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Dear readers,

When pain lingers: the microbiota and the gut–brain axis confront the enigma of chronic pelvic pain

Chronic pelvic pain remains one of the most complex and frustrating challenges in clinical practice. Often diffuse, persistent, and disproportionate to visible lesions, it affects patients across multiple specialties—from gynaecology to gastroenterology and urology—while frequently escaping organ-centred approaches. Endometriosis, irritable bowel syndrome, and bladder pain syndrome differ in diagnosis, yet share a common clinical reality: persistent pain that is difficult to relieve.

These conditions are increasingly understood as disorders of altered pain processing rather than isolated organ diseases. Peripheral and central sensitization, neural remodeling, lowered pain thresholds, and viscero-visceral cross-talk help explain why pain can persist, spread, and resist conventional treatments long after the initial trigger has subsided.

Within this neuro-immune framework, the gut microbiota has emerged as a key modulator of chronic pain. Through its interactions with immune pathways and sensory neurons, it can influence inflammation and nociceptive signalling. Evidence is now accumulating in IBS and, more recently, in endometriosis, pointing to shared mechanisms involving microbial dysbiosis, pelvic inflammation, and hormonal metabolism—and opening new perspectives for integrated, microbiota-targeted therapeutic strategies.

By exploring how microbes interact with neural pain pathways, this issue of *Microbiota Mag* invites a shift in perspective. In chronic pelvic pain, the gut may be far more than a bystander—and listening to the microbiota may help pave the way toward more coherent and effective patient care.

Enjoy your reading!

Therapeutic approaches targeting the microbiota —through diet, prebiotics, probiotics, postbiotics, or even faecal microbiota transplantation— are still in their early days, but they represent a promising complement to existing treatments.

JAMES HOFFMAN - I need to tell you why coffee makes you poop

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Chronic pelvic pain and brain-gut axis: what is the involvement of the gut microbiota?

Chronic pelvic pain is common, debilitating and particularly affects women. It can be caused by identified lesions, such as endometriosis, or functional syndromes characterised by visceral hypersensitivity, such as irritable bowel syndrome (IBS) or interstitial cystitis. In such cases, peripheral and central sensitisation mechanisms trigger a lowering of pain thresholds and lead to diffuse and difficult-to-treat symptoms.

The gut microbiota plays an increasingly important role in understanding this type of pain. Through its interactions with the immune and nervous systems and metabolism, it exerts a direct effect on the excitability of sensory fibres and pain circuits. Some bacterial metabolites favour neuronal inflammation and hyperexcitability, while others exert a protective effect *via* anti-inflammatory mediators or endogenous opioids.

In IBS, dysbiosis characterised by a loss of beneficial bacteria (*e.g. Faecalibacterium, Roseburia*) and increase of opportunistic bacteria has been documented, with experimental evidence of its causal role. In endometriosis, the gut microbiota may contribute to lesion progression and oestrogen modulation, suggesting a bidirectional interaction between the gut microbiota and disease.

These findings are paving the way for new therapeutic avenues including probiotics, prebiotics, postbiotics and even faecal-microbiota transplantation. Although data remain preliminary, targeting the microbiota appears to offer a promising strategy for improving the management of chronic pelvic pain.


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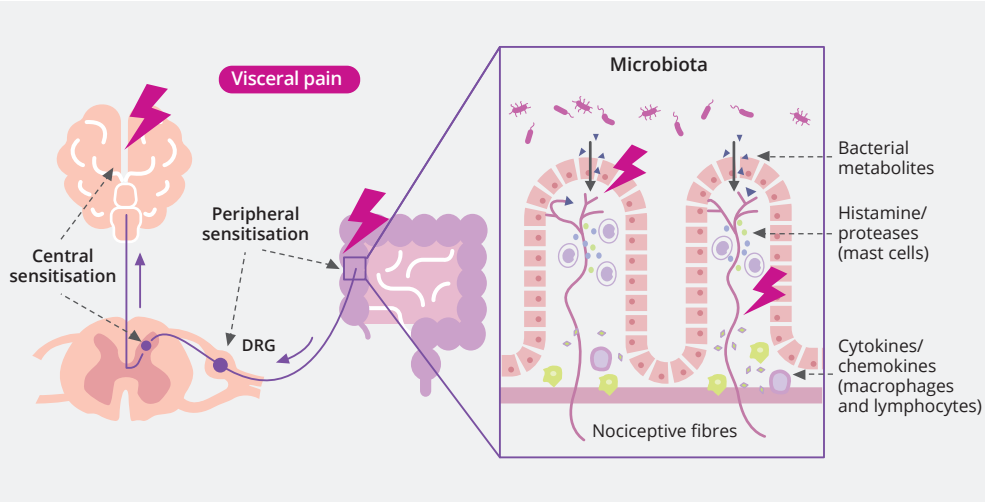
From nociperception to chronic pelvic pain

Pain is defined as an unpleasant sensory and emotional experience, which may or may not be associated with tissue damage. It is referred to as chronic when the pain persists for over three months, resists treatment and leads to changes in functional abilities and social relationships. Chronic visceral pelvic pain is characterised by deep, throbbing and diffuse pain, making its diagnosis complex and imprecise. This type of pain is particularly common in women and may require input from several internal-medicine specialists, *i.e.* gastroenterology, gynaecology and urology. Pelvic pain can be caused by several types of organ lesions (*e.g.* endometriosis) or functional syndromes characterised by visceral hypersensitivity (*e.g.* irritable bowel syndrome (IBS) or interstitial cystitis). A combination of several painful pel-

FIGURE • 1 **Mechanisms of abdominal pain.**

It is referred to as chronic when the pain persists for over three months, resists treatment and leads to changes in functional abilities and social relationships.



vic conditions is often observed in a single patient. Other patients may experience debilitating pain for which no specific cause has been identified. Internal-medicine specialists usually analyse pain as the expression of a single lesion. While it is certainly necessary to treat the injured organ, this may sometimes not be enough to provide relief to patients with chronic pelvic pain. Some patients present with an extensive set of symptoms, combining sensitivity disorders with issues affecting several pelvic organ functions at the same time. Such phenomena are linked to sensitisation mechanisms that appear several months or years after the onset of pain [1]. This type of sensitisation is characterised by a lowering of sensitivity thresholds leading to increased pain or pain provoked by stimuli of normally non-nociceptive intensity (*e.g.* intolerance to the sensation of rectal filling). Pain spreading over time has also been described. In fact, the painful sensation can persist despite the lack of stimulation (*e.g.* post-defecation pain). Finally, pain spreading beyond the stimulated area is also observed (*e.g.* pain during rectal filling causing bladder pain).

