MICROBIOTA Mag

| 23 | OCTOBER 2025

| OVERVIEW |

Drugs & microbes: a bidirectional dialogue

BIOCODEX Microbiota Institute

SUMMARY

WHAT DID YOU MISS ON SOCIAL MEDIA?

THE TOP TIPS TO STAY SUPER HEALTHY

By **Eric Berg - My Best Tips of All Time**
1,2M de vues,
50k likes,
2k comments

MY HEALTH, MY MICROBIOTA

In July, the Biocodex Microbiota Institute's Facebook post "Microplastics in takeaway packaging could harm your gut microbiota" attracted the most shares, comments and reactions.
37k followers, 37k views,
7k engagements

CARB PROVEN TO PREVENT COLON CANCER, UNCLOG ARTERIES & END CHRONIC INFLAMMATION!

A Florida chiroprator reminds us how soluble fiber, a prebiotic carb, fuels gut microbes to produce anti-inflammatory compounds that protect the colon and heart.

By **Dr.Alan H. Mandell - Motivational Doc**
225,139 views, 14,925 engagements

EDITO

Dr. Maxime Prost, MD
France Medical Affairs Director

Barbara Postal, PhD
Head of Global Medical Affairs Microbiota & Mature Products

Dear readers,

When drugs meet microbes: a dialogue too long overlooked

In recent years, we have become accustomed to thinking of the gut microbiota as a cornerstone of digestive and immune health, a complex organ in itself. But what happens when it meets another key player in modern medicine, drugs? This is the fascinating focus of our latest issue, expertly synthesized by Prof. Emmanuel Montassier (University of Nantes, France).

We are learning that the dialogue between gut microbes and medications is not only real, it is profoundly bidirectional. Drugs can reshape the microbiota, sometimes with long-term consequences. In turn, microbes can metabolize, activate, or inactivate medications, influencing both their efficacy and toxicity. According to studies referenced in this issue, around **24% of non-antibiotic drugs inhibit at least one commensal species**, and **10–15% are metabolized by the microbiota**, with possible clinical implications ranging from reduced therapeutic benefit to adverse drug reactions.

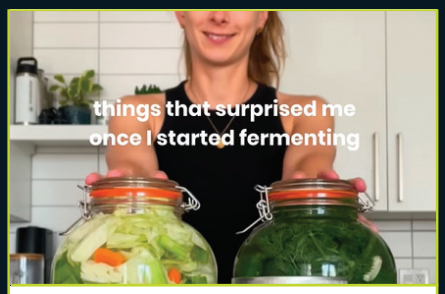
These interactions, still largely overlooked in drug development and prescribing, are now forming the foundation of a new field: **pharmacomicrobiomics**. By combining microbiome data with genomics and clinical information, we are on the verge of personalizing treatments in ways previously unimaginable.

In this edition, Prof. Montassier takes us through key findings, including the collateral damage of common antibiotics on gut flora, the underestimated effects of drugs like PPIs and metformin on microbial communities, and emerging strategies to preserve and restore the microbiota, ranging from microbial enzyme inhibitors to microbiota-sparing drugs and even AI-guided treatment design.

By bringing clarity to this fast-moving field, we hope to foster a broader awareness of the gut microbiota not just as a passive victim of medications, but as a **therapeutic actor in its own right**.

Enjoy your reading!

We are learning that the dialogue between gut microbes and medications is not only real, it is profoundly bidirectional.



FERMENTED FOODS

The post, titled “5 things that surprised me once I started fermenting” became the standout post of the month, sharing unexpected benefits of fermented foods, from smoother digestion to a brighter mood.

By **Kirsten Kaminski, the tasty k**
Video viewed over 406,000 times, over 10K reactions and 1,336 shares

GUT MICROBIOME & WEIGHT LOSS

A Bluesky post from The Conversation U.S. explains that food choices and the gut microbiome activate a natural GLP-1-like pathway for weight control, echoing the action of Wegovy, Ozempic and Mounjaro.

Reach : 50 000 +

EXPLANATION BY DR. LOPEZ ROSETTI ON THE MICROBIOME, MOOD & GLOBAL HEALTH

By **Dr. Daniel López Rosetti**
2,2M followers, 22k likes for 461k views

By Prof. Emmanuel Montassier

Emergency Department, CHU Nantes; Inserm, Center for Research in Transplantation and Translational Immunology, UMR 1064, Nantes Université, Nantes, France

When drugs meet microbes: a bidirectional dialogue with therapeutic implications

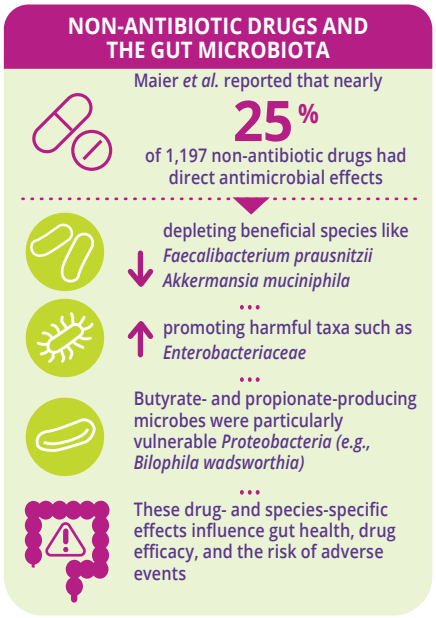
Bidirectional interactions between oral drugs and the gut microbiome are increasingly seen as crucial to drug efficacy, safety, and tolerability. While antibiotics are known to disrupt microbial communities, about 24% of non-antibiotic drugs also inhibit at least one commensal species. Additionally, 10–15% of oral drugs are transformed by gut microbes *in vivo*, affecting their effectiveness or toxicity. Common medications—such as proton pump inhibitors (PPI), nonsteroidal anti-inflammatory drugs (NSAIDs), metformin, and statins—can alter microbiota composition and function, influencing host metabolism and immunity. Despite these findings, the microbiome is often overlooked in prescribing and in drug development. This review summarizes key clinical and mechanistic insights, highlights notable drug–microbiota interaction, and explores emerging strategies to enhance outcomes. Integrating pharmacomicrobiomics into clinical care may reduce adverse effects and support precision medicine.

The gut microbiota acts as a metabolic organ, supporting digestion, immunity, and homeostasis [1]. Its interaction with drugs, however, is bidirectional: medications can disrupt microbial balance, while microbes can alter drug activity. This makes the microbiome a significant yet often overlooked factor in adverse drug reaction (ADR) risk [2, 3]. Gut microbial enzymes can transform drugs into more toxic forms, increasing tissue exposure and harmful effects. Growing evidence highlights microbial variability as a key driver of individual differences in drug response and ADRs [2, 4]. Integrating pharmacomicrobiomics into risk assessment—alongside genetics and clinical data—could help predict susceptibility to drug-related harm and guide personalized prevention strategies.

Drug-induced microbiota disruption: antibiotics and beyond

Antibiotics are well known to disrupt the gut microbiota by reducing diversity, altering composition, and promoting resistant strains (**table 1**) [5, 6]. Van Zyl *et al.* found that antibiotics—especially quinolones and β-lactams—consistently disrupt microbial communities across body sites, with combination regimens causing prolonged dysbiosis and increased pathogenic burden [5]. Similarly, Maier *et al.* showed that different antibiotic classes have distinct effects on gut bacteria, with macrolides and tetracyclines causing sustained losses in anaerobes, and drugs like amoxicillin and ceftriaxone shifting populations toward Proteobacteria. Despite individual variability, a common trend emerged: depletion of obligate anaerobes (*e.g.*, Firmicutes) and enrichment of facultative and potentially pathogenic microorganisms [6].

Beyond antibiotics, many non-antibiotic drugs—including PPIs, metformin, NSAIDs, antipsychotics, and statins—also alter the gut microbiota (**figure 1, table 2**) [7, 8]. Drugs influence the gut microbiota through various mechanisms—direct antimicrobial action, altered pH, bile acid modulation, intestinal motility changes, and mucus secretion [9].



Up to 25% of commonly prescribed drugs have measurable antimicrobial activity.

