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Dysosmobacter: from a serendipitous discovery to a next-generation beneficial gut commensal linked to health

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ABSTRACT

Dysosmobacter is a genus of strictly anaerobic, butyrate-producing bacteria of the family *Oscillospiraceae* that was first described in 2020 with the human faecal type species *Dysosmobacter welbionis* J115^T. The organism was recovered by chance during a targeted attempt to isolate new *Subdoligranulum* species, a project that failed in its original aim but yielded a bacterium that fulfilled the criteria for an entirely new genus. Since then, *D. welbionis* has emerged as one of the best-supported examples of a candidate next-generation beneficial microbe. It is carried by roughly two thirds to more than 90% of adults depending on the cohort and detection method, its faecal abundance correlates inversely with fasting glucose and glycated haemoglobin, and, in contrast to most microbiome markers, live supplementation of the type strain reproducibly improves diet-induced insulin resistance, brown adipose tissue thermogenesis and hepatic steatosis in mice. At the biochemical level *D. welbionis* is distinguished by a non-canonical route that converts *myo*-inositol to butyrate, by the production of bioactive lipids that act as peroxisome proliferator-activated receptor gamma agonists and by the capacity to convert cholesterol to poorly absorbed coprostanol. A rapidly growing body of clinical and veterinary literature now places *Dysosmobacter* at the crossroads of metabolic disease, liver disease, cardiovascular health, oncology, the gut–brain axis and drug–microbiome interactions. This review provides a comprehensive overview of the genus. It brings together its discovery, taxonomy, physiology and genomics, its ecology and prevalence in humans and animals, the mechanistic and preclinical evidence, the expanding catalogue of human disease associations and the prospects and hurdles for its development as a live biotherapeutic.

INTRODUCTION

The recognition that the gut microbiota shapes host metabolism has reoriented a large part of gastroenterology and hepatology research towards the identification of individual bacterial species that could be developed into live biotherapeutics.[1] The proof of principle came from *Akkermansia muciniphila*, whose supplementation improved metabolic parameters in mice[2 3] and then in a human pilot study,[4] establishing the concept of the next-generation beneficial microbe.[5] The search for further candidates has usually started from metagenomic association studies, in which a taxon that co-varies with a favourable phenotype is selected for functional testing. The difficulty is that correlation rarely survives causal scrutiny, and the field is littered with promising markers that did nothing when tested in isolation.[6] *Dysosmobacter* is a striking exception, and its story is a useful reminder that the most interesting organisms are sometimes found while looking for something else. In this review we bring together the current knowledge on the genus. We describe how *Dysosmobacter welbionis* J115^T was isolated, why it was given a name that means 'bad-smelling rod', how the genus has since expanded and what makes its metabolism unusual. We then look at how prevalent it is across human populations, what the preclinical models show and the mechanisms through which it acts. Finally, we take a dive into the clinical literature which now connects *D. welbionis* to a surprisingly wide range of conditions. We aim to provide a comprehensive and balanced account by covering both the findings that support a beneficial role for *Dysosmobacter* and those that point to context-dependent or divergent associations.

DISCOVERY: A BACTERIUM FOUND WHILE HUNTING *SUBDOLIGRANULUM*

The discovery of *Dysosmobacter* began with a different genus. Large-scale metagenomic surveys had repeatedly flagged *Subdoligranulum* as a marker of metabolic health, a taxon that co-occurs with *A. muciniphila*, responds to dietary fibre and metformin (the standard first-line drug for type 2 diabetes), and is depleted in metabolic or liver diseases.[7–11] To test whether that association was causal, our team supplemented obese and diabetic mice with the only cultured isolate then available, *Subdoligranulum variabile* DSM 15176^T. Despite its promising epidemiological profile,

