invasive "test & treat" strategy (*i.e.*, urea breath test or *H. pylori* fecal antigen detection) is common, with endoscopy for alarm factors (*e.g.*, age > 55 years old, or lower in certain regions of the world, evidence of gastrointestinal bleeding or iron-deficiency anemia, dysphagia, unintentional weight loss and family history of gastric or esophageal cancer) [1-3]. Nevertheless, the Rome criteria for FD do not exclude the presence of microscopic pathology in the gastroduodenal region or the stomach and proximal small intestine.

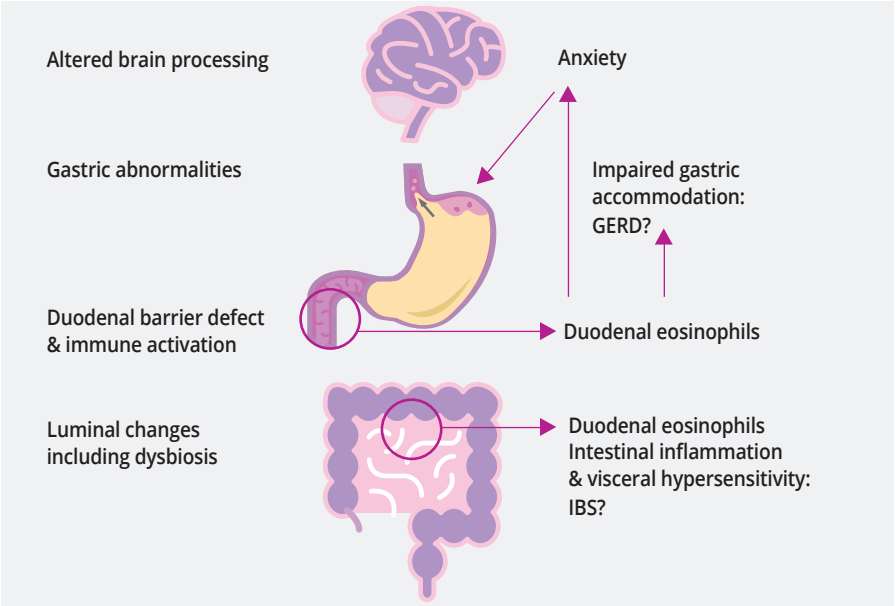
FIGURE • 1 **Cardinal and associated symptoms in functional dyspepsia, including the main subgroups.**



Duodenum and dysbiosis

Abnormalities of gastric function in FD include impaired accommodation or expansion of the stomach, hypersensitivity to distention and delayed emptying, but these changes correlate poorly or not at all with symptoms [1-2]. Both visceral sensory signaling (gut to brain) and central modulation (brain to gut) can be abnormal in FD, as up to 50% of subjects identify stress as a trigger for their symptoms, and anxiety and depression are common. Recent research focused on mucosal and luminal alterations in the proximal small intestine or duodenum [5]. Interestingly, duodenal mucosal eosinophilia was linked to impaired gastric accommodation, reflux symptoms and anxiety, which may influence gastric function [5]. Overlapping IBS symptoms in FD may also be explained by more extensive intestinal inflammation with visceral hypersensitivity (**figure 2**).

FIGURE • 2 **Gut-brain axis and mechanisms of overlap in functional dyspepsia.**



GERD: gastro-esophageal reflux disease; IBS: irritable bowel syndrome. *Source: smart.servier.com.*

Recent research focused on mucosal and luminal alterations in the proximal small intestine or duodenum.

Duodenal mucosal changes in FD patients include a barrier defect and immune activation, with increased numbers and degranulation of eosinophils and mast cells. Even if a mucosal T-helper (Th) 2-signal was not commonly found, increased interleukin (IL)5/13-production after stimulation of peripheral blood mononuclear cells (PBMC) from FD patients suggested a shift from Th1- to Th2-type inflammation [5]. In addition, increased IL1β-production of cultured PBMC pointed to a Th17-response, and production of GM-CSF from Th17 cells may also drive mucosal eosinophil recruitment in FD (see below). Moreover, CD4+ T cells co-expressing integrin α4β7 and CCR9, indicative of small intestinal mucosal migration, were upregulated in FD and correlated with gastric emptying. This also suggests a primary role for duodenal mucosal changes, but the luminal or other triggers are unknown [5].

> Duodenal luminal changes include an altered microbial composition or "dysbiosis".

We found a lower abundance of *Neisseria* and *Porphyromonas* and microbial diversity in duodenal brush samples of FD patients compared to healthy controls, which inversely correlated with symptoms and duodenal eosinophils [6]. In contrast, microbial changes were associated with the effects of proton pump inhibitors (PPI) on duodenal bile salt concentrations in healthy controls, which persisted after long-term PPI-therapy in FD patients [6, 7]. The presence of intestinal-like bacteria in the gastric fluid of FD patients also suggested small intestinal bacterial overgrowth (SIBO) in a subset, even if a standardized diagnosis of SIBO is not available yet and the majority of studies have used non-invasive breath tests in FD with different substrates (glucose or lactulose), doses or type of exhaled gases (hydrogen and/or methane) [8].

We and others have hypothesized that the duodenal barrier defect may lead to mucosal immune activation through antigen presentation with a predominant Th2- or Th17-response and recruitment and degranulation of eosinophils, triggering visceral hypersensitivity and dysmotility. Possible causes include the duodenal microbiome, which may also be a therapeutic target [5, 8].

Current and novel therapies

Despite the recognition of meal-related symptoms, no specific dietary factors have been convincingly linked with the pathophysiology of FD. In the absence of controlled dietary intervention studies, standard dietetic advice includes frequent small size meals with avoidance of high-fat food items, alcohol or coffee and smoking cessation [1-3]. Acid-suppressive therapy with PPI is recommended as first-line therapy in *H. pylori*-negative patients or after eradication if *H. pylori*-positive [3]. Indeed, PPIs are more effective than placebo in the reduction of global symptoms of FD with a number needed to treat (NNT) of 11 in a Cochrane meta-analysis [9]. This includes a standard once-daily dose of PPI during 4 to 8 weeks, with discontinuation of therapy in case of insufficient improvement. While PPIs are beneficial for overlapping reflux symptoms, we have also shown beneficial effects of short-term PPI-therapy on duodenal permeability and immune activation in FD patients [7].

A significant reduction in dyspeptic symptoms was also found with prokinetics, with a NNT of 12 when cisapride (withdrawn from the market due to cardiac adverse events) was excluded in a systematic review and meta-analysis [10]. However, QT-prolongation (domperidone) or extrapyramidal side effects (metoclopramide) limit their chronic use and other prokinetics such as itopride or acotiamide are not widely available. In addition, amitriptyline (tricyclic antidepressant) but not escitalopram (selective serotonin-reuptake inhibitor) was effective for ulcer-like FD, with threefold higher odds of reporting adequate relief of symptoms compared to placebo [11]. Another systematic review and network meta-analysis confirmed the superior efficacy of antidepressants or neuromodulators for symptom improvement, and that symptom resolution was most common with standard-dose PPIs [12].