Gut microbiota modifies drugs metabolism

The gut microbiota can biotransform therapeutic drugs, altering their activity, efficacy, and toxicity (**figure 2, table 3**) [12-14]. Zimmermann *et al.* mapped microbial metabolism by screening 271 oral drugs against 76 gut bacterial strains, finding

TABLE 1 • COMMON GUT BACTERIA AFFECTED BY ANTIBIOTICS

BACTERIA	IMPACT ON THE GUT MICROBIOTA
Commonly decreased by antibiotics	
Bifidobacterium spp.	• Frequently reduced by β-lactams, macrolides, and fluoroquinolones. • Bifidobacteria play a key role in carbohydrate fermentation and gut barrier protection.
Faecalibacterium prausnitzii	• A major butyrate-producing bacterium with anti-inflammatory properties, often depleted by broad-spectrum antibiotics such as ciprofloxacin, β-lactams and clindamycin.
Akkermansia muciniphila	• A mucin-degrading bacterium involved in maintaining the gut barrier, sensitive to amoxicillin and metronidazole.
Roseburia spp. and *Ruminococcus* spp.	• Important butyrate producers often reduced after macrolides and amoxicillin-clavulanate.
Increased by antibiotics (opportunistic expansion)	
Enterococcus faecalis and *Enterococcus faecium*	• Frequently enriched following vancomycin, cephalosporins, and carbapenem use. • May contribute to resistance gene reservoirs.
Escherichia coli	• Some strains may expand post-treatment due to reduced competition, particularly after third generation cephalosporins.
Proteobacteria (*e.g., Klebsiella* spp., *Citrobacter* spp.)	• Often increase in relative abundance postantibiotics (β-lactams, cephalosporins, carbapenem, clindamycin). • Potentially promoting dysbiosis and inflammation.
Clostridioides difficile	• Although not a commensal, it flourishes in the wake of microbiota collapse—especially after clindamycin, cephalosporins, and fluoroquinolones.

that 176 were metabolized by at least one strain. Notably, *Bacteroides dorei* and *B. uniformis* metabolized nearly 100 drugs. Over 40 microbial enzymes were identified, mediating a wide range of reactions—including reduction, hydrolysis, decarboxylation, dealkylation, and demethylation [12].

Javdan *et al.* developed a personalized platform (MDM-Screen) to assess microbial drug metabolism using *ex vivo* microbiota from individual donors. Screening 575 drugs, they found that 13% were metabolized by gut microbes, including many previously unrecognized interactions. These transformations—such as hydrolysis, reduction, and deacetylation—can activate, inactivate, or increase drug toxicity. The study also revealed significant inter-individual variability and identified key microbial genes (*e.g.*, uridine phosphorylase, β-glucuronidase) linked to specific metabolic pathways [15].

The efficacy of some drugs may depend more on the microbiota composition than on the host genetics.

Clinical consequences: toward personalized medicine

Microbiota-drug interactions have major clinical implications, as individual differences in gut microbiota may explain variability in drug response and side effects. Importantly, it is not just microbiota composition but also its functional stability that influences treatment outcomes.

In advanced melanoma, patients responding well to anti-PD-1 therapy showed stable microbial functions and CD8+ T cells reactive to bacterial peptides from *Lachnospiraceae*, which mimic tumor antigens—highlighting microbial functionality as a potential prognostic marker and therapeutic adjunct in cancer immunotherapy [16].

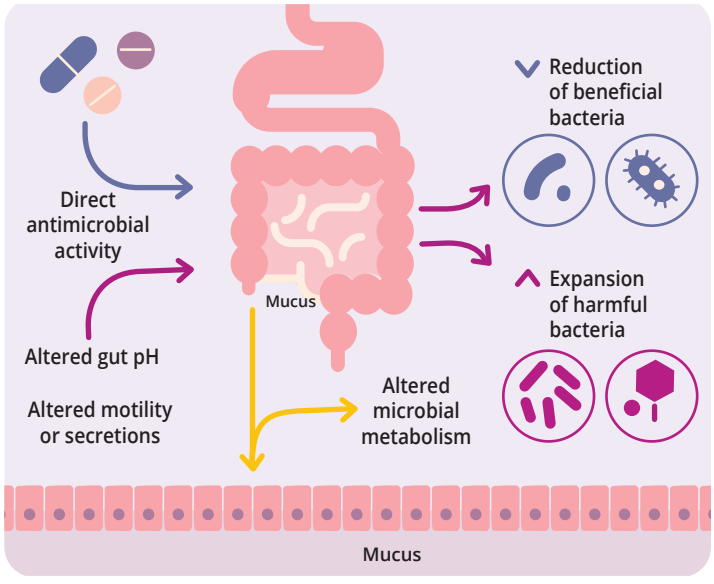


FIGURE • 1


Photo: Shutterstock.

These insights underscore the need to integrate both human and microbial genomics into pharmacological assessments. In drug development, simulating microbiota–drug interactions *in silico* has become key. Dodd and Cane proposed a detailed framework combining *in vitro* sys-

TABLE 2 • COMMON GUT BACTERIA AFFECTED BY NON-ANTIBIOTIC TREATMENTS

Drug class	Example drug	Microbiota impact	Affected specific microbial groups
Antidiabetics	Acarbose	↑ *Lactobacillus*, ↓ *Bacteroides*; modulates SCFA levels.	*Lactobacillus, Bacteroides*
Immuno-suppressants	Tacrolimus	Microbial degradation affects absorption and efficacy.	*Clostridium symbiosum, Eggerthella*
Antidepressants	Sertraline	Antibacterial activity; impacts diversity and metabolic outputs.	↓ Overall diversity, ↑ Firmicutes
Chemotherapy	Irinotecan	Microbial β-glucuronidase reactivates toxic metabolites.	↑ β-glucuronidase expressing bacteria
Antihypertensives	Amlodipine	Alters Firmicutes/Bacteroidetes ratio.	↑ Firmicutes / ↓ Bacteroidetes
Proton Pump Inhibitors (PPIs)	Omeprazole	Reduced diversity, ↑ oral-origin taxa, ↑ *Enterobacteriaceae*, Direct bacteriostatic effect, reduction in butyrate producers and amino-acid synthesis pathways.	*Rothia, Haemophulis, Veillonella parvula, Streptococcus salivarius, S. vestibularis, Rothia dentocariosa, Actinomyces, Lactobacillus spp., Enterococcus faecalis*
Biguanides	Metformin	↑ SCFA producers, ↑ *Akkermansia*, alters bile acid metabolism.	*Enterobacteriaceae, Escherichia coli, Shigella, Citrbacter, Streptococcus mutans, Akkermansia muciniphila, SCFA producing bacteria* (*e.g., Blautia, Butyrivibrio*)
NSAIDs	Ibuprofen	Barrier disruption, ↑ inflammatory taxa. Metabolism influenced by the gut microbiota. Cytotoxicity in association with PPI.	*Escherichia coli,* ↓ *Faecalibacterium, Prevotella*
Statins	Atorvastatin	Modifies microbial metabolism, ↑ bile acid-transforming bacteria, ↓ adipose tissue inflammation, ↑ SCFA producing bacteria.	*Bacteroides, Clostridium*
Antipsychotics	Risperidone	Reduced diversity, metabolic shift towards energy extraction.	↑ *Firmicutes* / ↓ *Bacteroidetes* ratio
GLP-1 receptor agonists	Liraglutide	Alters gut barrier, ↑ SCFA, ↓ pathogens.	↑ *Akkermansia*, ↓ *Desulfovibrio*
Antiepileptics	Valproic acid	↑ diversity, ↓ pro-inflammatory bacteria.	↑ *Bifidobacterium*, ↓ *Bacteroides*
Antifungals	Fluconazole	↓ fungal diversity, ↑ Proteobacteria *via* cross-kingdom effect.	↓ *Candida*, ↑ *Proteobacteria*
SGLT2 Inhibitors	Dapagliflozin	Alters glucose fermentation profile, ↑ SCFA producers.	↑ *Butyrivibrio, Lactobacillus*

tems (*e.g.*, strain libraries, stool-derived communities), genetic tools (gain/loss-of-function assays), and metagenomics to identify microbial genes involved in drug metabolism. Gnotobiotic mouse models further help disentangle microbial from host effects on pharmacokinetics.

As this field advances, microbiota-informed prescribing is emerging as a way to tailor treatments and reduce adverse effects. In the future, pharmacomicrobiomics could guide drug choices and dosages based on microbial biomarkers, enabling truly personalized medicine [17].

Personalizing treatment could one day require a microbiota fingerprint.

Preserving and restoring the microbiota: a therapeutic frontier

Protecting the gut microbiota during drug therapy is a promising strategy to reduce ADRs and preserve efficacy. While probiotics and prebiotics show some benefit against drug-induced dysbiosis, their effectiveness varies. Targeted probiotics tailored to specific drug effects, and fecal microbiota transplantation (FMT), particularly for recurrent *C. difficile* infection, offer more reliable options.