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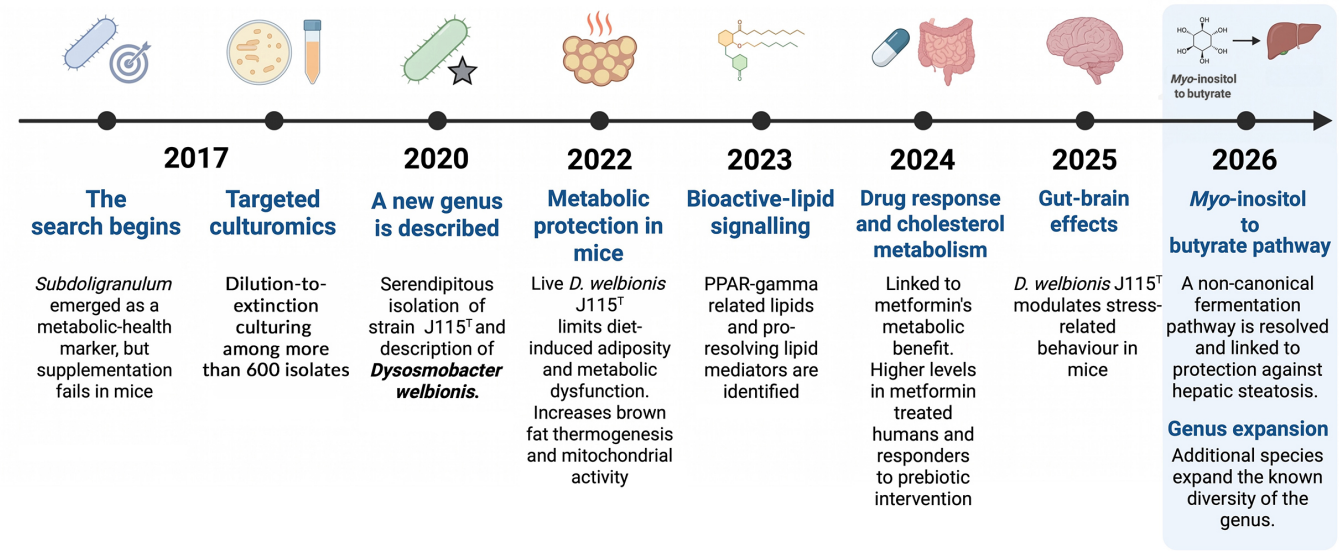


Figure 1 Timeline of the discovery and characterisation of *Dysosmobacter*. A chronological timeline that begins with the identification of *Subdoligranulum* as a metabolic-health marker and the failure of *Subdoligranulum variabile* supplementation to improve metabolic disorders in mice, proceeds through the targeted culturomics campaign and the serendipitous isolation of strain *Dysosmobacter welbionis* J115^{T} among more than 600 isolates, the formal description and naming of *D. welbionis* in 2020 and then the successive functional milestones with the prevention of diet-induced metabolic disorders in 2022, the bioactive-lipid mechanism in 2023, the metformin interaction in 2024, the gut–brain effect in 2025 and the *myo*-inositol to butyrate pathway with the liver phenotype in 2026, alongside the expansion of the genus to additional species. Created with BioRender.com. PPAR, peroxisome proliferator-activated receptor.

the type strain failed to improve diet-induced metabolic disorders in mice, a negative result suggesting that the human health signal might not be causal for this strain, or might instead arise from other, closely related and still uncultured members of the genus[12] (figure 1).

To capture those uncultured relatives, a targeted culturomics campaign was launched and 14 faecal samples from healthy donors were screened. Among them, three samples in which *Subdoligranulum* represented roughly 1.5% of the microbiota, both by quantitative PCR and 16S ribosomal RNA gene sequencing, were selected for isolation by the dilution-to-extinction technique. No new *Subdoligranulum* species were recovered. Instead, among more than 600 isolates, one bacterium fulfilled the criteria for a novel genus and species. We formally described and named *D. welbionis* with type strain J115^{T}[13] (figure 1). The genus name derives from the Greek for bad smelling and the Latin for rod, a reference to the characteristic odour the organism produces in culture and the specific epithet honours the WELBIO programme, the 'Walloon Excellence in Life Sciences and Biotechnology' initiative under which the work was funded.

TAXONOMY AND EXPANSION OF THE GENUS

Dysosmobacter belongs to the family *Oscillospiraceae*, formerly *Ruminococcaceae*, within clostridial cluster IV of the phylum *Bacillota*. Phylogenetically it forms a distinct monophyletic lineage that is sister to the *Oscillibacter* and *Oscillospira* clade. The type species shares only 95.4% 16S ribosomal RNA gene identity with *Oscillibacter ruminantium* and 94.1% with *Oscillibacter valericigenes*, values well below the genus and species thresholds, and genome-based metrics such as average nucleotide identity (ANI) near 73% and in silico DNA-DNA hybridisation around 20% confirmed that a new genus was warranted. During characterisation, we also produced fresh biochemical data for the two *Oscillibacter* type strains and formally emended the descriptions of that genus and its species.[13] Since its discovery, the genus has grown and two other species have been published. *Dysosmobacter hominis*, with type strain NSJ-60, was described from human faeces in China as part of a large, cultured biobank. The same study proposed another species, named *Dysosmobacter segnis*, which remains not validly published because the type strain was not deposited in the required culture collections.[14] A third validly published species, *Dysosmobacter acutus*, was isolated from the faeces of a cynomolgus monkey and named for its pointed cell morphology, making it the first member of the genus recovered from a non-human host.[15] Based on genome relatedness, *D. hominis* is now treated as a later heterotypic synonym of *Pusillibacter faecalis*, and *Oscillibacter acetigenes* as a later heterotypic synonym of *D. welbionis*. We note, for accuracy, that these synonyms reflect genome-taxonomy clustering rather than a formal act under the International Code of Nomenclature of Prokaryotes, and that the two names still appear as separate valid names in nomenclatural databases. We further isolated a human-associated species, through phytate enrichment, provisionally named *Dysosmobacter phytatis*, which shares the *myo*-inositol to butyrate trait of