However, inappropriate and prolonged use of PPIs is frequently reported, and may increase the risk of dysbiosis. Probiotics are live microorganisms that exert a health benefit on the host, and spore-forming probiotic strains may outperform traditional supplements because of gastric-acid resistant endospores with improved survival in the small intestine. We showed that *Bacillus coagulans* MY01 and *subtilis* MY02 spore-forming probiotics (daily dose of $5 \cdot 10^9$ colony-forming units or CFU)

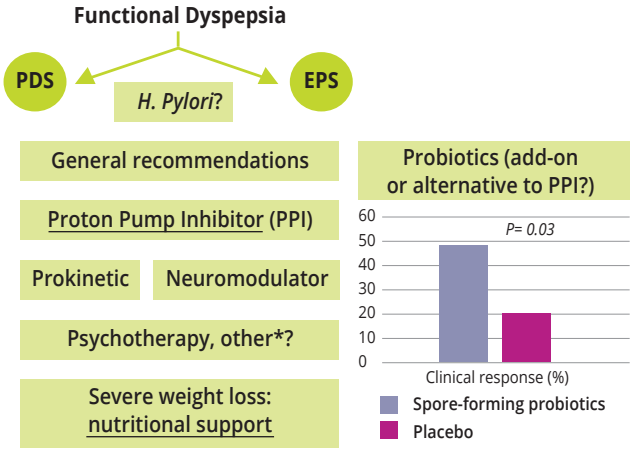
were effective and safe in FD patients as mono- or add-on therapy to long-term intake of PPI [13]. Importantly, PDS (primary outcome), EPS and individual symptoms significantly decreased with spore-forming probiotics compared to placebo. We extended the clinical efficacy during an open-label phase (total 16 weeks), with symptom-improvement in the original placebo-group.

Inappropriate and prolonged use of PPIs is frequently reported and may increase the risk of dysbiosis.

Interestingly, longer-term probiotic treatment decreased systemic Th17-signaling, including Th17 cells and IL-17A, which were associated with clinical efficacy. Additional decreases were found in Th2, gut-homing and GM-CSF+ T cells in FD patients on-PPI, suggesting that the probiotic effects may be more pronounced in case of PPI-related dysbiosis. Moreover, a reduction in SIBO was found in FD patients on-PPI after 8 weeks. Finally, despite the absence of major shifts in relative or quantitative fecal microbiota community composition, a proportional increase in the beneficial *Faecalibacterium* was also associated with probiotic efficacy. Another RCT found a higher clinical efficacy for the probiotic *Bifidobacterium animalis* subsp. *lactis* BL-99 in a high ($5{\cdot}10^{10}$ CFU/day) compared to low dose ($1{\cdot}10^{10}$ CFU/day) or positive-drug control (rabeprazole) and placebo after 8 weeks [14]. Interestingly, this effect was sustained 2 weeks after stopping therapy, including the persistence of the probiotic strains in stool samples. Moreover, an increase in short-chain fatty acid (SCFA)-producing microbiota such as *Faecalibacterium* and *Roseburia* was also found with the probiotic, with increased levels of SCFA in stool and blood samples, pointing to underlying microbial mechanisms.

While the use of probiotics was not yet included in the European consensus on FD [3], agreement or consensus support was only reached for PPI and nutritional support in case of severe weight loss (**figure 3**). Based on the novel evidence, probiotics (particularly spore-forming) could be considered as add-on or alternative to (long-term) PPI-therapy.

FIGURE • 3 **Proposed treatment algorithm for functional dyspepsia, including probiotics.**



Consensus support is underlined. *Other: herbal, acupuncture and alternative medicine.
EPS: epigastric pain syndrome; PDS: postprandial distress syndrome; PPI: proton-pump inhibitor.

Probiotics could be considered as add-on or alternative to PPI-therapy.


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Conclusion

Emerging data increasingly point towards the duodenum as a key integrator in FD, including the microbiome of the small intestine [15]. While first-line or PPI-therapy may be effective in the short term, long-term therapy is frequently reported with an increased risk of dysbiosis. Alternative therapies include probiotics, with not only beneficial clinical but also immunological and microbial effects of spore-forming probiotics. Although these could be considered as monotherapy, changes in immune activation were more pronounced in FD patients on-PPI, suggesting additional effects on the observed chronic alterations with PPI-therapy, which is especially useful in case PPI-withdrawal is not feasible.

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COMMENTED ARTICLE

Adults' section



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By Prof. Harry Sokol

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Gut micro-organisms associated with health, nutrition and dietary interventions

Comments on the article by Asnicar et al. Nature 2026 [1]

The incidence of cardiometabolic diseases is increasing globally, with both poor diet and the human gut microbiome implicated. However, large-scale comprehensive studies exploring these links in diverse populations are lacking in this area. Using data from over 34,000 participants in the US and the UK, this study highlights significant associations between diet, the gut microbiome and cardiometabolic health markers. The authors have established "ZOE Microbiome Health Ranking 2025" to rank the species most and least favourably associated with human health markers, validated in over 7,800 independent samples. In dietary interventional clinical trials (746 participants), favourable species increased while unfavourable species decreased. These findings strengthen the link between diet, the microbiome and health, whilst emphasising that prospective and interventional studies are still needed to establish a causal link.

What do we already know about this subject?

••• Cardiometabolic diseases, particularly cardiovascular disease and type 2 diabetes, are the leading cause of morbidity and mortality in Western countries [2]. Whilst diet is the main modifiable risk factor, the gut microbiome is now recognised as playing a key role in metabolism, due to its influence on diet and involvement in obesity, as well as insulin resistance and inflammation. However, studies available up to now have been based mainly on small and lowly diversified cohorts, with limited microbiological and phenotypic data [3–5], which has hindered the identification of robust and universal microbial biomarkers and definition of a "healthy microbiome" applicable at a population level.

What are the main insights from this study?

••• To answer these questions, the authors pooled and harmonised five metagenomic cohorts (United States and United Kingdom), representing a total of 34,694 participants with high-depth shotgun metagenomic sequencing, validated diet questionnaires, and a complete panel of cardiometabolic markers. Using machine-learning models trained on the relative abundance of 661 microbial species (defined as species-level genome bins, or SGBs), the authors showed that the microbiome reproducibly, albeit moderately, predicts numerous cardiometabolic health markers [AUC 0.64–0.73 for classification; Spearman's correlation coefficient 0.30–0.46 for regression analysis (**figure 1**)].

These findings led to the creation of "ZOE Microbiome Health Ranking 2025", a ranking system for 661 species based on their clear link to host health markers (personal health, fasting markers, postprandial markers) and diet quality. The highest-ranked species (close to 0), which are associated with a favourable metabolic profile, belong mainly to the phylum Firmicutes and *Clostridia* and *Ruminococcaceae* classes, but most are uncharacterised. Of the top 50 species, 22 were unknown organisms represented solely by genomes reconstructed from metagenomic data. Among the known species, *Faecalibacterium prausnitzii* and *Eubacterium siraeum* are classified in the most favourable category, a finding consistent with the literature. Conversely, the lowest-ranked species (close to 1), which are associated with an unfavourable profile, include organisms already known for their harmful effects such as *Ruminococcus gnavus, Ruminococcus torques, Flavonifractor plautii, Enterocloster bolteae* and *Eggerthella lenta*.

The authors then validated this classification using over 7,800 independent public samples. On average, obese individuals had 1.95 more unfavourable species than normal-weight individuals, and 5.2 fewer favourable species. Finally, in two clinical trials on dietary intervention (n = 746), favourably ranked species increased in abundance and prevalence after the intervention, whilst unfavourable species decreased, suggesting that the microbiome can be modified by diet, even though causality cannot be inferred.