Precision tools such as microbial enzyme inhibitors (*e.g.*, β-glucuronidase blockers for irinotecan toxicity), bioengineered probiotics, microbiota-sparing drug designs, and diet-based interventions are under investigation. Clinical trials are exploring synbiotics customized to drug regimens to improve outcomes with minimal microbiota disruption. Postbiotics like butyrate are also being evaluated for anti-inflammatory and gut barrier-supporting effects.

Integrating microbiota-targeted strategies into pharmacology will require advanced tools—multi-omics, machine learning, and systems microbiome modeling—to predict and manage microbiota–drug interactions effectively.

Manipulating the gut microbiota may enhance treatment success and reduce complications.

FIGURE • 2

Gut microbiota effect on drug metabolism

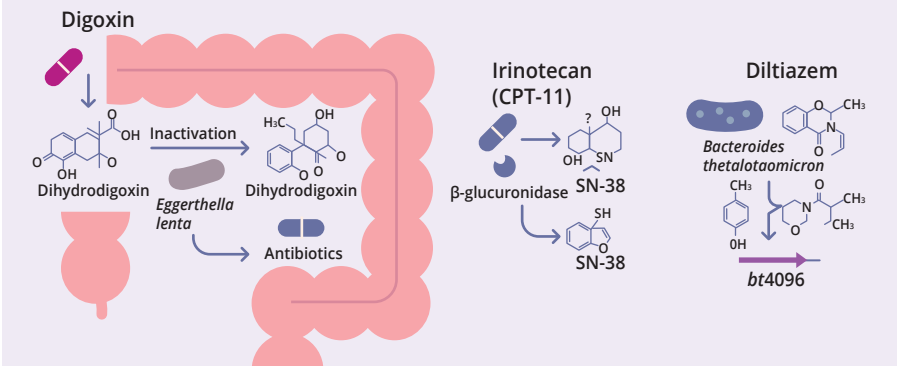


TABLE 3 • IMPACT OF THE MICROBIOME ON THE METABOLISM OF COMMON DRUGS

Drug	Microbial	Mechanism / enzymes influence	Consequence involved
Digoxin	Inactivation by *Eggerthella lenta*	Cardiac glycoside reductase (Cgr operon)	Reduced efficacy
Irinotecan	Reactivation of SN-38 in colon	Microbial β-glucuronidase	Intestinal toxicity (diarrhea)
Levodopa	Premature decarboxylation	Tyrosine decarboxylases from *Enterococcus*	Reduced bioavailability
Sulfasalazine	Activation via azoreduction	Azoreductase from anaerobic bacteria	Therapeutic activation in colon
Balsalazide	Prodrug activation in colon	Azoreductase from anaerobic bacteria	Local anti-inflammatory effect
Tacrolimus	Reduced absorption via microbial metabolism	Unknown reductive pathways	Decreased immunosuppressant efficacy
Metformin	Altered bioavailability and hepatic uptake	Microbiota-mediated SCFA profile shift	Improved glucose control
Lovastatin	Hydrolysis of lactone ring	Esterase-producing gut microbes	Altered systemic exposure
Diltiazem	Reduced absorption via microbial metabolism	Reductive transformation by Bacteroides	Altered drug efficacy
Acarbose	Hydrolysis by microbial enzymes	Glycoside hydrolases	Impaired glucose-lowering effect
Duloxetine	Microbial demethylation and oxidation	Oxidative and demethylating enzymes	Alteration in antidepressant effect

Conclusion

Microbiota–drug interactions are an emerging and often overlooked aspect of medicine with major implications for treatment outcomes. Integrating these insights into clinical practice is key to developing safer, more precise, and microbiota-aware therapies. As evidence grows, new opportunities arise to modulate the microbiome to boost efficacy, reduce toxicity, and rescue drug responses.

Innovative approaches—such as live biotherapeutics, engineered microbes, and microbiota-derived metabolites ("pharmabiotics")—are reshaping pharmacotherapy. Although regulatory interest is increasing, standardized clinical protocols are still developing. In the near future, microbiome engineering could become a routine component of personalized, systems-based medical care.


Photo: Shutterstock

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COMMENTED ARTICLE

Adults' section

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By Prof. Harry Sokol

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Towards a health-associated core keystone (key species) index for the human gut microbiota

Comments on the original article by Goel et al.*, Cell Reports 2025 [1]*

A robust index of gut microbiome taxa, encompassing their association with host health and microbiome resilience, would be valuable for the development and optimisation of microbiome-based therapeutics. In this article the authors present a single ranked order for 201 taxa, the Health-Associated Core Keystone (HACK) index, derived using their prevalence/community association in non-diseased subjects, their temporal stability and their association with host health. This index was constructed using 127 discovery cohorts and 14 validation datasets (a cumulative total of 45,424 gut microbiomes from subjects aged over 18 years, representing 42 countries, 28 disease categories and 10,021 longitudinal samples). The authors show that this index is reproducible regardless of microbiome profiling strategies and cohort lifestyle. Specific consortia of high HACK index taxa respond positively to Mediterranean diet-based interventions, are associated with better immune checkpoint inhibitor responsiveness and display specific functional profiles at the genome-level. The availability of HACK indices thus provides a rational basis for comparing microbiomes and facilitating the selection and design of microbiome-based therapies.

What do we already know about this subject?

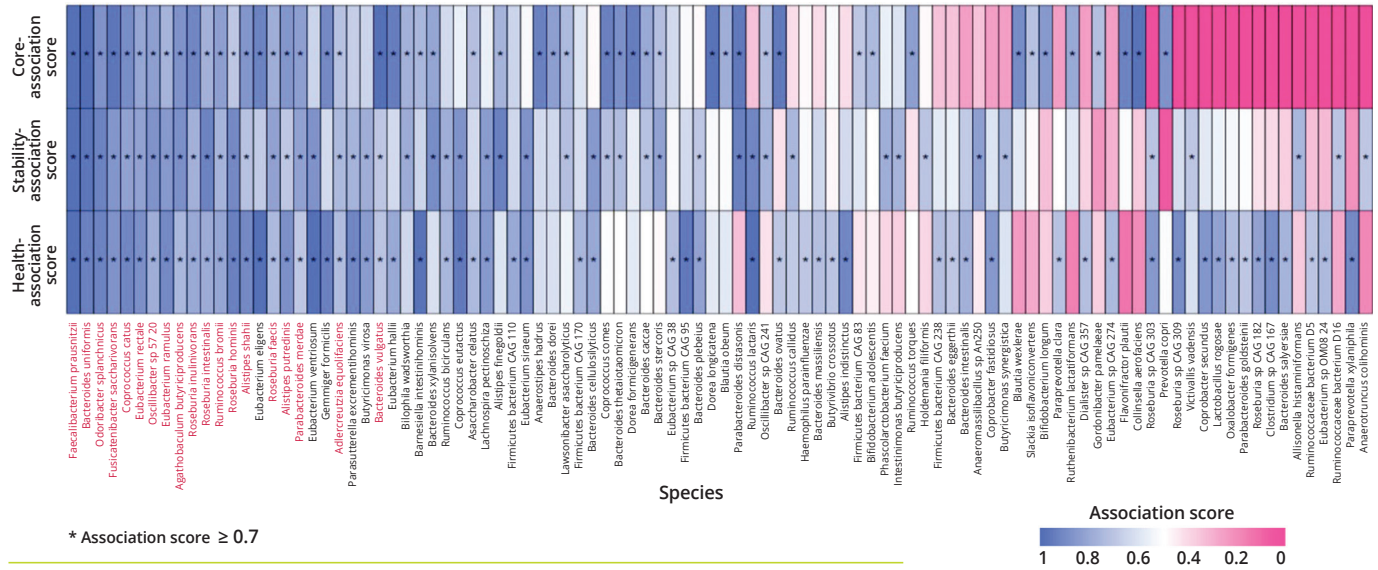
••• Gut microbiome-based therapeutics (including probiotics, live biotherapeutic products, prebiotics/synbiotics and faecal transplantation) aim to restore a healthy microbiota, but with varying degrees of success depending on the population. To optimise these approaches, a consensual definition of a "healthy" microbiome would be needed - a challenging task due to the high degree of interindividual variability. However, meta-analyses reveal taxa that are consistently depleted or enriched across multiple diseases, suggesting that microbes can be positioned along a spectrum of association with host health [2, 3]. High-ranking species on this scale would have the greatest potential: i) as direct therapeutic agents or targets for enrichment; ii) as markers of clinical efficacy. The authors therefore propose creating a priority index integrating three criteria: positive association with health, contribution to microbiota stability and strong community "interaction". This index, which can be applied to large public datasets, would serve as a rational tool for selecting and evaluating future microbial therapeutic strategies.