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the type species (unpublished). Finally, a chicken caecal metagenome-assembled genome has been proposed as *Candidatus Dysosmobacter* (or *Oscillibacter*) *pullicola*.[16] The recurring discordance between the *Oscillibacter* and *Dysosmobacter* boundaries in genome-based taxonomy, on the one hand, and validly published names on the other, is worth keeping in mind whenever the genus is discussed, because several apparent renamings are database artefacts rather than nomenclatural decisions.

CELL BIOLOGY, GENOMICS AND UNUSUAL METABOLISM

D. welbionis J115^{T} is a strictly anaerobic, non-motile and non-spore-forming rod that stains Gram negative despite its position within the Gram-positive phylum *Bacillota*. This staining phenotype should not be equated with a classical diderm Gram-negative envelope: available genomic analyses do not support the presence of a coherent lipid A and lipopolysaccharide (LPS) biosynthesis pathway, LPS transport or outer-membrane assembly module, whereas genes consistent with a Gram-positive-type monoderm envelope, including teichoic-acid-related genes, are present. Cells are mostly straight rods of 1.8–3.0 µm, although elongated forms up to 18 µm occur and the cell wall contains meso-diaminopimelic acid. The complete genome is a single contig of about 3.58 megabases with a DNA G+C content of 58.9%, and it carries three identical copies of the 16S ribosomal RNA gene.[13] Recently, we isolated and performed comparative genomics of 23 *D. welbionis* strains, including the type strain and 18 new isolates recovered by antibody-guided anaerobic cell sorting. They showed an intraspecies ANI above 98% and an open pangenome of roughly 8000 genes with a core of about 2100 genes, indicating substantial accessory diversity within a well-defined species.[17] The metabolic signature of the genus is what sets it apart. Common sugars such as glucose, ribose and xylose are not used, whereas growth is supported in *myo*-inositol-containing media.

The type species is a butyrate producer, with butyrate being the main short-chain fatty acid (SCFA) secreted after *myo*-inositol fermentation, followed by acetate.[13 17] The molecular basis of this preference was resolved in detail using phenotype microarrays, isothermal microcalorimetry, stable-isotope tracing and proteogenomics. Of 190 substrates screened, only *myo*-inositol and the nucleosides uridine and inosine increased growth by more than 50%. Notably, *myo*-inositol supported maximal growth at a concentration of 0.75%, resulting in a threefold increase in bacterial biomass and a decrease in medium pH from 8.25 to 5.70. Its fermentation yielded mainly butyrate and acetate, together with traces of branched-chain acids. Metabolic tracing using position-labelled ^{13}C *myo*-inositol, combined with high-resolution ^{13}C nuclear magnetic resonance and mass spectrometry, enabled reconstruction of the complete biochemical route from *myo*-inositol to butyrate.[17] Proteogenomic analysis further indicated that this conversion relies on an unusual, non-canonical genetic pathway. The inositol utilisation gene cluster is induced two to threefold on *myo*-inositol, yet the gene that normally performs the key aldol cleavage, *iolJ*, is absent from the genome. Two genes annotated as class II fructose-bisphosphate aldolases, both carrying the conserved domain associated with 5-dehydro-2-deoxy-D-gluconate 6-phosphate aldolase activity, are proposed to substitute for it and a downstream oxoacid dehydrogenase complex is proposed to convert 3-oxopropionate to acetyl coenzyme A. Electron flow is handled by an Rnf complex and ferredoxin, and a dedicated inositol transport binding protein imports the substrate. Importantly, the entire pathway is conserved across all 23 strains. Crucially, *D. welbionis* appears to be the only reported gut bacterium that converts inositol to butyrate, since other inositol users such as *Anaerostipes* species make propionate and acetate. It is essential to note that the pathway is inferred from proteogenomics and isotope tracing rather than proven by gene knockout, because genetic tools for strict anaerobes of this group are still lacking, and therefore the necessity of these enzymes for the metabolic and hepatic benefits remains formally unproven.[17]