On a pathophysiological level, a distinction is made between psychological, adaptive and chronic pain. Acute pain occurs following an initial painful stimulus (heat, pressure, pH variation or algogenic substances) which activates receptors at the endings of the peripheral nociceptive fibres. In the case of the viscera, sensory innervation occurs in the spinal nerves whose endings are located in the muscles and/or mucosa of organs with cell bodies in the dorsal root ganglia [2]. Under physiological conditions, activation of Aδ or C afferent fibre receptors generates an action potential (AP) that is translated into nociceptive information in the dorsal root ganglia. This information is then transmitted to the second-order spinal neurons in the dorsal horn. Second-order neurons transmit information to the thalamus *via* the spinothalamic and spinoreticulothalamic pathways. In the thalamus, the third neuron transmits the message to various areas in the cortex (prefrontal, cingulate, somatosensory or insular cortex). This third neuron is responsible for transforming nociception into pain, with its affective-emotional, sensitive, cognitive and behavioural dimensions.

In the case of chronic visceral pain observed in conditions such as IBS or chronic inflammatory bowel disease (IBD), particularly in remission, hypersensitivity of intestinal nociceptive fibres has been documented [3].

This hypersensitivity may be partly due to gut permeability alterations, thereby increasing the passage of food or bacterial antigens, leading to inflammatory mechanisms with mast-cell recruitment and release of pro-inflammatory mediators such as histamine or proteases. This inflammatory environment could contribute to hyperexcitability in the nociceptive fibres, which in turn contribute to maintaining the inflammatory microenvironment by releasing neuropeptides (Substance P: SP and Calcitonin-Gene Related Peptide: CGRP). These repeated stimuli trigger phenotypic and excitability alterations to the nociceptive neurons in the Dorsal Root Ganglion (DRG), known as peripheral sensitisation. In the spine, repeated APs from DRG neurons increase the release of excitatory neurotransmitters such as glutamate [4]. In the long term, this leads to synaptic reinforcement by increasing the number of glutamatergic receptors, combined with dysfunction of the inhibitory systems, resulting in sensitisation of the spinal neurons [4]. Thus, central sensitisation is a pathological state of nociception function linked to its dysfunction, with a strengthening of facilitatory systems and lowering of pain-inhibitory systems.

The microbiota, a potential pain modulator

The factors responsible for visceral fibre hypersensitivity are still poorly understood, but the gut microbiota could play a role (**figure 1**).

The microbiota is defined as all of the microorganisms living in a specific environment in a host. It is mainly composed of bacteria, but also includes viruses, yeasts and protozoa. These micro-organisms may be present without having any impact on their host (commensalism) or they may interact closely with it.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The number of bacteria colonising the human body ($3.8 \cdot 10^{13}$) is roughly the same as the number of human host cells in adulthood ($3.0 \cdot 10^{13}$) [5]. The gut microbiota plays a key role in the two-way communication between the gut and various organs, including the brain. For several years this has been referred to as the microbiota-gut-brain axis. In the past, studies have typically focused on the role of the microbiota in gastrointestinal disorders (irritable bowel syndrome, inflammatory bowel disease). More recently, it has been recognised that dysfunction of this axis is involved in the pathophysiology of many other conditions, including metabolic diseases (obesity, diabetes) and neurological diseases (autism, Parkinson's disease, depression).

The gut microbiota and the brain communicate with each other using several pathways such as the vagal nervous system, the immune system and the humoral pathways, after modulation of enteroendocrine functions. Nociceptive afferent fibres can also be directly modulated by various bacterial metabolites [6]. The main mediators identified are bacterial metabolites (*e.g.* short-chain fatty acids, secondary bile acids), neurotransmitters and neuromodulators (*e.g.*: GABA) and bacterial products (*e.g.*: PAMPs, tryptophan derivatives). Some molecules increase neuronal excitability by activating nociceptors, producing Nerve Growth Factor (NGF) and increasing local inflammation. Others, however, have the opposite effect (*e.g.*: GABA) and can inhibit the transmission of the nociceptive message by producing endogenous opioids or anti-inflammatory mediators.

Clinical examples: irritable bowel syndrome and endometriosis

Irritable bowel syndrome

IBS is characterised by chronic functional intestinal disorders, mainly combining abdominal pain and bowel disorders (diarrhoea, constipation or both in alternation). This condition affects around 5-10% of the population, primarily young adult females. The pathophysiology of IBS is not yet fully understood but it has been extensively documented that gut-brain-communication changes lie at the root of digestive motility disorders and visceral hypersensitivity. On a central level, IBS patients experience impaired information processing, hypervigilance and increased anxiety. In recent years, the gut microbiota has been proposed as one of the causative factors in IBS [7] and several studies have highlighted changes in the composition and diversity of the microbiota in IBS.

For instance, a systematic review showed that the phylum Firmicutes decreased and the phylum Bacteroidetes increased in patients with IBS and diarrhoea [8].

A reduction in the abundance of the genus *Bifidobacterium* was also found in stool and mucosal samples from IBS patients, along with an increase in the genus Bacteroides. An increase in pathogens (*i.e. Escherichia coli* and *Enterobacterium* families) was also observed. An enrichment of certain bacterial taxa such as *Enterobacteriaceae, Streptococcus, Fusobacteria, Gemella and Rothia*, and a depletion of bacterial genera recognised as beneficial to health, such as *Roseburia* and *Faecalibacterium*, were also observed in IBS patients. In IBS, the causal role of these kinds of microbiota changes has been strongly suggested in view of the ability to induce some symptoms in preclinical models after the transfer of stools from IBS patients [9]. In addition, the role of several mediators produced by the intestinal microbiota, such as LPS, short-chain fatty acids and secondary bile acids, has been suggested in chronic abdominal pain, visceral hypersensitivity and gut inflammation [10]. The gut microbiota could therefore be a major cofactor in chronic abdominal pain and associated inflammation.

Endometriosis

Endometriosis is defined as the transplantation of endometrial cells outside the uterine cavity, which may be facilitated by chronic pelvic inflammation. The main symptoms of endometriosis are chronic pelvic pain, gastrointestinal problems and infertility. A systematic review analysed studies of the gut microbiota in women with endometriosis and chronic pelvic pain [11]. A total of 28 clinical studies and six animal studies were included in the review. In these human and animal studies, increased gut microbiota diversity was observed in the endometriosis groups. However, there was no clear consensus on the composition of the microbiota associated with endometriosis. None of the studies analysed composition or diversity based on the characteristics of pain.

Animal studies (6/6) confirmed the bidirectional relationship between the gut microbiota and the onset and progression of endometriosis. Endometriosis induction in mice actually induced changes in the gut microbiota.