FIGURE 1 The 15 top and bottom health-ranked SGBs show consistent associations across the five PREDICT cohorts.

Average percentiles for the 15 most favourably (top) and unfavourably (bottom) ranked SGBs across all five PREDICT cohorts. Percentiles are computed from the ranking of the correlations between SGBs and the different markers in each clinical data category. Percentiles close to 0 reflect SGBs consistently correlated positively with positive markers and negatively with negative markers, and conversely for percentiles close to 1. For each cohort, the average percentiles for three clinical data categories are shown (personal, fasting and postprandial) and the cohort-level average. The rightmost column of the *heatmap* reports the *ZOE MB health-ranks* with the distribution of their relative abundance values when present (derived from n = 34,694 participants spanning the five PREDICT cohorts).

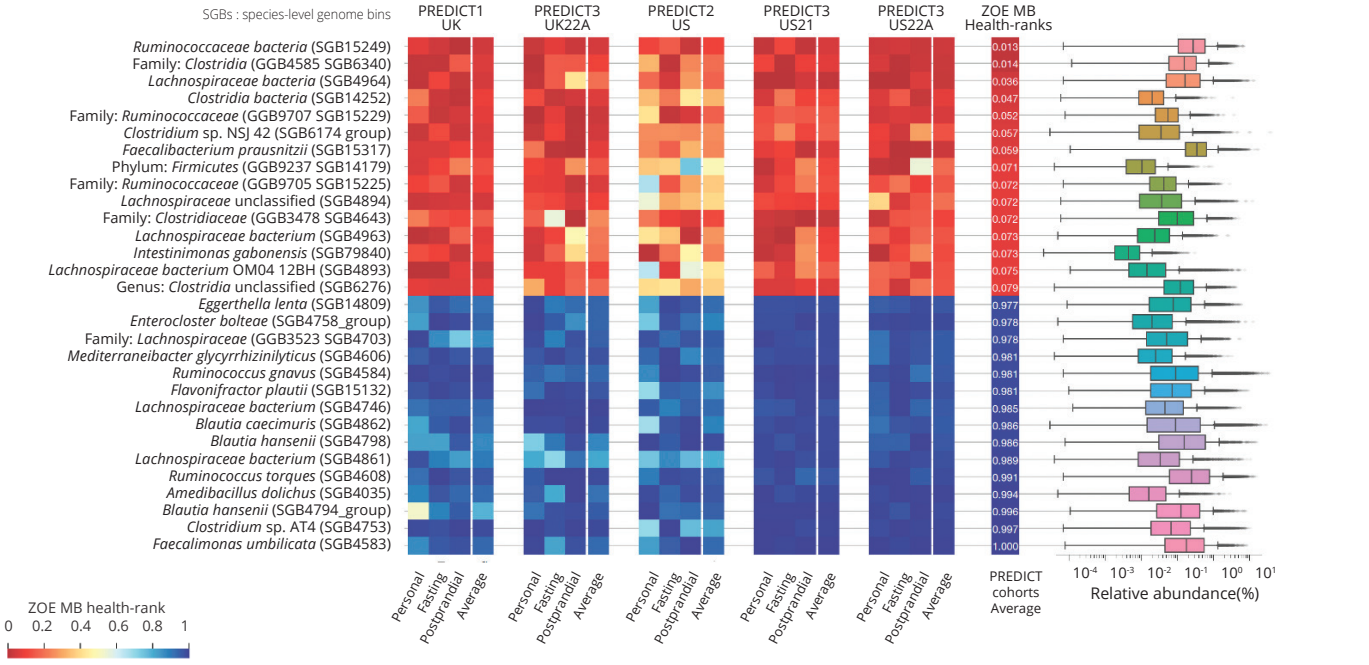


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What are the consequences in practice?

••• This study confirmed, on a large scale, that the gut microbiome is a measurable and modifiable factor in cardiometabolic health, and that "ZOE MB Health Ranking 2025" is a stratification tool that could potentially be used to guide personalised dietary recommendations. It could also be used as an intermediate endpoint in future trials. The immediate clinical implications remain limited given that the associations are correlational, no direct mechanism has been demonstrated, and causality will need to be established in randomised controlled trials. The researchers believe that 22 of the top 50 species remain totally uncharacterised, therefore, cultivating and identifying their metabolites are a priority. The study also calls for the systematic use of shotgun metagenomics in large-scale epidemiological studies.

Key points

- This is the largest metagenomic study combining dietary and clinical data
- Most taxa associated with good health are yet to be cultivated and lack a phenotypic description, highlighting a major blind spot in microbiome research
- Favourable species increase and unfavourable species decrease with improved diet, providing proof of concept of their modifiable nature, but not of causality

[CONCLUSION]

This study is a significant methodological advance in the field of nutritional metagenomics, providing a microbial ranking system that is reproducible on a large scale, across different populations. It confirms the relevance of the microbiome as a marker associated with cardiometabolic health and strengthens the assumption that diet can be used to modulate the microbiome. However, it does not conclude that the microbiome plays a causal role in cardiometabolic disease, and its direct therapeutic implications are speculative in the absence of dedicated interventional trials. The main value of this research is perhaps the provision of the mapping and assumptions required to design such trials in an informed manner.

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By Prof. Emmanuel Mas

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Antenatal depressive symptoms impair offspring neurodevelopment by inducing maternal gut microbiota dysbiosis during pregnancy

Comments on the original article by Zhou et al. [1]

In a cohort of more than 2,000 pregnant women, the authors of this article found that depressive symptoms occurring during any trimester of pregnancy were associated with delayed neurocognitive development in infants. Analysis of the maternal gut microbiota revealed a reduction in butyrate-producing bacteria and disruptions in amino acid metabolism among women experiencing antenatal depression. These alterations were correlated with poorer cognitive outcomes in their children. To investigate causality, the microbiota from women with depressive symptoms was transplanted into germ-free pregnant mice. This transfer led to impaired intestinal barrier function, increased foetal brain inflammation, and metabolic disturbances in the developing brain, ultimately resulting in cognitive deficits in the offspring. Maternal supplementation with butyrate partially reversed these abnormalities. Overall, the findings suggest that antenatal depression may influence foetal brain development through changes in the maternal gut microbiota and its metabolites, highlighting potential preventive and therapeutic targets.

What do we already know about this subject?

••• Antenatal depression (AND) prevalence is around 20% worldwide and is associated with increased risk of miscarriage, preterm birth and low birth weight. However, the link between AND and neurodevelopmental issues has not been clearly established.

Studies show that depression can alter the composition of the gut microbiota (GM). According to some studies, maternal mood disorders during pregnancy can also disrupt both their own GM and that of their offspring.

Disruption to maternal GM during pregnancy could therefore alter brain development.