Two further metabolic capabilities deserve to be mentioned. First, the genus produces an unusual repertoire of bioactive lipids, discussed in the mechanistic section below. Second, *D. welbionis* encodes the intestinal sterol metabolism A enzyme, IsmA, which converts cholesterol to the poorly absorbed sterol coprostanol. The *IsmA* gene family was originally defined in uncultured *Oscillospiraceae,* and its carriage tracks with lower host cholesterol,[18] and later work in the Framingham Heart Study explicitly associated *Oscillibacter* and *D. welbionis* with reduced faecal and plasma cholesterol, with their contribution adding to that of other *IsmA*-encoding taxa.[19] Within the genus, *IsmA* appears to be a feature of *D. welbionis* specifically. Regarding antimicrobial resistance (AMR), the type strain carries the tetracycline resistance gene *tet*(*W*), and AMR profiling across strains revealed variable carriage of aminoglycoside, macrolide and vancomycin-cluster genes, several of which are likely to be non-functional remnants. However, two isolates lacked any predicted resistance genes and were therefore proposed as more suitable candidates for further therapeutic development.[17]

ECOLOGY AND PREVALENCE IN HUMANS

Dysosmobacter is a common member of the healthy adult microbiota, and its prevalence estimates rise as detection methods improve and databases are enriched with newly discovered species.[20] In the foundational functional study, the genus was detected in 62.7% of the Human Microbiome Project cohort, 69.8% of the American Gut Project, 65.9% of the Flemish Gut Flora Project and in every one of the insulin-resistant, overweight participants of the Microbes4U cohort, with relative abundances reaching up to 9% in certain individuals and an average absolute abundance close to 6×10^{8} bacteria per gram of stool.[21] Using species-resolved metagenomics, later work

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detected the genus in about 94% of the TwinsUK cohort and 93% of a Chinese cohort spanning ages 3 to over 100 years. *D. welbionis* was the dominant species, with prevalences of 81% and 72%, respectively, and remained the most prevalent species even among the youngest participants.[17] Evidence specifically addressing the early-life trajectory of *Dysosmobacter* remains limited. In the longitudinal CHILD cohort, *D. welbionis* was detected in metagenomic profiles at 1 year of age, although its association with the fungal-derived-age measure did not reach statistical significance.[22] In a separate longitudinal cohort of 207 infants sampled at 1, 6, 12, 18 and 24 months, *D. welbionis* was detected during the first 2 years of life, and its abundance was inversely associated with fluctuations in postnatal PM_{10} exposure.[23] Beyond infancy, a cross-sectional Chinese cohort spanning ages 3–100+ years showed that *Dysosmobacter* was less abundant in children, although *D. welbionis* was already the dominant species in children aged 3–6 years.[17] Thus, available evidence supports its presence during infancy and early childhood, but does not yet define its normal acquisition or longitudinal developmental trajectory.

D. welbionis has not only been detected in stools, but also in the blood metatranscriptome of healthy individuals and patients with irritable bowel syndrome, consistent with a gut-derived blood microbiome signal shaped by intestinal permeability.[24] Interestingly, two specific observations point to host control of abundance. First, in monozygotic twins the relative abundance of *D. welbionis* was correlated within pairs whereas in dizygotic twins it was not, which suggests a heritable component.[17] Second, carriage strongly tracks with socio-economic status (SES), introducing the concept of the 'sociobiome' to *D. welbionis* ecology. In a diverse, multiethnic US cohort (the FAMiLI study), *D. welbionis* was identified as one of the most prominent 'higher SES-associated' taxa. Its abundance was significantly decreased in individuals with lower SES, showing a strong inverse correlation with both individual-level indicators (such as occupation) and neighbourhood-level factors (like the Social Deprivation Index). This marked depletion in lower SES populations is hypothesised to be linked to environmental and socio-demographic disparities, such as reduced access to fresh, high-fibre produce. These disparities may ultimately contribute to the disproportionately higher rates of diabetes and metabolic disorders observed in these demographic groups.[25] The species is also common in African populations, and appears far more prevalent in postindustrial than in traditional horticultural communities, a pattern consistent with a diet-linked ecological niche.[26 27] Indeed, food intake composition has been linked to *D. welbionis* relative abundance in multiple human cohorts. It was positively associated with coffee consumption in a UK cohort,[28] while it was inversely correlated with a modified healthy eating index in a cohort of 92 German participants.[29] Moreover, in a Swiss cohort of more than 1000 participants, multiple *Dysosmobacter* amplicon sequence variants (ASVs) were detected, each displaying distinct associations with various food groups, suggesting the existence of species-specific or strain-specific dietary associations within the genus.[30] Because food-derived free colonic *myo*-inositol is scarce, the genus is thought to occupy a specialised, possibly mucosa-associated niche in which it acts as a secondary degrader dependent on the liberation of inositol from phytate by other members of the community, a hypothesis that also fits its modest carbohydrate-active enzyme repertoire.