What are the main insights from this study?

••• Using a discovery cohort comprising 39,926 gut microbiomes from 127 cohorts (including cross-sectional and longitudinal data, spanning 42 countries and 28 different diseases), the authors generated a ranking of 201 prevalent (core) gut microbiota taxa (those detected in ≥ 5% of samples in ≥ 50% of the studied cohorts), the HACK index (Health-Associated Core Keystone Index), each being assigned a score based on three quantifiable properties: i) prevalence/community association in non-diseased subjects; ii) temporal stability; and iii) negative association with disease.

The HACK index was calculated as the product of two scores: i) the mean of the association scores of a taxon for all the three properties; and ii) a reward score assessing the similarity (or how evenly distributed) these three scores were with respect to each other. Analysis of the highest-ranked taxa based on this order revealed 17 taxa having a HACK index of ≥ 75% (**figure 1**). These taxa all had individual scores of ≥ 70% for the three properties. These included *Faecalibacterium prausnitzii*, a well-recognised marker of microbiome health [4], followed by *Bacteroides uniformis*. The list also features

FIGURE 1 **Profile of 98 taxons and their association scores across the three properties.**
Scores ≥ 70% are indicated with an asterisk (*). Taxons having scores of ≥ 70% for all three properties are displayed in red.



several species from the genera *Roseburia, Alistipes*, and *Eubacterium*, as well as *Coprococcus catus*.

The authors then demonstrated the reproducibility of both the individual scores and the overall HACK index by recalculating the association scores within each cohort separately, using different sequencing methods (Shotgun or 16S) and across different type of populations (industrialised urban versus other), followed by an additional validation dataset composed of 14 additional cohorts totalling 5,498 microbiomes.

Beyond their stronger association with health and microbiota stability, some taxa with a high HACK index were also associated with favourable responses to various microbiota-related interventions, such as the Mediterranean diet or anti-cancer immunotherapy.

By analysing genome-level functional annotations from 32,005 genomes representing 122 of the 201 taxa, the authors identified 150 functional features (tags or fragments) specifically enriched and conserved in the genomes of taxa having high HACK indices. These represent a wide range of functions: production of butyrate/propionate with anti-inflammatory properties, synthesis of numerous vitamins, biosynthesis of neuroactive amino acids like tryptophan, and their beneficial anti-inflammatory derivatives such as indoles, or chondroitin sulphates. These are functionalities which warrant exploration to understand underlying mechanisms.

What are the consequences in practice?

The HACK indices were calculated from a global cohort of 45,000 gut microbiomes spanning the six major continents, making this one of the most comprehensive studies to date. These indices represent a step forward in the rational prioritisation of gut microbial species as potential candidates for microbiome-based therapeutics. In addition, functionalities associated with high HACK indices may help identify pathways and metabolic capabilities linked to the general health and stability of the microbiome.

[CONCLUSION]

Drawing on a very large database, this study identifies a group of 17 taxa that are particularly prevalent (core taxa), stable over time and associated with health. In addition to progressing towards the definition of key components of the human microbiota in terms of both taxonomy and function, this work provides a rational basis for the development of novel therapies based on the gut microbiota or targeting it.

Key points

- Based on 45,454 microbiomes from 141 cohorts (42 countries and 28 disease groups), this study ranked 201 taxa according to their association with three key traits of host and microbiome health: i) prevalence in non-diseased subjects; ii) temporal stability; and iii) negative association with disease
- Among the 17 bacteria with the highest scores, *Faecalibacterium prausnitzii* and *Bacteroides uniformis* ranked first and second, respectively
- The ranking was reproducible regardless of sequencing method or lifestyle of the cohorts
- The highest-ranked taxa are associated with positive responses to various microbiota-related therapeutic interventions

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By Prof. Emmanuel Mas

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Interconnected pathways link plasma lipids, fecal microbiota and brain activity to cognition related to childhood malnutrition

Comments on the original article by Portlock et al., *Nat Commun [1]*

Malnutrition affects more than 30 million children every year and has profound immediate and long-lasting repercussions. Children who survive often suffer long-lasting neurocognitive sequelae that impact on their school performance and socio-economic status. The mechanisms behind these consequences are poorly understood. Using SHAP models interpreted by multisystem random forest and network analysis, the authors show that moderate acute malnutrition (MAM) is associated with increased stool *Rothia mucilaginosa* and *Streptococcus salivarius* and decreased *Bacteroides fragilis* in a group of one-year-old children in Dhaka, Bangladesh. These changes in the microbiome form interconnected pathways involving reduced plasma levels of odd-chain fatty acids, decreased electroencephalogram gamma and beta power in temporal and frontal brain regions, and reduced vocalization. These results support the hypothesis that prolonged colonization with oral commensal species delays the development of the gut and brain microbiome. Although causal links need to be validated by empirical data, this study provides useful information to improve interventions targeting neurodevelopmental deficits associated with MAM.

What do we already know about this subject

••• Childhood malnutrition is a major public health problem and one of the leading causes of death before the age of five. Moderate acute malnutrition (MAM) is associated with delayed neurocognitive development, but the link remains poorly understood. It is also associated with dysbiosis of the gut microbiota (GM), whose establishment is slowed and marked by enrichment in *Bifidobacterium* and *Escherichia* species. These disturbances in the gut microbiota could have an impact on cerebral development via the gut-brain axis, due to defective nutrient absorption or accumulation of toxic metabolites. This inter-organ communication could be mediated indirectly by plasma lipids, as lipids are the essential constituent of the brain and are modulated by MI metabolites such as bile acids.

What are the main insights from this study?

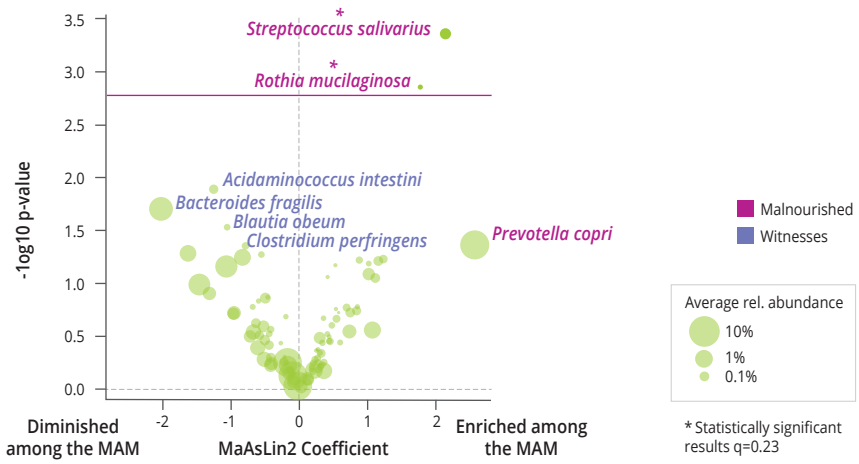
••• The study was carried out in the Mirpur region of Bangladesh, and compared 159 children with MAM with 75 well-nourished controls at 12 months of age. MAM was defined by a weight/height ratio between -2 and -3 z-scores. The MAM group was significantly associated with social-demographic factors (toilet, mode of delivery and water treatment - kettle).

MAM was associated with decreased bacterial alpha diversity (Shannon), increased prevalence and abundance of *Rothia mucilaginosa* and *Streptococcus salivarius* (**figure 1**), and an increased Bacteroidetes/Firmicutes ratio. Functional analyses of the MI showed no differences.

The electroencephalogram (EEG) showed a significant decrease in beta (12-30 Hz) and gamma (30-45 Hz) frequencies in the temporal and frontal regions of children with MAM. Significant decreases in expressive communication, fine and gross motor scores, and vocalization were also observed.

After adjusting for mode of delivery, gender and duration of exclusive breastfeeding, MAM was associated with changes in plasma lipidome, with relative abundance increased by 128 (16%) compounds and decreased by 189 (24%) (**figure 2**).

FIGURE 1 **MAM impacts fecal microbiota at 12 months of age.**
The red horizontal line corresponds to the significance threshold.