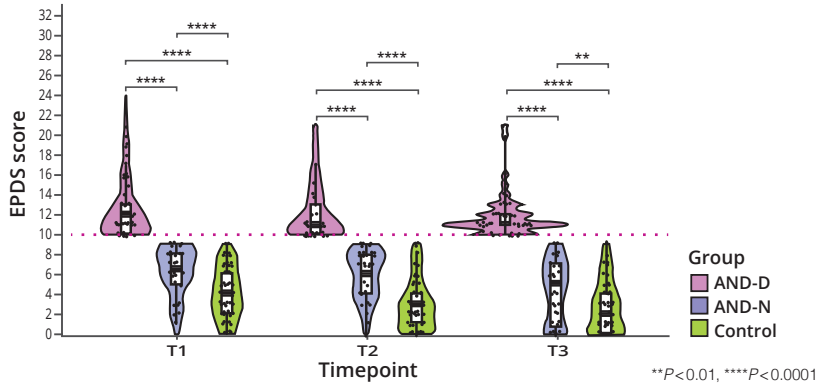
What are the main insights from this study?

••• This was a prospective study of 2,053 women who gave birth between July 2019 and July 2022. Participants were recruited during the preconception period or the first trimester of pregnancy. Depression was assessed using a questionnaire (Edinburgh Postnatal Depression Scale – EPDS). Three hundred and twenty-six women (15.9%) had EPDS ≥10 (AND group). A nested case-control study was conducted to compare 135 women at risk of AND with 135 women matching on age, pre-pregnancy BMI, education level, parity, delivery mode, gestational age, Group B Streptococcus (GBS) infection, and use of corticosteroids, antibiotics, and probiotics.

Neurodevelopment was assessed up to the age of one year. Infants in the AND group scored lower for problem-solving at the age of 3 and 6 months ($p = 0.039$ and 0.040), and communication ($p = 0.039$) and motor skills ($p = 0.013$) at 12 months.

A GM analysis was conducted on 504 maternal faecal samples, divided into three groups based on the EPDS score at the time of the sample collection: AND-D (EPDS ≥10), AND-N (EPDS <10 but considered as at-risk of AND due to a high score at another stage of pregnancy) and controls (EPDS <10 in each trimester of pregnancy) (**figure 1**). The factors influencing the GM were, firstly, the trimester of pregnancy and, secondly, AND, with significant differences between the AND-D group and the control group, and between the AND-N group and the control group. Compared with the control groups, the abundance of *Eubacterium, Ruminococcae, Faecalibacterium* and *Oscillospira-*

FIGURE 1 **Antenatal depression scores among at-risk antenatal depression and control groups during pregnancy.**



**P<0.01, ****P<0.0001

ceae, which produce butyrate, was reduced, while *Streptococcus* abundance was increased in the AND-D and AND-N groups. Two hundred and ninety faecal samples were collected. A 16S RNA analysis of GM showed that AND had no effect. However, a correlation existed between maternal GM and infant neurodevelopment.

To assess the effect of maternal GM, prior to mating the authors transferred GM taken from AND at-risk mothers (AND-M group) and controls (Control-M group) to germ-free mice. The GM composition of the mice was similar prior to mating while at E18.5 (late foetal stage) it was similar to that of the respective donors. AND-M mice exhibited impaired performance in neurodevelopmental tests (escape time, platform crossing) (**figure 2**). A foetal brain metabolomic analysis showed reduced activity of polyunsaturated fatty-acid (PUFAs) pathways and amino-acid pathways in AND-M mice. An increase in inflammatory cytokines (TNF-α and IL-1β) was also observed in the AND-M group. Finally, mothers in the AND-M group had increased intestinal permeability, as assessed by immunohistochemistry, which revealed lower expression of tight junction proteins (claudin-1, ZO-1, and ZO-2) (**figure 3**), as well as higher inflammatory cytokine levels in serum (TNF-α and IL-1β).

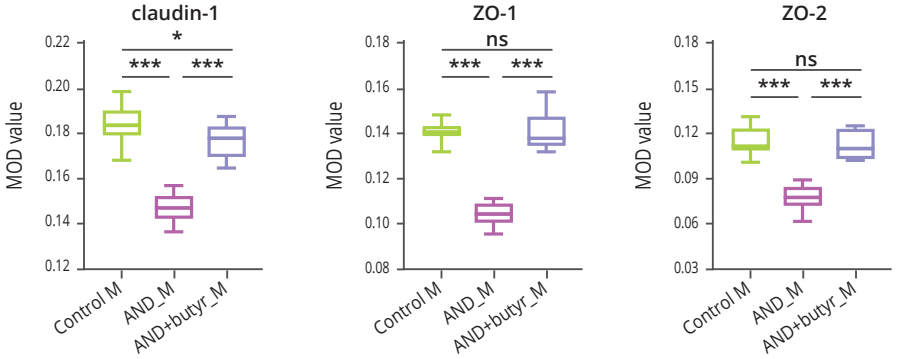
Butyrate supplementation given to mice during pregnancy not only improved the neurodevelopment of their offspring but also increased PUFA and amino-acid levels, improved maternal gut barrier function and reduced the levels of inflammatory cytokines in foetal brains and maternal serum.

What are the consequences in practice?

••• The effects of antenatal depression on the developing foetal brain may be mitigated through butyrate supplementation.

FIGURE 3

Measurement of tight junction protein expression (claudin-1, ZO-1, ZO-2) between colonocytes, reflecting gut permeability in germ-free mice having received faecal matter from control mothers (Control_M), AND mothers (AND_M) and AND mothers treated with butyrate during pregnancy (AND+butyr_M). Permeability was impaired in the AND_M group and improved with butyrate supplementation during pregnancy.



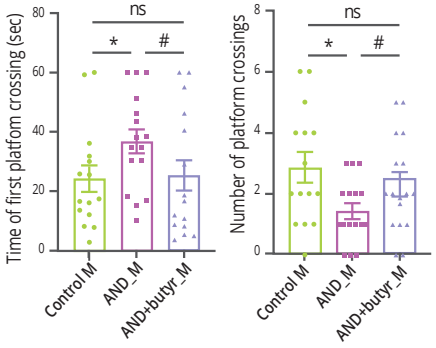
*P<0.05, ***P<0.001, ns: not significant

Key points

- Antenatal depression is associated with altered infant neurodevelopment
- Maternal gut microbiota could play a role in this association
- In mice, butyrate mitigated the anomalies observed and improved neurodevelopment

FIGURE 2

Assessment of neurodevelopment in the offspring of germ-free mice having received faecal matter from control mothers (Control_M), AND mothers (AND_M) and AND mothers treated with butyrate during pregnancy (AND+butyr_M). Tests assessed mobility on a platform and showed that mobility was impaired in the AND_M group and improved after receiving butyrate supplementation during pregnancy.



#P<0.1, *P<0.05, ns : not significant.

[CONCLUSION]

Antenatal depression impairs neurodevelopment in children, linked to changes in the maternal GM. Experiments conducted in mice showed that maternal gut dysbiosis increases intestinal permeability, which in turn is associated with foetal brain inflammation and lower activity of metabolic pathways (PUFAs and amino acids). Butyrate supplementation during pregnancy mitigated these effects.

Source

• **1**. Zhou F, Wang L, Zhao Y, *et al.* Antenatal depressive symptoms impair offspring neurodevelopment by inducing maternal gut microbiota dysbiosis during pregnancy. *Gut Microbes* 2026 ; 18 : 2672188.