HUMAN ASSOCIATIONS ACROSS HEALTH AND DISEASE

The most consistent human associations concern obesity, dysglycaemia and metabolic dysfunction-associated steatotic liver disease (MASLD). In a cohort (Microbes4U) of 53 non-diabetic, insulin-resistant adults with overweight or obesity, *Dysosmobacter* was detected in all participants and its faecal abundance correlated inversely with body mass index (BMI), fasting glucose and glycated haemoglobin (HbA1c).[21] In this cohort, faecal *Dysosmobacter* concentration correlated positively and exclusively with a large set of plasma lipid species, including phosphatidylcholines, lysophosphatidylcholines, acylcholines, diacylglycerols, arachidonate, a ceramide and cholesterol, and with no non-lipid metabolite, which suggests a specific role in host lipid handling.[31] In a small paediatric shotgun-metagenomic study comprising 10 controls, 10 participants with metabolic syndrome and 10 with type 2 diabetes, *D. welbionis* was reduced in metabolic syndrome, whereas a specific association with type 2 diabetes was not established.[32] For liver disease, *D. welbionis* was significantly reduced in MASLD and metabolic dysfunction-associated steatohepatitis (MASH) in two independent cohorts (USA: 51 controls, 17 patients with MASLD without fibrosis and 25 with cirrhosis; Finland: 26 controls and 12 patients with MASLD) and was inversely associated with fibrosis score in a third Italian-Spanish cohort (n=686), although its abundance did not differ according to MASLD status in that third cohort.[17] Collectively, these observations support an association between *D. welbionis* and metabolic and hepatic health, but remain observational and do not establish causality.

The genus was also depleted in women with polycystic ovary syndrome (PCOS), recently renamed polyendocrine metabolic ovarian syndrome (PMOS),[33] a condition with prominent metabolic alterations, and was further reduced in hyperandrogenic disease.[34] Several cardiovascular signals are also emerging. In the Swedish CArdioPulmonary bioImage Study (SCAPIS) cohort, a *Dysosmobacter* species was associated with lower 24-hour systolic and diastolic blood pressure, with the diastolic association replicated in an office-measurement subsample,[35] and the Framingham cholesterol work discussed above ties the genus to lower circulating cholesterol through IsmA.[19]

In oncology, *D. welbionis* was significantly depleted in the stool of patients with intrahepatic-metastatic hepatocellular carcinoma and restored by healthy-donor faecal microbiota transplantation in a

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humanised mouse model, linking its loss to neutrophil extracellular trap driven metastasis.[36] *Dysosmobacter* was enriched in responders to anti-B-cell maturation antigen chimeric antigen receptor T-cell therapy for multiple myeloma[37] and associated with longer overall survival in a phase I glioblastoma trial combining radiotherapy, nivolumab and an indoleamine 2,3-dioxygenase 1 inhibitor.[38] In people living with HIV, *D. welbionis* contributed to a metagenomic signature of infection in a large African cohort,[27] and in a separate cohort on integrase strand-transfer inhibitors it was the second strongest microbial predictor of less treatment-associated weight gain and lower BMI.[39] In inflammatory bowel disease, a recent reanalysis of the Inflammatory Bowel Disease Multi'omics Database (IBDMDB) cohort found that *D. welbionis* was significantly less frequently detected in dysbiotic Crohn's disease and ulcerative colitis samples, while its abundance among carriers was not significantly reduced. This suggests that the association is driven mainly by loss of carriage in dysbiotic samples rather than by a uniform abundance decrease across all inflammatory bowel disease (IBD) samples.[40] Two datasets run counter to these generally favourable associations and warrant consideration. First, in a machine-learning study of major depressive disorder, *Dysosmobacter* was enriched in patients and selected as a potential diagnostic biomarker, contrasting with the generally favourable associations reported in metabolic studies, although no mechanism was proposed.[41] Second, in a Swedish cohort of women with frailty, while *Dysosmobacter* sp900544615 was associated with a lower frailty mortality index and better health outcomes, *Dysosmobacter* sp900770295 was positively associated with frailty, but only after adjustment for gene richness.[42] These observations are a useful corrective to any assumption that higher *Dysosmobacter* abundance is uniformly associated with favourable outcomes and emphasise the importance of disease and cohort context.