In the study by Yuan, no early-stage differences were observed after endometriosis induction versus a control group. Differences appeared 21 days after the start of the experiment and increased thereafter, with decreased diversity and richness of the microbiota in the endometriosis group, increased genera *Bifidobacterium, Proteobacteria* and *Verrucomicrobia*, decreased Bacteroidetes and Firmicutes phyla and an increase in the Firmicutes/Bacteroidetes ratio, reported [12]. Conversely, treatments targeting the microbiota, such as broad-spectrum antibiotics, reduced the volume and weight of endometriosis lesions, induced a decrease in endometrial lesion cell proliferation and inflammatory markers (cytokines, macrophages) [13]. Apart from inflammation, the gut microbiota could contribute to the pathophysiology of endometriosis through its role in regulating oestrogen metabolism. A specific microbiota called the estrobolome actually plays a central role in hormone regulation, especially oestrogen [11]. Estrobolome contains bacteria that produce beta-glucuronidase, which is an enzyme able to modify the active form of oestrogen. Gut microbiota disturbances could therefore lead to increased circulating oestrogen levels and favour the development of endometriosis. However, these studies did not investigate pain modulation.

Therapeutic prospects *via* the microbiota

The development of therapeutic approaches modulating the composition or function of the gut microbiota are becoming increasingly recognised as playing a complementary role in the current therapies used for the management of chronic functional digestive or pelvic conditions. The basic aim of these approaches is to restore a balanced and functional microbiota (*via* dietary interventions or prebiotics), or provide bacteria with beneficial effects for the host (probiotics). Specific combinations of probiotics, or specific species and strains [14], appear to have beneficial effects on overall IBS symptoms and abdominal pain [15]. Symbiotic approaches combining pre- and probiotics are also used. Approaches based on the transplantation of faecal microbiota also suggest a therapeutic potential in functional diseases such as IBS [16]. In addition, an increasing number of studies are suggesting that the effectiveness of the response to faecal-microbiota transplantation in IBS or to probiotic treatments is influenced by the composition of the microbiota of the recipient.

> In the case of endometriosis, two randomised clinical studies have suggested that probiotics were effective in improving pain [17, 18], although one study reported that the efficacy did not last after discontinuing the probiotics.

In general, the effectiveness of these approaches in the management of pelvic functional disorders, although demonstrated in some cases, remains limited by the small population sizes in the studies and, above all, by the wide diversity and variability of symptoms observed in these disorders, as shown in a recent literature review [15].

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By Prof. Harry Sokol

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Pooled analysis of 3,741 faecal metagenomes from 18 cohorts for the identification of reproducible microbial biomarkers at different stages of colorectal cancer

Commentary on the article by Piccinno et al. (Nature Medicine 2025 [1])

Associations between the gut microbiome and colorectal cancer (CRC) have been uncovered, but larger and more diverse studies are needed to assess their potential clinical use. The authors of this article used 12 metagenomic datasets of patients with CRC (n = 930), adenomas (n = 210) and healthy controls (n = 976; total n = 2,116) and added six new cohorts (n = 1,625) providing granular information on cancer stage and the anatomic location of the tumours. They improved CRC prediction accuracy based solely on gut metagenomics (average area under the curve = 0.85) and highlighted the contribution of 19 new species and distinct *Fusobacterium nucleatum* clades. Specific gut species distinguish left-sided versus right-sided CRC (area under the curve = 0.66) with an enrichment of oral-typical microbes. The authors identified strain-specific CRC signatures with the commensal species of *Ruminococcus bicirculans* and *Faecalibacterium prausnitzii*, showing subclades associated with advanced CRC. The analysis confirmed that the microbiome can be a clinical target for CRC screening and characterised it as a biomarker for CRC progression.

What do we already know about this subject?

••• CRC is the third most frequent and the second most lethal tumour type worldwide [2]. CRC originates in the epithelial layer of the proximal colon (right colon) or distal colon plus rectum (left colon). Progression from benign precancerous lesion (adenoma) to a malignant tumour (carcinoma) may take several years and is characterised by an accumulation of tumour-cell mutations, alteration in the gut mucosal barrier and intestinal inflammation.

The gut microbiome is proposed as one of the important hallmarks of cancer. Certain microbes have been put forward as major contributors to carcinogenesis, particularly *Escherichia coli pks+* and *Fusobacterium nucleatum* [3]. Several studies have observed distinct microbiome signatures in CRC patients when compared with patients with adenomas or healthy controls [4]. A few metagenomic studies also investigated microbiome changes along the adenoma-carcinoma sequence and based on the primary neoplasia location, and have suggested links between CRC and oral species. Further evidence points toward the enrichment of oral-typical microbes and of oral biofilm-forming species in the gut metagenomes of patients with proximal CRC. However, no metagenomic studies have gone beyond characterising already well-known strain-specific factors that influence CRC risk, and no untargeted searches for subspecies and strain-level genomic associations with CRC phenotypes are available.

What are the main insights from this study?

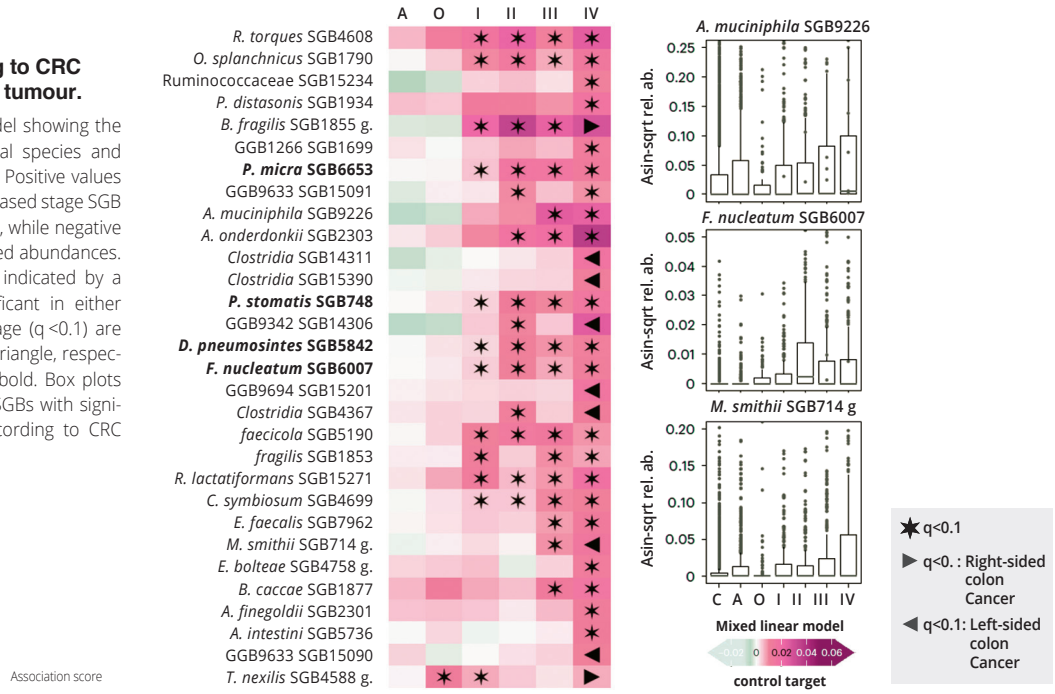
••• Using 3,741 samples from 18 cohorts and applying new strain-level computational methodologies, the authors investigated the links between faecal microbiota and CRC. They improved CRC prediction accuracy based solely on gut metagenomics, with an average area under the curve (AUC) = 0.85. The five SGBs (Species-level Genome Bins) assigned to the species *F. nucleatum* were more abundant in CRC than in the controls: *F. nucleatum subsp. animalis, vincentii*, *nucleatum, polymorphum*. This was in addition to other well-characterised CRC-associated microbes such as Parvimonas micra and *Bacteroides fragilis*. The authors also identified 19 additional uncharacterised SGBs with neither cultivated strains nor taxonomically defined species, which highlighted a more complex CRC-associated microbial signature than previously thought.