58th ANNUAL MEETING



By Prof. Erick Manuel Toro Monjaraz
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ESPGHAN

JUNE 2026

Highlights from the ESPGHAN 2026

From June 24 to 27, the 58th edition of the Annual Congress of the European Society of Paediatric Gastroenterology, Hepatology, and Nutrition was held in Lille, France. This congress brings together pediatric gastroenterologists, hepatologists, and nutritionists from around the world, making it one of the most important congresses in these paediatric specialties.



Photo: Shutterstock.

Rome V criteria and its presentation at ESPGHAN

Among the topics presented, the one that generated the most anticipation was the presentation of the pediatric Rome V criteria, and this was evidenced by the large number of people who filled the auditorium, leaving many outside, and with an online broadcast. The Rome V criteria differ from the Rome IV criteria in that they divide the pathologies of the upper and lower digestive tracts, in addition to removing the age division. Dr. Samuel Nurko introduced these criteria, and together with Drs. Rachel Rosen, Miguel Saps, Carlo Di Lorenzo, Ana Maria Staiano, Marc Benninga, Nikhil Taphar, and Michiel van Wijk, each section that comprises this new nomenclature was presented. One of the topics that generated the most questions was the renaming of infant colic to infant distress syndrome. The justification for the change arises from the concept that these disorders are not exclusively generated by the intestine, but rather by the bidirectional gut-brain communication, and this sometimes limits the treatment exclusively to the gastrointestinal tract, forgetting that it also involves the brain. Another important change was the addition of esophageal functional disorders, disorders that already existed in the adult classification but were, with Rome V, added to the pediatric section. Additionally, supragastric belching and aerophagia were included, as well as pediatric feeding disorders, which contributes to the classification of these pathologies that are common in daily pediatric consultations. In pain disorders, Dr. Di Lorenzo emphasized the inclusion of centrally mediated abdominal pain, a disorder included in the adult section, but which had not yet taken a significant role in the pediatric section. This entity, unlike other abdominal pain disorders, is continuous, debilitating, and from the outset requires multidisciplinary treatment to help this group of patients. Undoubtedly, a disorder that will require further research to understand its pathophysiology and treatment. Finally, biliary pain, which, although not common in pediatrics, should be considered when we have patients with pain in the right upper quadrant and no stones or pathology that explains the origin of the pain [1, 2].

Without any doubt, the Rome V criteria were an important part of this congress; however, other presentations and papers were highly compelling, especially regarding gut microbiota.

Probiotics for hyperbilirubinemia in newborns

A randomized clinical trial evaluated the effect of supplementation with *Bifidobacterium breve* M-16V and *Bifidobacterium animalis* subsp. *lactis* Bb-12 as an adjunct treatment to phototherapy in 79 newborns with neonatal jaundice. The objective was to determine if the modulation of the gut microbiota could improve the clinical

response and counteract the dysbiosis induced by phototherapy.

Neonates who received probiotics had a higher frequency of bowel movements and a faster reduction in transcutaneous bilirubin levels compared to those treated solely with phototherapy. Additionally, a trend toward shorter hospital stays and better motor and language development scores between 18 and 24 months was observed, although these differences did not reach statistical significance.

The analysis of the microbiota showed an increase in bacterial diversity, a higher abundance of *Bifidobacterium breve*, and an increase in the *Bifidobacterium/Escherichia coli* ratio. Likewise, the metabolomic analysis revealed a profile compatible with increased DNA synthesis, cell growth, and regulation of the inflammatory response, suggesting a possible role of the microbiota in bilirubin metabolism and neurodevelopment.

Overall, these findings support the potential of *Bifidobacterium* M-16V and Bb-12 strains as a complement to phototherapy to promote the restoration of gut microbiota and optimize bilirubin clearance. However, multicenter studies with larger sample sizes are required to confirm these benefits and define their incorporation into clinical practice [3].

Microbiota as a tool for predicting late-onset sepsis in newborns

A multicenter case-control study evaluated whether the intestinal microbiota profile can early identify very premature newborns at risk of developing late-onset sepsis (LOS). Faecal samples from 58 infants with LOS confirmed by blood culture and 58 matched controls, obtained up to 10 days before the clinical onset of the infection, were analyzed using 16S rRNA gene sequencing.

The changes in the microbiota were closely related to the microorganisms causing sepsis. Cases of LOS caused by *Escherichia coli* and other non-staphylococcal pathogens showed a progressive increase in *Escherichia/Shigella* before the onset of symptoms. A predictive model based on machine learning (Random Forest) showed excellent performance in identifying LOS due to *E. coli* in the development cohort, although its performance decreased in the external validation cohorts.

The authors conclude that the intestinal microbiota profile could become a promising tool for early detection, especially of *E. coli* sepsis, which would open the possibility of implementing preventive strategies and targeted interventions in high-risk premature newborns. However, additional studies are required to improve and validate these predictive models before their clinical application [4].

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INTERNATIONAL MICROBIOTA OBSERVATORY

4th edition - 2026



For the fourth consecutive year, the Biocodex Microbiota Institute commissioned Ipsos bva to conduct a major survey on the microbiota, with the aim of assessing people's knowledge and understanding of microbiota. Part of the 2026 survey focused on the development of the gut microbiota during the first 1,000 days of life. Here are some of the key findings.

Awareness of the "first 1,000 days" and baby microbiota development is limited, though higher among pregnant women

43% **have heard of the scientific concept** of the **"first 1,000 days of life"**
→ **62%** among pregnant women

65% are aware that **administering antibiotics during the early stages after birth can significantly** impact the gut microbiota development
→ **72%** among pregnant women

29% only are aware that **by age 5, the gut microbiota becomes similar to that of an adult**
→ **40%** among pregnant women

11% only know that **the baby microbiota does not start developing in utero**
→ **20%** among pregnant women

Information by healthcare professional monitoring the child remain insufficient

57% » received information about the importance of **preserving their child's microbiota balance to prevent future diseases**

55% received explanations on how the **antibiotic prescription for newborns can affect microbiota health** and possible mitigation measures

39% received information on **microbiota development in the first 1,000 days**

» Methodology:

This fourth edition of the International Microbiota Observatory was conducted by Ipsos-bva on 7,500 individuals across 11 countries (France, Portugal, Poland, Finland, Italy, Germany, USA, Brazil, Mexico, China, and Vietnam) **between February 3 and March 13, 2026.**

Focus on young researchers



Photo: Adobe Stock

Why do different people behave differently to probiotics?





- **Sourav Goswami**
- **Alisha Ansari**
- **Dr. Tarini Shankar Ghosh**

Indraprastha Institute of Information Technology Delhi, India

When probiotics meet the gut microbiome

Walk into any pharmacy and you will find an entire shelf devoted to probiotics. Among the most common are *Bifidobacterium* species, marketed for gut health, immune support, healthy aging, and an expanding range of clinical conditions. Yet probiotic benefits sharply differ among individuals. Clinical trials consistently show that while some people experience clear and lasting improvements, others derive little or no benefit. One reason could be the persistence of the probiotic in the individual's gut and which could be influenced by the gut microbiome that already exists before a probiotic is consumed. Think of it like a new tenant moving into a crowded building: the incoming bacterium has to compete for resources and find its place among microbes that were already there. Its ability to colonize and persist depends not just on its own traits but on the resident community it walks into. Previous studies had implicated baseline microbiome composition in probiotic engraftment, but they were limited to small cohorts and individual strains, leaving open whether microbiome features can reliably predict probiotic success. In this study, we address this question through the largest global meta-analysis of its kind, identifying the microbial features that shape Bifidobacterium colonization and introducing a framework to predict an individual's receptiveness to probiotic treatment [1].