PRECLINICAL EVIDENCE AND MECHANISMS

What distinguishes *Dysosmobacter* from many taxa identified through microbiome association studies is that evidence extends beyond correlation: supplementation with the live type strain reproduces favourable metabolic effects in animal models, establishing causality in these experimental settings and providing biological plausibility for the associations observed in human cohorts, which remain correlative. In our foundational interventional study, we gave diet-induced obese mice a daily oral dose of >1 × 10^9 colony-forming units of live *D. welbionis* J115^T for 6–13 weeks. Treatment reduced adipocyte size at 6 weeks, with stronger effects over longer periods, decreased mesenteric, subcutaneous and epididymal fat depots without affecting lean mass, improved oral glucose tolerance and lowered fasting insulin, the insulin resistance index, leptin and resistin.[21] From a mechanistic point of view, white adipose tissue shifted towards smaller, more insulin-sensitive adipocytes with reduced inflammatory gene expression. The most striking effect was the impact on brown adipose tissue, where the bacterium prevented diet-induced whitening, increased the expression of thermogenic and respiratory-chain genes, raised citrate synthase activity by around 70% and increased rectal temperature on average by a quarter of a degree, indicating enhanced mitochondrial content and non-shivering thermogenesis. The effect required viable bacteria, as pasteurisation at 70°C for 30 min abolished the benefit. It was also dose dependent, being absent at a tenfold lower dose and model-specific, as no benefit was observed in genetically obese *ob/ob* mice. In addition, the faecal microbiota was barely altered, and colonisation was transient, arguing for a direct effect on host metabolism rather than an indirect remodelling of the community.[21] This supports the idea that the phenotype is driven by bacterial activity or specific bacterial products rather than by a broad ecological reset of the microbiota.

The mechanistic basis of the metabolic effect was pursued through the analysis of the lipidome. In vitro, when compared with the benchmark probiotic *Escherichia coli* Nissle 1917, *D. welbionis* pellets contained at least 19 bioactive lipids at concentrations 2–60 times higher than those detected in *E. coli* Nissle 1917. These included the brown-fat-activating oxylipin 12,13-dihydroxy-octadecenoic acid and the peroxisome proliferator-activated receptor gamma (PPARγ) agonist 3-hydroxyoctadecanoate (C18-3OH).[43] In high-fat-diet mice, treatment raised the PPARγ ligand 15-deoxy-Δ-12,14-prostaglandin J2 in brown adipose tissue by around 700% and the pro-resolving mediator resolvin D5 in the colon by around 660%, together with coordinated changes in a set of circulating lipids. These lipids provide a plausible link between the bacterium and the activation of thermogenic and mitochondrial gene programmes, as well as the resolution of inflammation, offering a mechanism that is at least partly independent of butyrate[43] (figure 2). As *D. welbionis* has been consistently associated with better glucose homeostasis, a third study addressed the clinically important question of how the bacterium interacts with metformin, the first-line treatment in type 2 diabetes.[44] In mice, metformin and *D. welbionis* J115^T acted through independent routes with no additive or synergistic effect. Metformin was more effective at limiting fat mass, whereas the bacterium was more potent at improving glucose tolerance and glucose-stimulated insulin secretion, and both normalised the diet-induced increase in liver weight. Part of the insulinotropic effect may be direct, since the bacterium stimulated glucagon-like peptide-1 (GLP-1)

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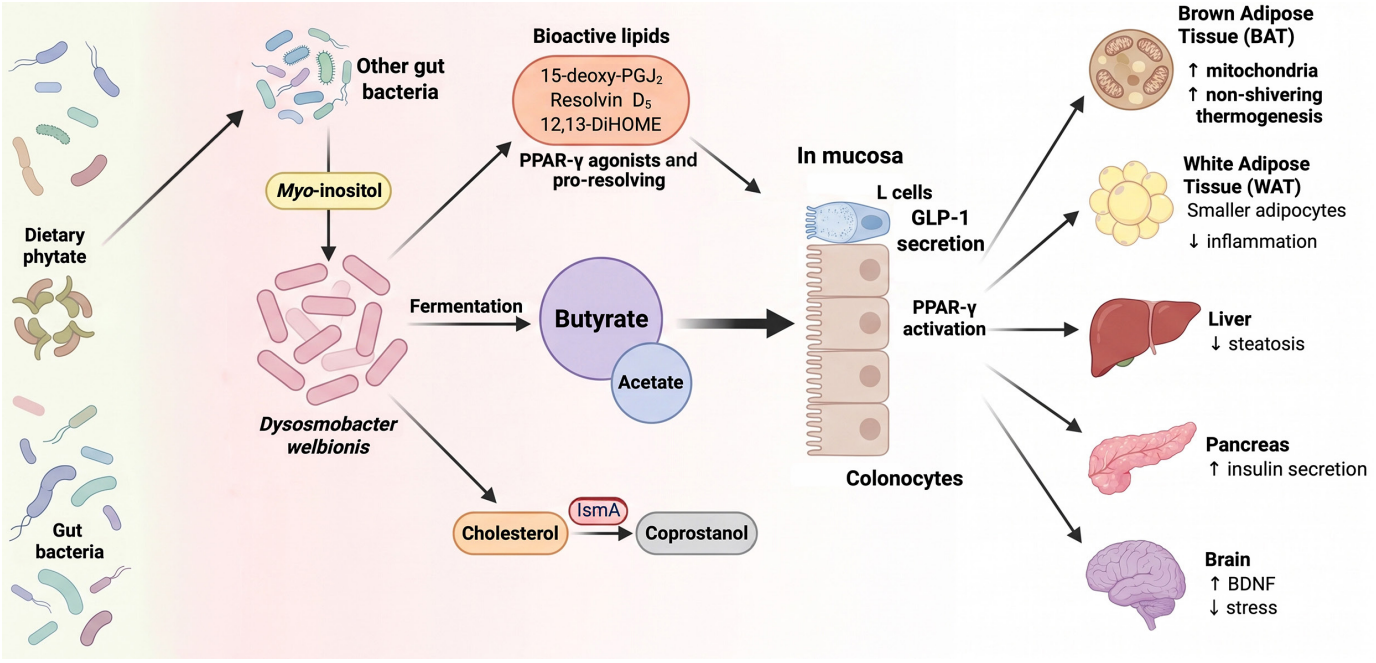