Integration of multimodal data showed that the best predictors of MAM at 12 months were: 1) plasma lipids (AUROC = 0.95 0.05); 2) brain and behavioral measures (Wolke score, EEG, Bayley score) (AUROC = 0.73±0.05, 0.71±0.10, 0.68±0.07 respectively) ; 3) the taxonomic, functional and predicted metabolite profile of the fecal microbiome (AUROC = 0.56±0.07, 0.53±0.07, 0.52±0.06). Note the high proportion of data related to the fecal microbiome for predicting MAM in multimodal analysis, despite the poor performance of the fecal microbiome (**figure 3**).

Multimodal network analysis predicted that a cluster of *B. fragilis*, pyruvate fermentation pathways, plasma ceramides, EEG and expressive communication was strongly correlated with good nutritional status at 12 months. Finally, the strongest effect as an interspecies interaction was observed between *R. mucilaginosa* and *S. salivarius*, whose combined presence amplified the prediction of MAM at 12 months.

What are the consequences in practice?

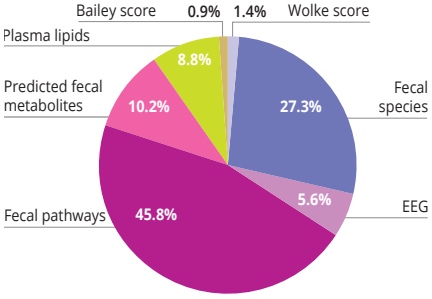
This study shows the importance of GM in the nutritional status of infants. The presence of commensal gram-positive and facultative anaerobic oral bacteria such as *R. mucilaginosa* and *S. salivarius* may be responsible for deregulation of bile acids. This could lead to lipid changes that are important for brain development.

In addition, it is important to highlight the benefit of *B. fragilis* in relation to fermentation pathways on nutritional status at 12 months.

Key point

- Intestinal persistence of commensal bacteria *Rothia mucilaginosa* and *Streptococcus salivarius* in MAM children overrides colonization by *Bacteroides fragilis*. This interferes with the synthesis of fatty acids essential for brain development

FIGURE 3 **Contribution of different data to the multimodal predictive model of MAM.**



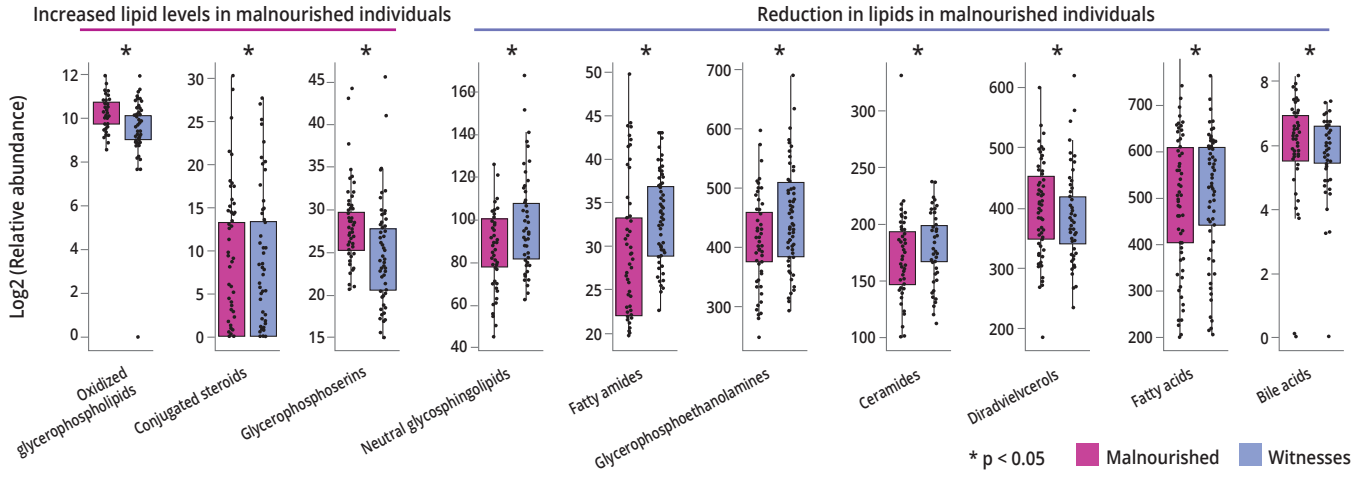
[CONCLUSION]

This study highlights that dysbiosis of the gut microbiota is associated with abnormalities in brain development present in children with MAM, via changes in plasma lipids.

Source

1. Portlock T, Shama T, Kakon SH, *et al.* Interconnected pathways link faecal microbiota plasma lipids and brain activity to childhood malnutrition related cognition. *Nat Commun* 2025 ; 16 : 473.

FIGURE 2 **Differences in plasma lipids at 12 months as a function of children's nutritional status.**

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Can we target microbiota in the management of children with functional abdominal pain disorders?

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She is also engaged in research, contributing to the advancement of diagnostic and therapeutic strategies in pediatric digestive diseases.

> The dysbiotic gut in functional abdominal pain disorders in children

Functional abdominal pain disorders (FAPDs), also referred to as functional gastrointestinal disorders (FGIDs), represent the one of the main etiologies of chronic abdominal pain in the pediatric population that involve interplay among regulatory factors in the enteric and central nervous systems [1]. The ongoing classification system, ROME IV, distinguishes several pain-predominant FGIDs based on their recognizable patterns of symptoms, such as functional dyspepsia (FD), irritable bowel syndrome (IBS), abdominal migraine, and FAP-not otherwise specified (FAP-NOS) [2]. During the past two decades numerous studies researched possible causes and underlying mechanisms of appearance, but the clear pathophysiology is yet to be revealed, despite pediatric neurogastroenterology findings in terms of intestinal motility, signaling molecules, changes in microbiota or epigenetic mechanisms [3]. Gut microbiota modifications, known as a dysbiotic gut, may play a role in functional abdominal pain disorders through gut immunity and integrity alteration [4, 5]. Several studies have reported a lower level of microbial diversity in patients with functional abdominal pain disorders [6, 7] and species such as *Lactobacilli* and *Bifidobacteria* are heavily altered [8]. Thus, a growing body of clinical data have been gathered around using probiotics in functional disorders' management, although study data are lacking on children [9].

> Research insights

The analysis of microbiota in 18 patients with FGIDs provided data about intestinal dysbiosis at the moment of the diagnosis and its changes over a period of three months of treatment with specific strains of probiotics and prebiotics (**figure 1**).

Individuals. Age 4-14 years and diagnosed with functional abdominal pain disorders (functional dyspepsia and irritable bowel syndrome) according to ROME IV criteria.

Intervention. Six bacterial strains (*Lactobacillus rhamnosus* R0011, *Lactobacillus casei* R0215, *Bifidobacterium lactis* BI-04, *Lactobacillus acidophilus* La-14,

FIGURE 1
Modification of microbiota before and after intervention.

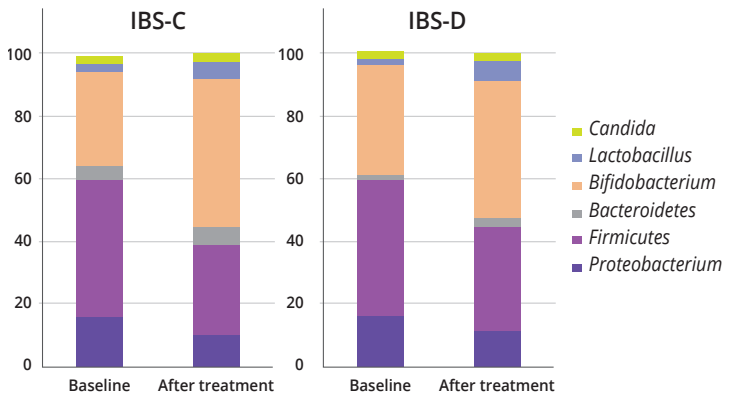

TABLE 1. MICROBIOTA CHANGES FROM BASELINE TO ENDPOINT

Bacteria	IBS-C		IBS-D	
	Mean count ± SD		Mean count ± SD	
	Baseline	Endpoint	Baseline	Endpoint
• *Bifidobacterium*	87.14 ± 55.33 × 10^6	88.85 ± 35.87 × 10^6	71.37 ± 11.21 × 10^6	88.75 ± 43.78 × 10^6
• *Lactobacillus*	35.85 ± 18.12 × 10^4	74.85 ± 29.78 × 10^4	39.25 ± 12.21 × 10^4	55.00 ± 22.89 × 10^4

IBS-D: diarrhea-predominant irritable bowel syndrome, IBS-C: constipation-predominant irritable bowel syndrome, SD: standard deviation.