Although interstage microbiome variations during CRC progression are not as

FIGURE 1

Microbial signature according to CRC stage and location of primary tumour.

Coefficients of the mixed linear model showing the associations between each microbial species and each stage, compared with controls. Positive values (from pink to dark pink) indicate increased stage SGB abundances compared with controls, while negative coefficients (green) indicate decreased abundances. Significant associations (q <0.1) are indicated by a star. Associations also found significant in either right- or left-sided CRC for each stage (q <0.1) are indicated by a right- or left-pointing triangle, respectively. Oral SGBs are highlighted in bold. Box plots represent the distribution of three SGBs with significant variations in abundances according to CRC stages.



strong as those observed between CRC and controls, the authors found several biomarkers for advanced and metastatic CRC, as well as several microbial species consistently and monotonically increasing (or decreasing) from control to cancer or advanced disease. In particular, late-stage CRC was enriched in oral-derived species, such as *P. micra*, already involved in the stimulation of tissue invasion pathways and *Hungatella hathewayi*, which was shown to promote intestinal cell proliferation in in-vitro experiments (**figure 1**). Compared with the other stages, metastatic CRC had a higher abundance of *Methanobrevibacter smithii*, supporting previous findings linking methane producers with stage IV CRC. Stool samples from patients with CRC originating in the right or transverse colon were also enriched in oral species.

These findings strengthen the notion that the number and cumulative abundance of orally derived species are significantly higher in CRC samples than controls and adenomas, but also show that later stages of CRC were particularly enriched in oral species. However, many non-oral bacteria were also associated with CRC, including species previously associated with high cardiometabolic risk. Interestingly, adenoma and later cancer stages were enriched in species linked with poor cardiometabolic health and immune-mediated diseases.

What are the consequences in practice?

••• This is the largest and most accurate study ever carried out on faecal microbiota associated with CRC. A signature based solely on faecal microbiota offers a relatively good predictive value. In addition, differences between early and late stages have been identified. However, it should be noted that the study did not identify sufficiently accurate markers for the diagnosis of pre-cancerous lesions (adenoma). This work paves the way for microbiota-based tests for diagnosing CRC, but their use in clinical practice still requires validation and, above all, the detection of pre-cancerous lesions needs to be improved.

Key points

- Using 3,741 samples from 18 cohorts, the authors investigated the links between faecal microbiota and CRC
- A signature based solely on faecal microbiota provides a relatively good predictive value, with an average area under the curve (AUC) = 0.85
- However, the study did not identify sufficiently accurate markers for the diagnosis of pre-cancerous lesions
- Alongside differences between control and CRC subjects, alterations in the microbiota were observed according to the stage and topography of the primary lesion, particularly in terms of the abundance of oral bacteria

[CONCLUSION]

A signature based solely on faecal microbiota offers prediction with an average area under the curve (AUC) = 0.85. However, the study did not identify sufficiently accurate markers for the diagnosis of pre-cancerous lesions (adenomas). In addition to differences between control and CRC subjects, alterations in the microbiota were observed according to the stage and topography of the primary lesion, particularly in terms of oral bacteria abundance.

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By Prof. Emmanuel Mas

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Symptom-specific gut microbial and metabolic profiles in ADHD reveal SCFA deficiency as a key pathogenic mechanism

Comments on the original article by Wang et al. (Gut Microbes 2025) [1]

Previous evidence links gut microbiota to attention-deficit/hyperactivity disorder (ADHD) through the gut-brain axis. However, the specific microbiota contributing to symptoms remain unclear. To characterise the gut microbial profile linked to different symptoms and explore the mediation mechanism between microbiota alterations and the main ADHD symptoms, the authors of the article conducted shotgun metagenomic sequencing and faecal metabolomics analysis on 94 ADHD patients and 94 age- and gender-matched controls. They analysed the microbial characteristics of three subgroups presenting with different main symptoms of ADHD. Faecal microbiota transplantation in mice validated the hypothesis that gut microbial composition affects ADHD symptoms through metabolic alterations. This study provided further insight into the mechanisms underlying metabolic disturbances in ADHD and shed light on the role of gut microbiota in these processes.

What do we already know about this subject?

••• Attention deficit hyperactivity disorder (ADHD) is common in paediatrics, with a 3-5% prevalence. Various environmental factors play a role in the development of ADHD, including perinatal, social-emotional and nutritional factors. These risk factors also influence the gut microbiota (GM). Early alterations can disrupt neurological development. These effects could be mediated by the microbiota-gut-brain axis through three pathways (immune, neuronal and endocrine/systemic).

What are the main insights from this study?

••• One hundred and eighty-eight (188) children were included in a single-centre Chinese study, including 94 children with ADHD who were divided into three subgroups, 56 with predominant inattention symptoms (group IA), 9 with predominant hyperactivity-impulsivity symptoms (group HA) and 29 with combined symptoms (group C), and 94 controls (group TD).

The GM analysis found no difference between these three groups in terms of bacterial richness and diversity, the relative abundance of the 10 main genera and the *Firmicutes/Bacteroidetes* ratio. However, specific taxa were associated with ADHD subtypes (**figure 1**).