Figure 2 Mechanisms of action of *Dysosmobacter welbionis*. Schematic view of the principal routes by which *D. welbionis* J115^T influences host physiology. In the colonic lumen and at the mucosal interface, *myo*-inositol, liberated in part from dietary phytate by other gut bacteria, is imported and fermented through a non-canonical route to butyrate and acetate. Butyrate feeds colonocyte energy metabolism, supports epithelial hypoxia, engages PPARγ and G-protein-coupled receptors, and stimulates GLP-1 secretion from enteroendocrine L cells. The bacterium also secretes bioactive lipids that act as PPARγ agonists and pro-resolving mediators, raising 15-deoxy-PGJ_2 in brown adipose tissue and resolvin D5 in the colon, and it converts cholesterol to coprostanol through IsmA. Downstream, these signals increase brown adipose tissue mitochondrial content and non-shivering thermogenesis, normalise white adipocyte size and inflammation, improve insulin secretion and glucose tolerance, reduce hepatic fat and act along the gut–brain axis. Created with BioRender.com. BDNF, brain-derived neurotrophic factor; 15-deoxy-PGJ_2, 15-deoxy-delta-12,14-prostaglandin J2; 12,13-DiHOME, 12,13-dihydroxy-octadecenoic acid; GLP-1, glucagon-like peptide-1; IsmA, intestinal sterol metabolism A; PPARγ, peroxisome proliferator-activated receptor gamma.

secretion from enteroendocrine L-cells in a dose-dependent manner in vitro[44] (figure 2).

The liver phenotype was consolidated in the most recent mechanistic paper, which pooled four independent mouse studies.[17] Across them, live *D. welbionis* J115^T limited high-fat-diet-induced hyperglycaemia, prevented the diet-induced increase in liver weight and reduced hepatic steatosis quantified histologically, although circulating transaminases were not normalised.[17] Finally, the reach of the bacterium extends to the gut–brain axis. In high-fat-diet-fed female mice, 6 weeks of oral *D. welbionis* J115^T reduced anxiety and stress-like behaviour across the light-dark, elevated plus maze and forced swim tests, blunted the rise in corticosterone after behavioural challenge and increased cortical brain-derived neurotrophic factor, all without changing body weight or fat mass (figure 2). The effect was attributed to the gut–brain axis and most plausibly to microbial butyrate, although SCFAs were not measured in that study.[45]

THE DRUG AND DIET MICROBIOME AXIS

A distinct and clinically interesting topic is the sensitivity of *Dysosmobacter* to commonly used medicines, raising the possibility that it may contribute to some treatment effects while being adversely affected by others. Metformin use has been associated with a higher faecal abundance of *D. welbionis*, and its abundance correlated inversely with the liver enzymes alanine and aspartate aminotransferase, two circulating markers of hepatocellular injury. Metformin had no direct effect on bacterial growth in vitro, so the human association was attributed to microbial cross-feeding rather than a drug-bacterium interaction.[44] Rosuvastatin similarly increased its abundance in patients with coronary heart disease, in whom it was proposed as a potential mediator of the statin lipid-lowering response, whereas concomitant beta-lactam antibiotics depleted it[46] (figure 3). In IgA nephropathy the immunosuppressant leflunomide significantly enriched *D. welbionis*, which was interpreted as part of an anti-inflammatory and eubiotic community shift involving butyrate and histone deacetylase inhibition, although the leflunomide subgroup was very small.[47] Conversely, oral prednisolone reduced *D. welbionis* in a randomised trial in healthy men, in parallel with markers of insulin resistance.[48] In mice, early-life exposure to linezolid, a broad-spectrum antibiotic targeting Gram-positive bacteria, also disrupted a butyrogenic and anti-inflammatory network in a dysbiosis that was inherited by offspring in a sex- and *Dysosmobacter* ASV-specific manner.[49] In mice, methylglyoxal-induced