Bifidobacterium longum BB536, *Lactobacillus plantarum* R1012) and 210 mg of fructo-oligosaccharides-inulin. One capsule was administered orally, daily, for 12 weeks, and the medication was provided by the healthcare practitioners.

Clinical outcome. The patients were scored for severity of abdominal discomfort, dyspepsia, flatulence, and epigastric pain on a ten-point ordinate (numerical rating) scale.

Fecal samples were collected from participants before and after treatment using a special laboratory kit with two sterile containers, which were then brought to the laboratory in conditions depending on the time spent from collection to laboratory delivery: if the interval was less than 24 hours, both containers were stored and transported in cooled conditions at 4 °C; if the period between stool elimination and laboratory delivery was more than 24 hours, one container was stored in a frozen condition at – 80 °C until analysis, and the other one was cooled at 4 °C. Stool samples were analyzed using the test *Colonic dysbiosis-basic profile* (*SBY 1*) performed by Synlab-Germany. Microbiota composition was expressed as number of colony forming units (CFU) for various aerobic/anaerobic bacterial and fungal species. The analysis provided data on fecal pH, IgA in µg/mL (normal ranges 510–2,040 µg/mL), lactoferine µg/mL (normal ranges < 7.2), calprotectin in mg/kg (normal ranges < 50.0 negative, 50–99 intermediary, > 100 positive).

In the fecal microbial analysis, there was an increasing proportion of bacterial genera associated with health benefits (*e.g., Bifidobacterium* and *Lactobacillus*), for both IBS-C and IBS-D (IBS-C: 31.1 ± 16.7% *vs.* 47.7 ± 13.5%, $p = 0.01$; IBS-D: 35.8 ± 16.2% *vs.* 44.1 ± 15.1%, $p = 0.01$). On the other hand, genera of harmful bacteria, including *Escherichia, Clostridium*, and *Klebsiella* were proven to decrease after treatment (21.3 ± 16.9% *vs.* 16.3 ± 9.6%, $p = 0.02$).

No particularities were found in children with FD.

At baseline, before any symbiotic intervention, *Bifidobacterium* profiles were significantly different between IBS-C and IBS-D (87.14 ± 23.19 *vs.* 71.37 ± 12.24; $p = 0.02$), with lower counts in IBS-D. The symbiotic administration had a significant effect on bacterial profiles from baseline to the end of treatment in both IBS-C and IBS-D groups (**Table 1**).

> Practical consequences

The clinical symptoms in study population were more diminished after treatment, with statistical significance, suggesting that influencing gut dysbiosis might also reduce patients' burden and improve clinical scores.

Overall, 14 (78%) patients reported treatment success (defined as no pain). The proportion of patients with adequate symptom relief was higher in the IBS-D than in the IBS-C group; however, the difference was not statistically significant (74.4% *vs.* 61.9%, $p = 0.230$). In both IBS-C and IBS-D groups, scores on the Bristol scale improved significantly after intervention (baseline *vs.* after treatment; 2.8 ± 0.6 *vs.* 3.9 ± 0.9, $p = 0.03$, 6.1 ± 0.9 *vs.* 4.1 ± 1.0, $P = 0.01$, respectively). Abdominal distension and flatulence were significantly improved in both IBS-C and IBS-D groups (IBS-C: 6.5 ± 2.8 *vs.* 3.7 ± 1.8, $p = 0.01$; IBS-D: 5.9 ± 2.2 *vs.* 2.9 ± 1.8, $p = 0.01$).

Key points

- The exploration of human microbiome revealed over time that dysbiosis has a substantial role in pathogenesis of functional abdominal pain disorders, although specific profiles as early biomarkers are still far from current practical use.
- There is a real need for future unitary studies in terms of microbiota-modifying interventions for a broader landscape of pediatric disorders.
- We can conclude that a novel perspective in the growing field of microbiota modifying therapies in children with FGIDs may offer valuable insights of disease mechanisms so personalized therapeutic strategies might improve patients' symptoms.

[CONCLUSION]

Microbiota targeted intervention might result in significant changes in the gastrointestinal dysbiosis and this finding is related to gastrointestinal symptoms relief in patients with functional abdominal pain disorders.

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ESPGHAN 57th ANNUAL MEETING
of the European Society for Paediatric Gastroenterology, Hepatology and Nutrition

MAY 2025

ESPGHAN 2025: Focus on microbiota-drug interactions

The 57th Annual Meeting of ESPGHAN placed a strong focus on the bidirectional interactions between the gut microbiota and medications in the context of pediatric gastroenterology, nutrition, and pharmacomicrobiomics. A recurring theme across presentations was the growing recognition of the gut microbiome as a central factor in drug therapy, immune modulation, and disease management in children.

Mechanisms

Microbiota includes a wide variety of bacteria, viruses, fungi, and other microorganisms which have been found to be crucial for immunologic, hormonal, and metabolic homeostasis of their host. We often referred to it as a "hidden organ".

When this ecosystem is disrupted (dysbiosis), it can contribute to a wide range of diseases - from gastrointestinal diseases to systemic metabolic and neurological disorders [1].

At birth, the newborn's gut is sterile, but it is rapidly colonized by microorganisms from the environment, including *Enterobacteria, Enterococci, Lactobacilli,* and *Bifidobacteria*. The gut microbiota undergoes dynamic and gradual changes from infancy to adulthood, shaped by various internal and external factors. These microbial shifts are critical for establishing a stable and resilient microbiome that supports health across the lifespan. In healthy adults, the gut microbiota is estimated to include over 1,000 species of bacteria. Importantly, this microbial community can influence drug pharmacodynamics by either directly metabolizing drugs or modifying the host's metabolic and immune responses.

Orally administered drugs travel through the gastrointestinal (GI) tract, with their absorption and metabolism influenced at each stage. Drugs that are not completely absorbed in the upper GI tract may reach the colon. In turn, the gut microbiome actively participates in the chemical transformation of these drugs, affecting their pharmacokinetics, bioactivity, and potential toxicity.

Several mechanisms are involved by which drugs affect gut microbiota, including:
1 / direct effects (antibiotics can kill some species of microbiota, including both harmful and beneficial species, leading to imbalances in gut microbiota);
2 / altered gut motility (particular drugs can slow down gut motility, which can lead to overgrowth of harmful bacteria);
3 / modulation of immune function (several drugs can interact with gut immunity which in turn can affect gut microbiota);
4 / changes in pH in the intestine (the pH balance plays a significant role in the gastrointestinal tract which affects the growth and survival of different types of species of gut microbiota. Some drugs can change the pH value of the gut, which affects the proliferation of different microbes, thereby affecting the overall composition of gut microbiota);
5 / interference with microbial metabolism (several drugs can interfere with microbial metabolism, which may have an effect on gut microbiota);
6 / dietary changes (certain drugs can change the dietary environment in the gut. This may influence gut microbiota by changing the availability of nutrients and other compounds that gut microbiota use to grow and survive) [2-4].

Photo: Shutterstock.

Gut microbiome-drug interactions are shaped not only by microbial activity but also by host genetics, environmental exposures, and their interplay, posing a complex challenge for personalized therapy. Genome-wide association studies (GWAS) have identified human genetic variants, especially in genes related to immunity, metabolism, and digestion (*e.g.*, C-type lectins and lactase) that influence gut microbiota composition.

The examples of irinotecan and cytochrome p450

Irinotecan, an anti-cancer medication, is reactivated in the gut by microbial enzymes causing severe diarrhea - a major side effect of the chemotherapy. Certain gut bacteria, particularly β-glucuronidase-producing species such as *Escherichia coli, Clostridium* and *Bacteroides*, produce enzymes that convert SN-38G back into its active form SN-38 in the intestine. This reactivation is toxic to intestinal epithelial cells, causing mucosal injury, inflammation, and severe delayed-onset diarrhea [3].

The gut microbiome can profoundly influence the host's drug-metabolizing enzymes, an emerging factor in personalized medicine. Cytochrome P450 enzymes, particularly CYP3A4, are modulated by gut-derived compounds. Short-chain fatty acids (SCFAs) can modulate enzyme gene expression through epigenetic mechanisms. Meanwhile, secondary bile acids interact with nuclear receptors like FXR, CAR, and PXR, altering drug metabolism [3].