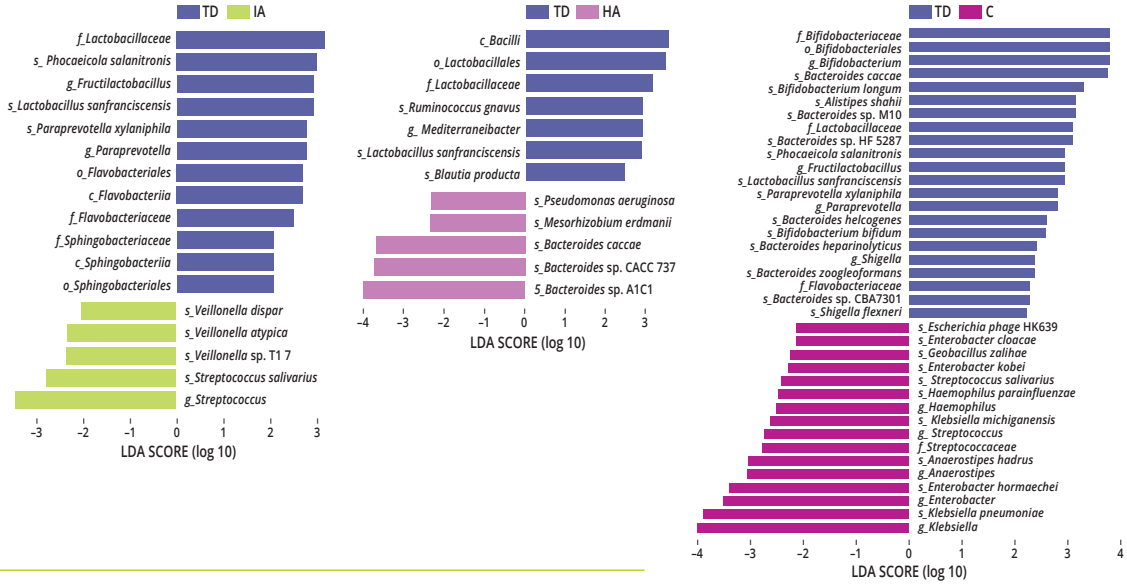
The family *Lactobacillaceae* and species *Lactobacillus sanfranciscencis* were enriched in the TD group. The TD group had more *Bifidobacteriales* than group C. In groups IA and C (inattention symptoms), the genus *Streptococcus* and species *Streptococcus salivarius* were identified as harmful bacteria, while family *Flavobacteriaceae, genus Paraprevotella, genus Fructilactobacillus, species Paraprevotella xylaniphila*, and species *Phocaeicola salanitronis* were beneficial. In groups HA and C (hyperactivity-impulsivity symptoms), the family *Lactobacillaceae* and species *Lactobacillus sanfranciscencis* were beneficial (**figure 1**).

The metabolomic profiles of ADHD children revealed disturbances in fatty-acid synthesis, with a significant reduction in the synthesis of unsaturated fatty acids and linoleic acid, as well as amino-acid metabolism (**figure 2**). The various symptoms, inattention, hyperactivity and impulsivity, were negatively correlated with different metabolites, imidazoleacetic acid and inattention ($p<0.001$). Thus, *Lactobacillus sanfranciscencis* had a direct effect and an effect mediated by imidazoleacetic acid on inattention.

An experiment with three cycles of faecal microbiota transfer (FMT) from children with ADHD and a low abundance of bene-

FIGURE 1

Specific differences in bacterial taxa between each of the ADHD groups.



Key point

- Gut microbiota alterations are specifically associated with the different symptoms of ADHD children. This effect is mediated by disturbances in fatty-acid metabolism

ficial *Lactobacillus sanfranciscencis* bacteria (FMT-A group) and controls (FMT-H group) was conducted in four-week-old male C57BL/6J mice. The mice then received either *Lactobacillus sanfranciscencis* (1 x10^8 organisms/mouse/day, group FMT-A-R1), or 150 mmol/L sodium acetate (group FMT-A-R2) or PBS for the controls (group FMT-A-C). Hyperactivity symptoms improved significantly in the FMT-A-R1 group but not in the FMT-A-R2 group, while inattention symptoms improved in both groups. Finally, fatty-acid and unsaturated fatty-acid synthesis was improved in the FMT-A-R1 and FMT-A-R2 groups versus FMT-A-C (**figure 3**).

What are the consequences in practice?

••• This study showed that GM alterations in ADHD children were symptom-dependent. The sub-group analysis showed that family *Lactobacillaceae* and species *Lactobacillus sanfranciscencis* were enriched in the TD group. In addition, after faecal transplantation with ADHD stools, treatment with *Lactobacillus sanfranciscencis* improved symptoms.

Fatty-acid and unsaturated fatty-acid synthesis was reduced in ADHD children. Similarly, after faecal transplantation with ADHD stools, treatment with short-chain fatty acids (imidazoleacetic acid) led to an improvement in symptoms.

FIGURE 3

Faecal transplantation with stools from ADHD children reduces fatty-acid synthesis, which is corrected by treatment with *Lactobacillus sanfranciscencis* or sodium acetate.

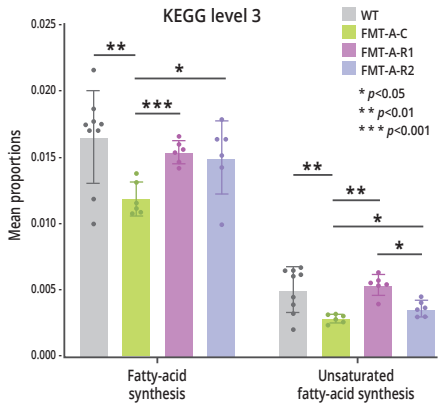
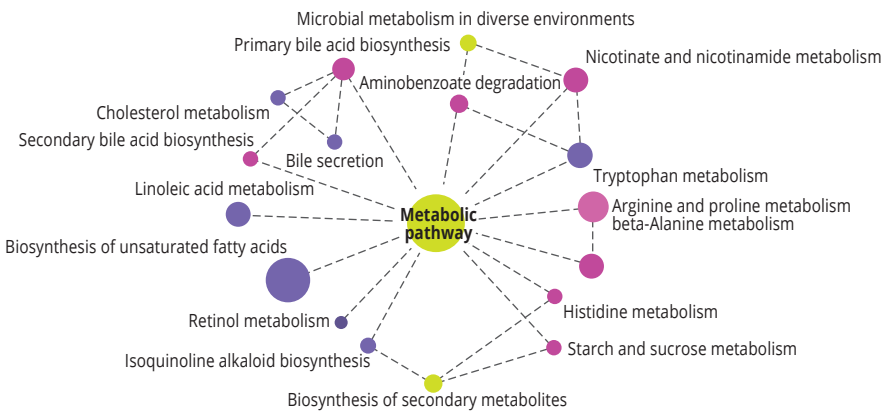


FIGURE 2 **Alteration of the different metabolic pathways, size of the circles corresponding to the number of genes involved.**



[CONCLUSION]

The findings of this study add to our understanding of the role the gut microbiota plays in children with ADHD, with metabolic alterations involved in the disturbance of the microbiota-gut-brain axis in this context.

Source

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XXV LASPGHAN

Merida, Yucatan, Mexico

By Prof. Natascha Sandy
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SEPTEMBER 2025

Microbiota and Pediatric Health: Highlights from LASPGHAN 2025

The 25th Congress of the Latin American Society for Pediatric Gastroenterology, Hepatology and Nutrition (LASPGHAN), held in Mérida, reinforced the central role of the microbiota in pediatric health and disease. The discussions emphasized a rigorous, evidence-based and strain-specific approach to probiotic use, aligned with the most recent ESPGHAN position paper and the forthcoming LASPGHAN consensus. The focus has shifted from empirical supplementation to indications supported by randomized clinical trials and meta-analyses, highlighting how microbial modulation can influence gastrointestinal, immune and metabolic outcomes across different stages of childhood. There is increasing recognition that early-life microbiota imprinting may have lifelong consequences for metabolic programming and immune tolerance, emphasizing the importance of perinatal nutrition, breastfeeding, and the avoidance of unnecessary antibiotics as key determinants of microbial resilience.