Bifidobacteria keep selective company

Investigating 51,244 gut microbiomes (from 149 study-cohorts, 45 countries), we observed consistent sets of bacteria being associated with specific *Bifidobacterium*. Butyrate producers and members of the genus *Collinsella* tended to show consistent positive links with certain *Bifidobacteria*, while certain pathobiont Firmicutes and Bacteroidota behaved negatively. We captured these patterns as Association-Scores, a measure of how reliably a microbe accompanies a given *Bifidobacterium*.

These scores were stable and recalculated across different diseases and compared against apparently non-diseased individuals, the associations largely held, and the same was true after adjusting for host properties like diet, medication, clinical factors and also for certain microbiome properties. Bacteria "helping" *Bifidobacterium* in healthy guts tended to do so in sick ones too, which points to a real microbial relationship rather than side effects of illness itself. Mapping only the associations that passed all checks revealed a compact, consistent pattern: *Collinsella* and a handful of oral, *Bacteroidetes*, and *Firmicutes* groups linked reliably to specific bifidobacteria, no matter which lens was used to look for them (**figure 1**).

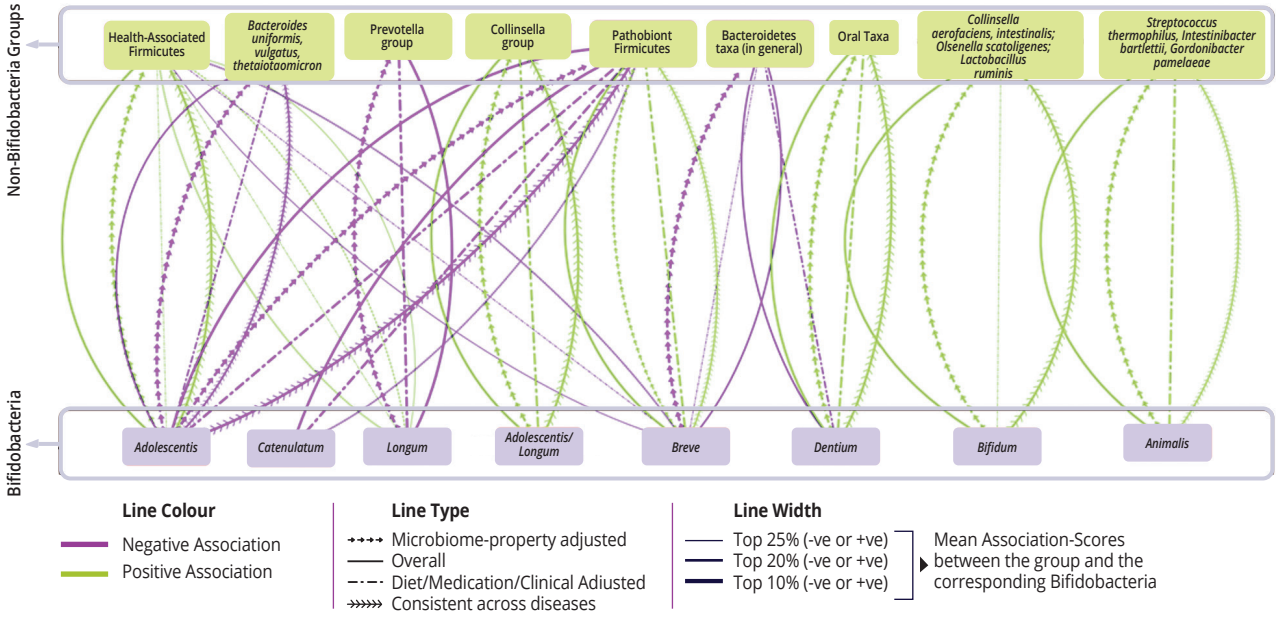
The Receptive-Score

We then asked whether these patterns could predict something practical: whether a probiotic actually persists after someone takes it? We developed the Receptive-Score, which measures how welcoming a baseline microbiome is to a specific *Bifidobacterium* using its known microbial associates and baseline abundance.

Investigating eight independent probiotic trials, the Receptive-Score combined with baseline *Bifidobacterium* levels correctly

Sourav Goswami and **Alisha Ansari** are joint first authors and PhD scholars working in Microbiome Informatics Lab headed by **Dr. Tarini Shankar Ghosh**, in the Department of Computational Biology at the Indraprastha Institute of Information Technology (IIIT-Delhi), India. Their work focuses on large-scale, multi-cohort microbiome analysis, probiotic science, and building data-driven tools that translate population-level patterns into personalized predictions.

FIGURE 1 **Network diagram summarizing the most consistent association patterns between Bifidobacteria and non-Bifidobacteria in different scenarios.**



predicted post-treatment persistence or increase in 69.23% of trial-probiotic combinations. In a *B. longum* trial [2], people who became persisters had significantly higher baseline Receptive-Scores than non-persisters. In two trials [3, 4] with clinical follow-up, higher scores also lined up with better clinical outcomes, including fewer gastrointestinal symptoms and lower inflammation markers, though sample sizes were too small for firm conclusions there.

> What the genes reveal

To understand what might drive these relationships, we examined the genomes of 66 non-*Bifidobacterium* taxa. We identified specific genes and metabolic pathways in bacteria that showed strong positive associations with the health-associated *B. adolescentis* and *B. longum*, revealing a cooperative cross-feeding network. Many of these bacteria encoded enzymes involved in the breakdown of complex dietary carbohydrates and the production of metabolites such as acetate that can be exchanged within the microbial community, creating a nutrient-rich environment that supports *Bifidobacterium* persistence. In contrast, bacteria associated with *B. breve* and *B. dentium* were enriched for genes involved in metal ion acquisition and scavenging, suggesting adaptation to resource competition and inflammatory conditions rather than metabolic cooperation. These findings indicate that the ability of different *Bifidobacterium* species to colonize in the gut may depend not only on their own biology but also on the metabolic capabilities of their microbial neighbours.

> Predicting how your gut microbes will change

While the study still has certain gaps (limited representation of non-industrialized populations, species-level investigations while probiotics are typically characterized at strain level, limited data on clinical outcomes), this framework of capturing the receptivity of a microbiome to a given microbe can be generalized to build "digital twin" models of a person's gut community that let scientists test probiotics, dietary changes, or other microbial therapies *in silico* before ever trying them on an actual patient.


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Key points

- A gut's existing bacteria shape whether a *Bifidobacterium* probiotic will persist
- Receptive-Scores predicted persistence in 69.23% of trial-probiotic pairs tested
- Analysis of functional signature linked to these association patterns identifies cross-feeding as a likely mechanism

[CONCLUSION]

This work shows that a person's existing gut bacteria actively gatekeep whether a probiotic takes hold. Reading that baseline community may one day help clinicians choose the right Bifidobacterial strain for the right person, rather than relying on trial and error.