Strategies for reducing the collateral damage of drugs on the microbiome [5]

To protect the gut microbiome, one key strategy is to avoid drugs known to disrupt microbial balance whenever possible. Minimizing direct interaction between drugs and gut microbes can reduce negative effects. In contrast, restorative approaches aim to repair microbial communities after disruption. These include dietary interventions, probiotics, live biotherapeutic products, and fecal microbiota transplantation. Dietary interventions act as microbiota-targeted therapies. Dietary fibers, for instance, foster the growth of SCFA-producing bacteria, which are essential for immune function, epithelial development, and maintaining an anaerobic gut environment5. Probiotics such as *Saccharomyces boulardii* CNCM I-745, *Lactobacillus reuteri* and *Bifidobacterium* spp. support colonization resistance, immune modulation, and gut barrier integrity. Postbiotics, composed of inactivated microbes or their components, also offer health benefits without requiring live organisms. Meanwhile, live biotherapeutic products represent a new category of medical interventions using live microbes specifically designed to treat or prevent disease, distinct from traditional supplements [3].

Restoring the microbial community involves more than simply recolonizing bacteria. It requires reestablishing a balanced ecosystem that supports immune, metabolic, and barrier functions. Strategies to protect the microbiome during drug therapy fall into two main categories: preventive approaches that minimize drug-induced disruption, and restorative approaches that aim to rebuild microbial diversity and function after damage has occurred [5].

Selecting the right strategy requires a precision-based approach, tailored to the drug, disease context, and patient. Success depends on a deep understanding of the ecological and biochemical principles that govern microbiota–drug interactions. Ongoing research is essential to guide effective recovery and protection of the gut microbiome during and after drug therapy.

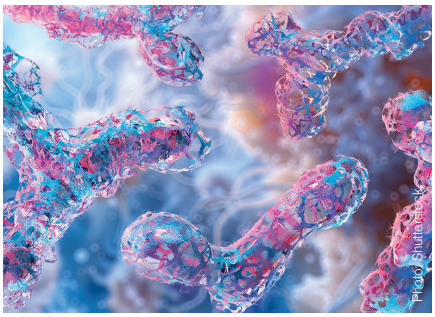

Photo: Shutterstock.

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GUT MICROBIOTA

Gut microbiota regulates insomnia-like behaviors *via* gut-brain axis

While sleep is known to be in bidirectional connection with the gut microbiota, the underlying mechanisms have been largely unknown. However, it seems that gut-derived metabolites can affect some behaviors in the host, such as anxiety-like behavior. Additionally, some clinical studies have reported alterations in the gut microbiota in individuals with chronic insomnia.

Wang *et al.* sought to clarify how the gut microbiota could shape sleep behavior. For this purpose, they studied sleep-wake behavior in specific pathogen-free (SPF) and germ-free (GF) mice. GF mice are free of all microorganisms, including those that are typically found in the gut, while SPF mice are free of a specific list of pathogens by routine testing. The recording of 24-h ambulatory electroencephalogram (EEG)-electromyogram (EMG) showed that GF mice had decreased time of wakefulness and REM sleep compared to SPF mice. To identify specific metabolites that are involved in gut microbiota-mediated sleep-related behavioral changes, the authors studied feces and hypothalamus tissue samples using targeted metabolomics. It was found that gut microbiota-derived short chain fatty acid, butyrate was the most significant modulator of sleep behavior. Further, oral administration of tributyrin, a precursor of butyrate administration led to a significant 39.50% reduction of wakefulness and 77.99% increase in REM sleep. The underlying mechanism seems to be that tributyrin inhibits lateral hypothalamus orexin neuron activity.

By studying humans, the authors also observed a decrease in 39 butyrate producers in insomnia patients compared with controls. Ultimately, the authors also showed that GF mice that received microbiota from insomnia patients exhibited sleep disturbances, which were recovered by butyrate supplementation. To conclude, the study highlights the potential of butyrate as a therapeutic agent to mitigate sleep disorders.

Wang Z, Wang Z, Lu T, *et al.* Gut microbiota regulate insomnia-like behaviors via gut-brain metabolic axis. *Mol Psychiatry* 2025; 30: 2597-611.

GUT MICROBIOTA

Campylobacter jejuni-derived cytolethal distending toxin promotes colorectal cancer metastasis

Several pro-tumorigenic bacteria, such as genotoxic *Escherichia coli* (*E. coli*), and enterotoxigenic *Bacteroides fragilis* (*B. fragilis*), have been associated with the promotion of cancer metastasis.

In addition, cytolethal distending toxin (CDT)-producing *Campylobacter* have been found to be enriched in tumor tissues compared to normal adjacent tissues. However, the connection between genotoxin-producing bacteria and cancer metastasis is poorly understood.

The authors of this study obtained primary colorectal cancer (CRC) tissues from 34 chemotherapy-naive patients (TNM stage I and IIA) with distant metastasis within 3 years (metastasis group) and 37 patients who remained metastasis-free (non-metastasis group) during 3 years' follow-up. They found a significant enrichment of *Campylobacter* in the metastasis group, and that the patients with intratumor *Campylobacter* had significantly poorer prognosis. They also confirmed their findings using a validation cohort and a publicly available database.

CDT is the major virulence factor responsible for *Campylobacter*-mediated pathogenesis, and in the host cells it induces DNA damage and cell-cycle arrest. The metastasis group expressed more bioactive CDT subunit *cdtB* and *Campylobacter* invasion antigen B (*ciaB*), a virulence factor specific to *C. jejuni*. *In vitro*, *C. jejuni* significantly increased cell migration and invasion ability of various CRC cell lines. In one mice model, administration of *C. jejuni* increased migration and invasion ability as compared with controls, and in another it significantly increased liver metastasis. Altogether, these findings prove that intestinal *C. jejuni* promotes CRC metastasis. Interestingly, the pro-metastasis ability was attenuated in the absence of *CdtB*. Mechanistically, it seems that CDT activated JAK-STAT signaling pathway leading to expression of MMP genes and tumor metastasis.

He Z, Yu J, Gong J, *et al. Campylobacter jejuni*-derived cytolethal distending toxin promotes colorectal cancer metastasis. *Cell Host Microbe* 2024; 32: 2080-91.

GUT MICROBIOTA

Quiescent Crohn's disease, sulfidogenic microbes and sulfur metabolic pathways: the functional consequences

In the quiescent inflammatory bowel disease, there is no active inflammation. However, the patients report persistent symptoms, especially with Crohn's disease (CD). The microbiome is shown to be altered in quiescent CD patients with persistent symptoms (qCD + S). Specifically, the patients with qCD + S have been shown to have more sulfidogenic microbes and microbial gene pathways of sulfur metabolism. Nevertheless, the functional significance of these changes has remained unknown.

In this multicenter observational study, metagenomic shotgun sequencing and metabolomics profiling of the qCD + S patients' feces were performed. Additionally, patient with active Crohn's Disease (aCD), with quiescent Crohn's disease without persistent GI symptoms (qCD-S) and with diarrhea predominant irritable bowel syndrome (IBS-D) were included and compared with qCD + S.

The authors report that fecal metabolites within cysteine/methionine, bile acid, and fatty acid pathways were among the most differentially abundant in qCD + S patients relative to other groups. The differences persisted even when inflammation, *i.e.*, calprotectin levels were lower. Glycine, serine, and threonine; glutathione; and cysteine and methionine were the most enriched pathways in qCD + S, and these are important sulfur metabolic pathways in the human gut. In addition to metabolites, many bacterial sulfur metabolic genes were dysregulated in qCD + S.

By integrating the metagenomic and metabolomic datasets, the authors further found that taurine and hypotaurine; nicotinate and nicotinamide; cysteine and methionine; and glycine, serine, and threonine were the top metabolic pathways associated with the enriched microbes in qCD + S. AS elevated H_2S concentrations inhibit mitochondrial functions of the host, the results suggest links between microbial-derived metabolites and host mitochondrial function in patients with qCD + S. Altogether, the results of this study suggest that strategies to decrease sulfidogenic microbes and associated sulfur metabolic pathways could represent a novel strategy to improve quality of life in quiescent Crohn's disease with persistent symptoms.


Photo: Shutterstock.

Golob J, Rao K, Berinstein JA, *et al.* Why Symptoms Linger in Quiescent Crohn's Disease: Investigating the Impact of Sulfidogenic Microbes and Sulfur Metabolic Pathways. *Inflamm Bowel Dis* 2025; 31: 763-76.