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Acute infectious diarrhea

Current guidelines recommend certain strains of *Saccharomyces* and *Lacticaseibacillus* as the best-documented strains for acute gastroenteritis and prevention of antibiotic-associated diarrhea in children [1-4]. Randomized controlled trials and meta-analyses consistently demonstrate a clinically relevant reduction in illness duration and stool frequency when these probiotics are used together with oral rehydration therapy. For antibiotic-associated diarrhea, both strains maintain moderate-quality evidence and favorable safety profiles, making them the most reliable options in routine pediatric care. Some *Limosilactobacillus* may also be considered in acute diarrhea and infantile colic, reflecting the increasing understanding that early-life microbial modulation supports immune tolerance and intestinal barrier maturation [5]. Beyond probiotics alone, emerging studies presented at LASPGHAN explored how combinations with prebiotic substrates can accelerate the restoration of microbiota diversity after infection or antibiotic exposure. This synergistic "biotic" approach may represent a new frontier for preventing recurrence and enhancing gut recovery in children.

Functional gastrointestinal disorders and colic

Beyond acute settings, the role of probiotics in functional gastrointestinal disorders is being more cautiously defined. Some


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strains of *Lacticaseibacillus* have shown potential to reduce the frequency of abdominal pain and improve quality of life in functional abdominal pain and irritable bowel syndrome when combined with dietary and behavioral measures [1, 3]. The overall strength of evidence remains modest, but emerging data suggest a role for specific strains as adjuvants within a multimodal management approach. In functional constipation, *Lacticaseibacillus* may also be used as an adjunct to standard therapy, while in infantile colic it remains the most consistently recommended strain. Preventive use of Lacticaseibacillus from birth to four months has been associated with lower incidence of colic in high-risk infants [2]. Strain selection should always consider patient phenotype, symptom pattern, and concomitant interventions such as fiber intake or behavioral therapy. Integrating probiotics or synbiotics into a holistic management strategy—rather than using them as isolated supplements—emerges as a key principle for optimizing outcomes in functional gastrointestinal disorders.

Food allergy and mucosal immunity

Increasing attention has been directed to the interaction between microbiota and immune development. *Lacticaseibacillus* administered for at least three months may help promote tolerance and clinical improvement in cow's-milk protein allergy [1 ,2]. These findings are biologically plausible given the immunomodulatory properties of the strain and its capacity to influence epithelial and cytokine responses. Nevertheless, further studies are needed to validate the magnitude and durability of these effects in long-term allergic outcomes.

Necrotizing enterocolitis prevention

Prophylactic use of certain strains of *Lacticaseibacillus* greater than or equal to 30 days remains supported for reducing necrotizing enterocolitis and mortality, provided that product quality, strain identity and clinical surveillance are ensured in preterm infants [1]. The benefit appears to be strain-dependent, underlining the need for standardized formulations and microbiological traceability in neonatal care. Recent evidence highlights how early microbial exposure—through vaginal delivery, breastfeeding, and dietary diversification—modulates mucosal immunity and oral tolerance. The concept of the "first 1,000 days" remains a crucial window for interventions aiming to prevent allergic and inflammatory diseases. Growing data on postbiotics, non-viable microbial products with signaling and anti-inflammatory potential, are opening new therapeutic perspectives in allergy prevention and immune education.


Photo: Shutterstock.


Photo: Shutterstock.

Sources

• **1**. Szajewska H, Berni Canani R, Domellöf M, *et al.*; ESPGHAN Special Interest Group on Gut Microbiota and Modifications. Probiotics for the Management of Pediatric Gastrointestinal Disorders: Position Paper of the ESPGHAN Special Interest Group on Gut Microbiota and Modifications. *J Pediatr Gastroenterol Nutr* 2023; 76: 232-47. • **2**. Cruchet S, Gutiérrez-Castrellón P, Vázquez-Frías R, and LASPGHAN WG on Biotics. Guía de Práctica Clínica Ibero-Latinoamericana del uso de Bióticos en Gastroenterología, Hepatología y Nutrición Pediátrica: Capítulo Probióticos. 2025 (submitted). • **3**. Szajewska H, Kołodziej M, Gieruszczak-Białek D, Skórka A, Ruszczyński M, Shamir R. Systematic review with meta-analysis: Lactobacillus rhamnosus GG for treating acute gastroenteritis in children – a 2019 update. *Aliment Pharmacol Ther* 2019; 49: 1376-84. • **4**. Czerucka D, Rampal P. Diversity of Saccharomyces boulardii CNCM I-745 mechanisms of action against intestinal infections. World J Gastroenterol 2019; 25: 2188-203. • **5**. de Vos WM, Tilg H, Van Hul M, Cani PD. Gut microbiome and health: mechanistic insights. *Gut* 2022; 71: 1020-32.

Photo: Shutterstock.

HOW TO TALK ABOUT

By Prof. Harry Sokol

Expert advice on communicating with patients

The 'How to talk about' page is a dedicated resource designed to help healthcare professionals respond clearly and confidently to patients' questions on topics related to the microbiota.

Covering key topics such as gut health and women's health, it offers short educational videos in which recognised experts share structured communication approaches and suggest clear and appropriate wording to answer the most frequently asked questions during consultations.

In this issue, Professor Harry Sokol focuses on how to address gut health in daily practice.

He explains how to describe the gut microbiota, clarify its role in digestion and overall health, respond to common misconceptions, and guide patients on dietary and lifestyle factors that promote microbiota balance, while maintaining scientific rigour and realistic expectations.

From one expert to another, discover practical information to enrich your consultations and deliver informed, evidence-based messages about the microbiota in your daily practice.

For a successful consultation:

01

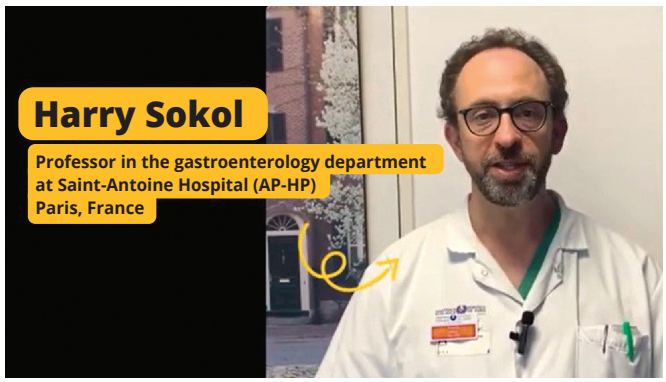
Avoid medical jargon as much as possible

02

Use everyday life examples that can directly resonate with the patient

03

Use illustrations to simplify the explanations of complex messages



For more information

Photo: Shutterstock.