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By Prof. Satu Pekkala

Academy of Finland Research Fellow, Faculty of Sport and Health Sciences, University of Jyväskylä, Finland

GUT MICROBIOTA

Gut microbiome is associated with recurrence-free survival in patients with resected high-risk melanoma receiving adjuvant immune checkpoint blockade

Immune checkpoint blockade (ICB) can be used to treat high-risk melanoma, but the results vary. About 25–40% of patients still have a recurrence of the disease, and some develop serious side effects. Therefore, better biomarkers are needed to predict who will benefit from the treatment, so that therapy can be tailored to each patient and work more effectively. Increasing evidence suggests that the gut microbiome influences ICB treatment outcomes in melanoma patients. However, microbes as biomarkers have shown great variability between studies. In this study, fecal samples from 674 patients enrolled in a phase III clinical trial – investigating the combination of nivolumab plus ipilimumab vs. nivolumab alone across five geographic regions – were analyzed to evaluate the predictive potential of the gut microbiome.
Shotgun metagenome sequencing identified bacterial strains linked to recurrence after treatment. *Eubacterium, Ruminococcus,* Firmicutes, and *Clostridium* have previously been associated with immune checkpoint therapy outcomes. *Eubacterium* may enhance immunotherapy via anti-inflammatory effects, while *Clostridium* and Firmicutes influence T cell-mediated immune regulation. *Ruminococcus* shows mixed pro- and anti-inflammatory roles. Additional taxa associated with recurrence included *Lawsonia, Bariatricus*, and *Blautia*, the latter also previously linked to ICB treatment outcomes. Pathway analysis showed that *Eubacterium* and *Ruminococcus* correlated with carbohydrate metabolism that has been formerly linked to the microbiome and cancer treatment success.
Overall, the findings of the study suggest that gut bacterial markers can predict recurrence after adjuvant ICB treatment in melanoma, supporting their potential as clinically valid biomarkers to guide personalized therapy.

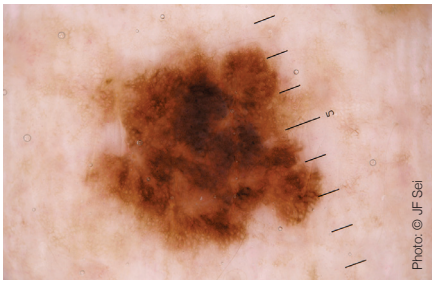


Photo: © JF Sei

Usyk M, Hayes RB, Knight R, *et al.* Gut microbiome is associated with recurrence-free survival in patients with resected high-risk melanoma receiving adjuvant immune checkpoint blockade. *Cell* 2026; 189: 3940-49.e14.

GUT MICROBIOTA

Microbiome signature of Parkinson's disease in healthy and genetically at-risk individuals

Parkinson's disease (PD) is a major cause of disability, and its prevalence is growing fast worldwide. In PD, dopaminergic neurons of the substantia nigra are lost and aggregated α-synuclein accumulate in brainstem and cortical regions. PD symptoms can be treated with levodopa, but to slow or prevent severe disability early detection of individuals at risk would be crucial. Genetic variants in the *GBA1* gene, encoding glucocerebrosidase, are found in about 15% of PD cases. Evidence also supports the role of the microbiota-gut-brain axis in PD, and microbiome analysis might enable identification of healthy individuals at higher risk of neurodegeneration.
This cross-sectional, multicenter study analyzed patients with PD carrying a *GBA1* gene variant, patients with PD not carrying the variant, healthy controls, and non-manifesting carriers of a *GBA1* variant, i.e. not diagnosed with PD. A total of 540 participants were included. Compared to healthy controls, non-manifesting carriers presented worse motor symptoms. Being carrier or non-carrier of *GBA1* variant did not influence the microbiome, whereas beta-diversity difference between healthy controls and either group was significant. The authors then pooled all patients with PD regardless of their genetic status to identify PD microbiome signature with optimally powered analysis. In PD, Actinobacteriota, Bifidobacteriaceae, *Streptococcus mutans, Bifidobacterium longum, Bifidobacterium dentium* and *Lactobacillus paragasseri* were enriched among many other taxa. In total, over 25% of the microbiome was altered compared to healthy controls. An interesting finding was that the microbiome of non-manifesting carriers of *GBA1* variant was an "intermediate" between healthy controls and patients with PD. This was associated with disease progression and symptoms indicative of future development of PD. The same phenomenon was reproduced in three independent cohorts from the United States, Korea and Turkey.
The authors reckon that gut microbiomes could be used to identify both genetically and non-genetically at-risk individuals who may be progressing toward PD and serve as an early disease marker.

Menozzi E, Ren Y, Geiger M, *et al.* Microbiome signature of Parkinson's disease in healthy and genetically at-risk individuals. *Nat Med* 2026; 32: 2096-106.

GUT MICROBIOTA

Antibiotic use and gut microbiome composition links from individual-level prescription data of 14,979 individuals

Recurrent and long-term use of antibiotics is associated with many non-communicable diseases likely due to alterations in gut microbiome. Known short-term alterations include decrease in diversity and increased abundance of potential pathogens. However, large population-based investigations examining the long-term effects of antibiotics on gut microbiomes have not been conducted.
This study combined individual-level data from the Swedish Prescribed Drug Register with fecal metagenomes of 14,979 adults to investigate the associations between oral antibiotic use over 8 years and gut microbiome. Participants using antibiotics at the time of sampling or with an ongoing infection were excluded.
The authors observed that gut microbiome diversity decreased with each additional antibiotic course within three periods: < 1 year, 1–4 years and 4–8 years before fecal sampling. Of the different antibiotics, clindamycin, fluoroquinolones and flucloxacillin had the largest effect on diversity, whereas no associations were detected for extended-spectrum penicillins. Even a single course of tetracyclines, flucloxacillin, fluoroquinolones, clindamycin, sulfamethoxazole-trimethoprim, cephalosporins or macrolides less than 4 years or 4–8 years before sampling caused reduced diversity. Taxonomically, use of clindamycin, fluoroquinolones and flucloxacillin was associated with a greater abundance of *Enterocloster bolteae, E. citroniae, Flavonifractor plautii, Ruminococcus B gnavus* and *Eggerthella lenta*, species previously associated with cardiometabolic risks. Overall, species negatively associated with antibiotic use were associated with better cardiometabolic and inflammatory outcomes.
The authors conclude that their results could inform future guidelines on outpatient antimicrobial practices, which should prioritize antibiotics that have a lower impact on the gut microbiome.


Photo: Shutterstock

Baldanzi G, Larsson A, Sayols-Baixeras S, *et al.* Antibiotic use and gut microbiome composition links from individual-level prescription data of 14,979 individuals. *Nat Med* 2026; 32: 1351-61.

VAGINAL MICROBIOTA

Vaginal microbiome profiles: a potential indicator of preterm birth risk

The vaginal microbiome undergoes changes throughout pregnancy and may help predict the risk of preterm birth.
To further investigate this association, researchers analyzed data from 677 pregnant women enrolled in the geographically and demographically diverse U.S. ECHO (Environmental influences on Child Health Outcomes) cohorts, of whom 12% delivered before 37 weeks of gestation. After adjusting for major maternal factors (including age, ethnicity, education level, and parity), women whose vaginal microbiome was dominated by *Lactobacillus crispatus* had a lower risk of preterm birth. In contrast, those with a more diverse microbiome (not dominated by lactobacilli) or one predominantly dominated by *Lactobacillus iners* were at higher risk. By combining the relative abundance of three key bacterial species (*L. crispatus, Gardnerella vaginalis*—which is associated with bacterial vaginosis—and *Prevotella timonensis*) with maternal characteristics, the researchers developed a predictive model with an area under the curve (AUC) of 0.77, a level of performance considered satisfactory by the authors for a clinical prediction tool.