VAGINAL MICROBIOTA

Transgender women: a specific neovaginal flora

Some transgender women correct the gender incongruity of feeling like a woman in the depth of their being despite the physical presence of male genitalia and being referred to as a man by undergoing "*penile inversion vaginoplasty*." In other words, by surgically transforming their penis into a vagina. However successful the surgery, the skin of this newly constructed vagina will combine skin from the penis and a skin graft from the scrotum and/or other area (s) (stomach, groin, etc.). How does this affect health? Vaginal microbiota makes a crucial contribution to good vaginal health in cisgender women. And American researchers have now turned their attention to the intimate flora of transgender women undergoing surgery: might the composition of neovaginal microbiota explain certain problems, including the frequently reported issue of vaginal discharge?

It is a question worth asking, and one that has now been answered thanks to a study comparing the vaginal microbiota of transgender women undergoing vaginoplasty with that of cisgender women. The results? They have very different microbiota. The vaginal flora of cisgender women is not very diverse and is dominated largely by lactobacilli, which creates an acidic environment that repels pathogens. That of transgender women has less than 3% of these precious allies and is much more diverse. Diversity in the vagina is not a sign of good health; quite the opposite. It is observed in cisgender women suffering from bacterial vaginosis, which increases risk of sexually transmitted infections (including HIV/AIDS) and miscarriage.


Photo: Shutterstock.

How is this new microbial ecosystem created? Or more precisely, which bacteria make up the neovaginal microbiota of transgender women having undergone surgery? They result no doubt from the flora of the skin (penis, scrotum, etc.) used during surgery. However, oral-genital and genital-genital transmission also appears to be involved. In fact, the neovaginal flora of transgender women having undergone surgery has been shown to include bacterial species typical not only of the skin and digestive tract, but also of the mouth. Since sexual relations influence the likelihood of a bacterium called *E. faecalis*, there is also genital transfer.

On the other hand, while the proliferation of protective lactobacilli in cisgender women can be explained by hormones, the hormonal status of transgender women (comparable to that of cisgender women due to treatment) seemed to make no difference. Further studies on larger numbers of transgender women will be needed to better understand their neovaginal health.

Winston McPherson G, Goldstein Z, Salipante SJ, *et al.* The Vaginal Microbiome of Transgender and Gender Nonbinary Individuals. *Transgend Health* 2024; 9: 205-11.

By Dr. Hanna Stolińska
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Live probiotic coconut yogurt, is it worth recommending?

Health influencers on social media are buzzing about a super-live probiotic coconut yogurt claiming to revolutionize gut health. Marketed as a superfood packed with billions of probiotics, it has gained a cult-like following among wellness enthusiasts. Fans praise its supposed benefits, from improved digestion to healthier skin, but what does science have to say compared to traditional probiotics? Is this coconut-derived probiotic-rich formula a true microbiome booster or just another overhyped wellness trend?

> What is inside the probiotic coconut-based yogurt?

The coconut-based yogurt is made from organic coconut meat and coconut water, fermented with 16 custom probiotic strains, including *Lactobacillus acidophilus, Bifidobacterium breve*, and *Streptococcus thermophilus*.

> What health benefits does it claim to offer? Is it truly beneficial for health?

It is reported online that consuming this yogurt may improve digestion, reduce bloating, promote healthier skin, and strengthen the immune system. While probiotics can offer health benefits, scientific evidence specifically supporting this yogurt's claim remains limited.

We know that coconut flesh is mainly composed of lipids and such yogurt provides very little protein. On top of that coconut oil is used as a cure for all sorts of ailments, such as fighting viruses and bacteria, supporting immunity, reducing cholesterol, supporting thyroid function, and even weight loss [1-4].

Coconut oil contains medium-chain triglycerides (MCT) fatty acids, which are more easily digested and less absorbed compared to longer-chain fatty acids. However, of all the saturated fatty acids in coconut oil, MCTs constitute only half. Some MCTs, such as lauric acid and capric acid, have antifungal and antiviral properties [1-3], but the purpose of consuming food is to provide components that strengthen the immune system, which then fights microbes [4].

> How does it impact the gut microbiota?

This probiotic-enriched yogurt contains live bacteria that can potentially influence gut microbiota. However, the effectiveness of probiotics depends on various factors, including the specific strains used, their ability to survive stomach acid, and the individual's existing gut microbiome composition.

Some studies suggest that medium-chain triglycerides (MCTs), found in coconut, may affect microbiome composition. However, research on probiotic coconut-based products is still in its early stages. Moreover, while coconut oil contains antimicrobial compounds like lauric acid (converted into monolaurin in the body), this does not necessarily translate into overall gut health benefits [1, 3].

An interesting rat study investigating different dietary oils' effects on gut microbiota found that coconut oil consumption led to reduced bacterial diversity, increased markers of metabolic endotoxemia, fatty liver disease, and higher LDL cholesterol levels [5]. While animal studies provide insight, further clinical trials in humans are needed to determine this probiotic-enriched yogurt's actual effects on gut health.

> From your dietary perspective, are these new probiotic products worth considering?

Remember that food is primarily intended to provide us with nutrients, vitamins and minerals. Coconut dairy, like yogurt, does not have high nutritional density [1, 3].

While this probiotic-rich yogurt presents an innovative approach to delivering beneficial bacteria, its high-fat content, low protein levels, and cost should be considered when recommending it as a dietary option [2, 6]. Traditional probiotic-rich foods, such as yogurt, kefir, and fermented vegetables, provide similar benefits with a more balanced nutritional profile. A varied and balanced diet, low in processed foods and rich in vegetables, fruits, whole grains, and legumes, supports microbiome diversity.

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Diagnosis of functional dyspepsia simplified

Functional dyspepsia affects about 7% of adults but is often misdiagnosed due to overlapping symptoms with reflux, gastroparesis, and IBS. As a disorder of gut-brain interaction, it involves altered motility, microbiota imbalances, and psychological factors, making diagnosis challenging.

To help Prof. Maura Corsetti, Prof. Nicholas Talley, and Prof. Lucas Wauters, leading experts in the field, in collaboration with the Biocodex Microbiota Institute, have developed a Functional Dyspepsia Diagnosis Checklist. This tool aids in more accurate diagnosis and clearer patient communication, improving management and care.

The printable PDF provides at a glance:

- screening algorithms for red flags and subtypes
- symptom frequency and exclusion rules
- visual disease maps and patient FAQs
- a table classifying tests as essential, optional or unnecessary

Download:

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BIOCODEX MICROBIOTA FOUNDATION

Gut Microbiota International Grant 2025 awarded to Prof. David Artis

Each year, the Foundation unveils a new line of research. The theme for 2025 was: "Transformation of bile acids by the gut microbiota: functional implications for the host and consequences for human health".
Professor David Artis (Weill Cornell Medicine, USA) has been chosen as the 2025 winner for his project:
"Microbiota-dependent regulation of bile acids in the control of metabolic homeostasis".

Project overview

Humans have evolved along with the beneficial microbes that colonize the gut and other barrier tissues. These partners facilitate food digestion and nutrient absorption, promoting normal physiology, immune defense and metabolism. They also produce bioactive molecules that further enhance nutrient absorption.
Bile acids are among the most influential molecules. Synthesized from cholesterol in the liver, they enter the intestine to emulsify fats, but their influence extends far beyond digestion: bile acid signaling guides development, immune responses, cognitive functions and overall metabolic health through a complex crosstalk between host and microbe.
Professor Artis's research will clarify how the microbiota sculpts bile acid stores and how this dialogue maintains metabolic balance. This knowledge could lead to new strategies for preventing or treating metabolic disorders linked to bile acid dysregulation.

2025 Gut Microbiota International Grant Winner

Prof. David Artis
Professor and Institute Director, Weill Cornell Medicine

Microbiota-dependent regulation of bile acids in control of metabolic homeostasis (acronym: MRBM)

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Performed by

Editor:
John Libbey Eurotext
Bât A / 30 rue Berthollet, 94110 Arcueil, France
www.jle.com

Publication director:
Gilles Cahn

John Libbey Eurotext

Realisation:
Scriptoria-crea

Photo credits:
Cover photo: Bacteroides sp. Kateryna Kon/ Science Photo Library /Getty images

ISSN : 2782-0505

22 rue des Aqueducs - 94250 Gentilly – France – Tél. : +33 1 41 24 30 00

BIOCODEX Microbiota Institute

BMI-25.06 - Imprimé en France