By Anaïs Bodon, Muriel Mercier-Bonin, Bruno Sovran

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PFAS and microbiota

Per- and polyfluoroalkyl substances (PFAS) are a large family of chemical compounds commonly referred to as “forever chemicals” due to their extreme persistence in the environment and ecosystems.
Their fire-retardant, water-repellent, and heat-resistant properties have led to their widespread use in many everyday products (nonstick cookware, waterproof textiles, etc.). PFAS are highly resistant to degradation, accumulate in the environment, contaminate the food chain, and lead to widespread human exposure. PFAS thus accumulate in the body with documented health effects, albeit for a limited number of compounds.

> Are the claims made on social media justified?

In the laboratory, under very specific experimental conditions, certain bacteria in the gut microbiota [1] or lactic acid bacteria [2] (often used as probiotics) can bioaccumulate PFAS or “sequester” them on their surface, suggesting a possible “detoxification effect.” In practice, however, ingested PFAS are almost entirely absorbed in the small intestine and rapidly enter the bloodstream. The gut microbiota is mainly located in the colon, a compartment that is only minimally exposed to PFAS, although a small fraction of the microbiota, located in the small intestine, is directly exposed. As a result, direct contact between PFAS and intestinal bacteria remains marginal compared with the total amount of PFAS already accumulated in the body. Based on current knowledge, it is therefore difficult to conclude that the microbiota and/or probiotic intake play a major role, contrary to what is suggested in the media, in the microbial detoxification of total PFAS from the body.

> What are the impacts on the microbiota?

Furthermore, this limited contact may nevertheless contribute to the increasingly documented effects of PFAS on the gut microbiota. Studies in rodents show that PFAS exposure can alter the composition of the microbiota and disrupt some of its functions, including metabolites production and interactions with the immune system [3,4]. These alterations have been shown to disrupt the balance of the digestive ecosystem and impair intestinal health.

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> Are there any important limitations to keep in mind?

Most of the available scientific data comes from animal models exposed to doses generally higher than those representative of human exposure, and concerns a very limited number of PFAS, often studied individually. In humans, studies remain rare, mainly observational, and influenced by many confounding factors (*e.g.* diet, age, sex). Research on PFAS-microbiota interactions is therefore still emerging.

> What are the implications for healthcare professionals and how should they respond to patients?

Given the media coverage of the topic and patients’ concerns, it is essential to communicate on the basis of scientific evidence, without excessive extrapolation. It should be explained that there is no “miracle” solution for PFAS detoxification, particularly through probiotics.

In practice, healthcare professionals can advise limiting known sources of PFAS exposure (*e.g.* food packaging, non-stick cookware) and encouraging a varied, minimally processed diet rich in fruits, vegetables, and whole grains to support the protective function of the microbiota.

Sources

• **1**. Lindell AE, Grießhammer A, Michaelis L, *et al.* Human gut bacteria bioaccumulate per-and polyfluoroalkyl substances. *Nat Microbiol* 2025 ; 10 : 1630-47. • **2**. Xing J, Wang F, Xu Q, *et al.* Screening of potential probiotic lactic acid bacteria based on gastrointestinal properties and perfluorooctanoate toxicity. *Appl Microbiol Biotechnol* 2016 ; 100 : 6755-66. • **3**. Zhang L, Rimal B, Nichols RG, *et al.* Perfluorooctane sulfonate alters gut microbiota-host metabolic homeostasis in mice. *Toxicology* 2020 ; 431 : 152365. • **4**. Shi L, Pan R, Lin G, *et al.* Lactic acid bacteria alleviate liver damage caused by perfluorooctanoic acid exposure via antioxidant capacity, biosorption capacity and gut microbiota regulation. *Ecotoxicol Environ Saf* 2021 ; 222 : 112515.

By Prof. Satu Pekkala

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

GUT MICROBIOTA

Exercise-induced microbiota metabolite enhances CD8 T cell antitumor immunity promoting immunotherapy efficacy

Sedentary lifestyle increases cancer risk, and exercise is known to enhance immune checkpoint inhibitor (ICI) efficacy. However, the mechanisms have remained largely unknown. Gut microbiota promotes antitumor immunity and exercise modulates gut microbiota, but whether these factors are linked has not been studied. Phelps *et al.* used preclinical cancer models to explore possible connections.

They found that prolonged exercise limited melanoma tumor growth without affecting body weight. Exercise also boosted CD4 and CD8 T cells in tumor-draining lymph nodes. Importantly, this effect required gut microbiota, which was involved in exercise-induced antitumor activity. To establish causality, the authors performed fecal microbial transplantations (FMTs) into antibiotic-treated mice using feces from exercised and sedentary donors. FMT from exercised mice suppressed tumor growth, prolonged survival, and enhanced tumor immunity. While bacterial cell wall components are known to increase immune responses, the exercise-FMT effect appeared dependent on microbiota-derived metabolites.

Indeed, oral administration of microbial metabolites from exercised mice's feces restrained melanoma tumor growth.

To understand the role of metabolites, the authors used targeted metabolomics of 1-carbon (1C) metabolites and found that mainly precursors of the folate-dependent 1C pathway were diminished in exercised mice. Further experiments revealed that elevated formate levels promoted antitumor immunity and restrained tumor growth, and that exercise specifically increased formate. In addition to melanoma, these effects were observed in adenocarcinoma and lymphoma models. Formate also dramatically reduced lung metastases. Formate's effect on antitumor immunity was mediated *via* nuclear factor erythroid 2-related factor-2. Ultimately, they provide some translational evidence that high-formate-producing human microbiota is associated with enhanced tumor suppression and immunity.

Phelps CM, Willis NB, Duan T, *et al.* Exercise-induced microbiota metabolite enhances CD8 T cell antitumor immunity promoting immunotherapy efficacy. *Cell* 2025; 188: 5680-700.

GUT MICROBIOTA

Temporal dynamics and microbial interactions shaping the gut resistome in early infancy

Antibiotic resistance stems from antibiotic resistance genes (ARGs), which enable bacteria to withstand antibiotics. ARGs existed before human antibiotic use, but modern overuse has amplified their prevalence globally. When resistance reaches pathogenic microbes, it threatens public health by undermining antibiotics. However, more studies, especially in infancy are needed to understand gut resistome's role in spreading antimicrobial resistance (AMR).

This study investigated infant gut resistome dynamics in a birth cohort with longitudinally collected fecal samples 8 times from birth up to five years of age. Early in infancy (3–6 days to 2 months), ARG richness showed a bimodal pattern, which disappeared by 6 months as most infants exhibited high ARG counts. At 12 months, bimodality reappeared, followed by a decline of ARGs at 60 months. ARG abundance relative to total genes was highest in the first 6 months and dropped after 12 months. Absolute ARG abundance varied largely between infants during the first 2 months of life, peaked at 6 months, and then dropped at 12 months.

It was further found that ARGs conferring resistance against tetracyclines, fluoroquinolones, penams, and cephalosporins were the most common and the most abundant until 6 months of age. ARGs against tetracyclines and fluoroquinolones remained the most common across all ages. ARG relative and absolute abundance did not differ between antibiotic-naïve infants and those exposed to antibiotics before the 1st sample at 3–6 days of life. Interestingly, microbial composition and birth mode seem to influence ARG diversity, while only few bacterial taxa have high number of ARGs.