Photo: Shutterstock

These findings suggest that the vaginal microbiome, when considered alongside maternal factors, could improve obstetric risk stratification and help identify women at increased risk of preterm birth.

McKee KS, Bassis CM, Golob J, *et al.*; ECHO Cohort Consortium. Vaginal microbiome structure in pregnancy and host factors predict preterm birth: Results from the ECHO Cohort. *Ann Epidemiol* 2025; 112: 110-8.



Photo: Shutterstock.





By Dr. Patrick Uzochukwu Eze[1,2], Prof. Lucky Legbosi Nwidu[2]

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Prof. Lucky Legbosi Nwidu is recipient of the 2024 Henri Boulard Grant awarded by the Biocodex Microbiota Foundation

Millets as powerful prebiotics

Millets have gained renewed scientific attention because they are rich in fibers, resistant starch and polyphenols that may support gut microbiome health. However, social media claims exaggerate their clinical benefits. Current evidence indicates that millets are nutritious whole grains with promising microbiome-modulating properties, but they should not be regarded as "superfoods" capable of restoring the gut microbiome or preventing disease.

> Are the claims justified?

Claims that modern diets "destroy" the gut microbiome and that millets uniquely restore gut health are not supported by strong clinical evidence. Although ultra-processed foods may adversely affect microbial diversity, the gut microbiome is influenced by multiple factors including age, medications, lifestyle and diet. Current human studies do not justify claims that millets alone can restore gut microbiota.

> How do millets influence the gut microbiome?

Millets contain fermentable fibers, resistant starch and polyphenols that reach the gut and are metabolized by beneficial bacteria (*Bifidobacterium, Lactobacillus*…). Fermentation produces short-chain fatty acids (such as propionate or butyrate), which support gut barrier integrity, immune regulation and metabolic health. Millet polyphenols are converted into bioactive metabolites that may reduce oxidative stress and promote beneficial microbial populations.

> Current limitations

Most evidence come from *in vitro* experiments, animal studies and small human trials. Although improvements in glycemic control, lipid metabolism and microbial composition have been reported, clinical evidence remains insufficient to conclude that millets uniquely restore the gut microbiome or prevent chronic disease. Elaborate, long-term randomized clinical trials are still needed.

> Practical implications

Healthcare professionals should encourage overall diet quality rather than reliance on "superfoods" only. Diets rich in vegetables, fruits, legumes, whole grains, nuts and seeds provide diverse fibers that support a healthy gut microbiome. Millets should be promoted as one nutritious whole-grain option within a balanced diet. Patients should also be cautious against misleading social media claims regarding foods that allegedly "repair the gut" or cure disease.

[CONCLUSION]

Millets represent nutritious, climate-resilient whole grains with potential benefits for gut microbiome health. Nevertheless, current clinical evidence supports their use as part of a diverse, plant-rich diet rather than as a unique superfood or cure for disease.



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WHAT IS FUNCTIONAL DYSPEPSIA?

and how is it related to my microbiota?

Functional dyspepsia (FD) is a **chronic disorder of gut-bain interaction (DGBI)** defined by **abdominal symptoms** originating from the **upper gastrointestinal region**. It is often associated with **psychological factors** and could be related to unbalanced **gut microbiome** [1,2].

FD is diagnosed according to Rome V criteria

DID YOU KNOW? [1, 2, 3, 4, 5]

one of the most common digestive pathologies

1 in 10
adults, under-diagnosed

Disorder of the gut-brain interaction

Chronic disorder with common overlap with gastroparesis, reflux, irritable bowel syndrome and bloating symptoms

SYMPTOMS AND SUBTYPES

Functional dyspepsia is a symptom-based disorder with no tissue nor organ damage.

- **Early satiation** *« I feel full even if I don't eat much. »*
- **Postprandial fullness** *« I feel like I have a stone in my stomach. »*

*at least 2 days per week**

- **Epigastric pain** *« It's like being stabbed from the inside out. »*
- **Epigastric burning** *« My stomach burns like it's on fire. »*

*at least once per week**

Postprandial Distress Syndrome (PDS)
→ the most common form
Symptoms occur/worsen within 2h after meals and can include: pain, burning, nausea, bloating, excessive belching.

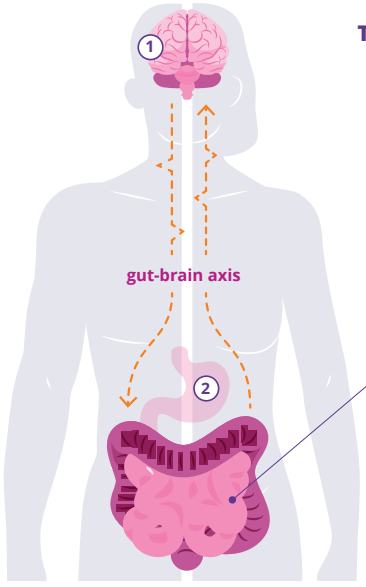
Epigastric Pain Syndrome (EPS)
PDS criteria must be absent. May or may not be related to meals.

**Criteria fulfilled for the last 3 months with symptom onset since at least 6 months*

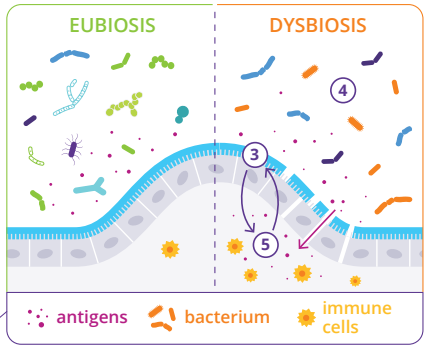
RISK FACTORS: Helicobacter pylori infection, being female, using certain pain relievers such as aspirine and ibuprofen, smoking, anxiety or depression, history of childhood physical or sexual abuse. [4]

THE BIOLOGICAL MECHANISMS [1, 2, 4]

1. **Psychological factors** including stress, anxiety or depression
2. **Stomach hypersensivity** Abnormal relaxation, visceral hypersensitivity, slow emptying, neuronal dysfonction
3. **Disruption of the gut barrier** Increased intestinal permeability
4. **Altered gut microbiota** Change in microbes composition and function, mostly in the duodenum
5. **Intestinal immune activation** Excessive response with immune cells infiltration



THE GUT MICROBIOME IMPACT ON FD [1, 2, 4]



→ Restoration of **gut microbiota**
→ Restoration of **gut barrier permeability**
→ Reduction of **visceral hypersensitivity**
→ **Anti-inflammatory** action
→ Regulation of **gastrointestinal motility** [8]

HOW TO RELIEVE SYMPTOMS AND IMPROVE QUALITY OF LIFE? [2, 4, 6, 7]


healthy diet


healthy lifestyle


specific medication


psychotherapeutic interventions
Cognitive Behavior Therapy (CBT), hypnosis, psychodynamic, relaxation...



Your doctor will advise you on the right combination of specific medication and alternative solutions.

BIOCODEX Microbiota Institute

For more information about the microbiota go to www.biocodexmicrobiotainstitute.com
References on the website

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