To conclude, this study revealed key temporal patterns and microbial interactions that shape the early-infant gut resistome, suggesting opportunities for targeted strategies to limit AMR during this critical developmental stage.

Chatzigiannidou I, Johansen PL, Dehli RK, *et al.* Temporal dynamics and microbial interactions shaping the gut resistome in early infancy. *Nat Commun* 2025; 16: 8139.

GUT MICROBIOTA

Quantifying the varying harvest of fermentation products from the human gut microbiota

The gut microbiota influences the host largely through the exchange of fermentation products, mainly short-chain fatty acids produced by microbes in the large intestine. The microbes metabolize complex carbohydrates from plant-based foods, as well as dietary proteins that escape digestion in the small intestine. While metabolomics can identify a wide variety of compounds, it gives only snapshots and provides little insight into the overall flux of fermentation products that microbes produce and that host's body takes up. To overcome this limit, this study established orthogonal approaches to quantify this flux, integrating data on bacterial metabolism, digestive physiology, and metagenomics.

This framework allowed creating many important findings. For instance, the bulk of carbon in microbiota-available carbohydrates, 90%, ended up in fermentation products, which were mostly taken up by the host. Variation in diet largely determined the total yield of fermentation products. Low yields may occur when diets are rich in highly processed foods lacking complex carbohydrates or when they include carbohydrates that resist digestion and pass through the gut unchanged. Somewhat surprisingly, the microbes themselves had less impact on the total daily fermentation product harvest, excluding some specific fermentation products, such as butyrate and lactate.

While not being the main aim of the study, it was found that mice harvest far more fermentation products per body weight than humans (≈400 mmol/kg/day vs. 7 mmol/kg/day), contributing over 21% of their daily energy needs compared to 1.7–12.1% in humans. Combined with differences in microbiome composition and gut anatomy, this disparity must be considered when extrapolating mouse data to human systemic effects.


Photo: Shutterstock.

The authors conclude that due to dynamic, flowing environment of the intestine, developing this type of framework is critical to move away from assumptions based on point measurements of metabolites toward an integrated model of host-microbiome functions in health and disease.

Arnoldini M, Sharma R, Moresi C, *et al.* Quantifying the varying harvest of fermentation products from the human gut microbiota. Cell 2025 ; 188 : 5332-42.

VAGINAL MICROBIOTA

Gut–mind–pelvic axis: New insights from microbiome science

Beyond surgery, hormones and tumor staging, women with endometrial cancer often experience persistent symptoms affecting mental health, gastrointestinal comfort and sexual well-being. A recent study from the University of Oklahoma suggests that the gut and vaginal microbiota may contribute to these quality-of-life outcomes.

Researchers followed 140 women scheduled for hysterectomy, including patients with endometrial cancer and women with benign gynecological conditions. Before surgery, participants completed validated questionnaires assessing physical and mental health, stress, gastrointestinal symptoms and sexual function. Vaginal and rectal samples were collected for microbiome analysis, enabling correlations between microbial profiles and patient-reported outcomes.

Women with endometrial cancer showed higher vaginal microbial diversity, a pattern usually considered unfavorable in other clinical contexts. Greater diversity was associated with increased vaginal dryness and irritation. Several bacterial species, including *Lactobacillus iners*, *Lactobacillus gasseri* and *Streptococcus agalactiae*, were more frequent in women reporting worse vaginal symptoms, suggesting that oncological conditions may alter vaginal ecosystem dynamics.

The gut microbiota also displayed meaningful associations. In endometrial cancer patients, certain bacterial taxa correlated with better mental well-being, lower stress levels and fewer gastrointestinal complaints, while others were linked to bloating, discomfort or reduced sexual interest. These findings reinforce the concept of a gut–mind–pelvic axis connecting microbial ecosystems with psychological and pelvic health.

From a clinical perspective, this work opens new opportunities for precision microbiome interventions, from targeted probiotics to dietary strategies in supportive cancer care. Microbiome profiling could help identify patients at risk of persistent symptoms and guide personalized interventions, including dietary strategies, lifestyle interventions or targeted microbiota modulation, with the aim of improving quality of life alongside standard oncological treatments.

Gautam NJ, Jimenez NR, Łaniewski P, *et al.* Microbiome impacts quality of life in patients with endometrial cancer and benign gynecological conditions. *Qual Life Res* 2025 ; 34: 2935-48.


Photo: Shutterstock.

Antimicrobial resistance: A digital fresco to learn, discuss and act

Antimicrobial resistance (AMR) is one of the top global public health and development threats. To address this global issue, the World Antimicrobial Awareness Week (WAAW), coordinated every year by the World Health Organization from 18 to 24 November, aims to move from awareness to action by promoting responsible antimicrobial use across all sectors.

In line with this objective, the Biocodex Microbiota Institute is committed alongside the WHO by supporting the WAAW 2025 and launching the Antimicrobial Resistance Fresco. This initiative represents the first educational fresco dedicated to AMR, designed for healthcare professionals, and freely accessible in a digital and downloadable format.

This pedagogical tool highlights the links between antimicrobial use, microbiota disruption and the spread of resistance, and explores the consequences for patients and healthcare systems. By encouraging collective reflection and dialogue, it aims to support a better understanding of AMR and promote concrete actions toward improved antimicrobial stewardship.

As a healthcare professional, you can use this fresco as a practical tool to inform, engage and mobilize your teams, patients and communities, and actively contribute to improving antimicrobial use and preserving their effectiveness.

Download >

*If nothing changes, AMR could become responsible for almost 10 million deaths worldwide by 2050. (World Health Organization)

BIOCODEX MICROBIOTA FOUNDATION

Henri Boulard Awards 2025...

In 2025, the Henri Boulard Awards continue to support innovative research projects addressing global public health challenges through microbiota science. Across three thematic areas, the Awards highlight initiatives responding to local unmet medical needs while contributing to global knowledge.

and the winners are:

> Microbiota & Human Health

• **Dr. Livia Hecke Morais** – Brazil
Uncovering microbiome pathways linking Toxoplasmosis and schizophrenia care in Brazil

• **Dr. Fernando Chirdo** – Argentina
Assessing the therapeutic potential of *Saccharomyces boulardii* in the management of chronic diarrhea in pediatric patients

> Microbiota & Human Health

• **Dr. Solayide Adesida** – Nigeria
Community profiling of the lung microbiome and its role in disease modification of tuberculosis patients in Lagos

> Microbiota & Environmental Concerns

• **Dr. Jennifer Osogom Clifford-Nkemdilim** – Nigeria
Reequiping the gut microbiota through drinking water. A community and school-based water purification through the use and distribution of ceramic water pot filters

www.biocodexmicrobiotafoundation.com