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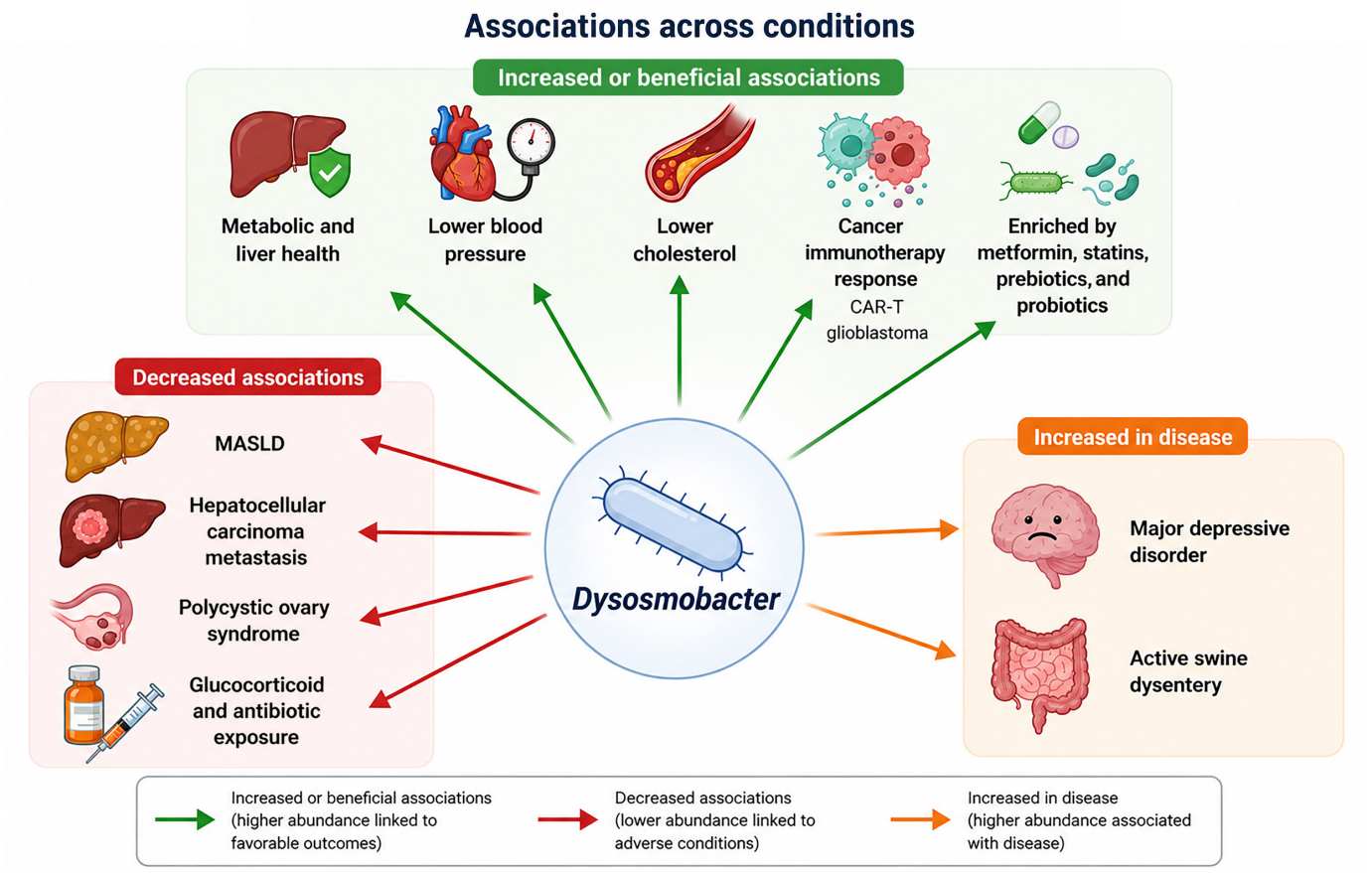


Figure 3 Health associations of *Dysosmobacter* across conditions. The panel maps the reported associations across organ systems and conditions, distinguishing settings in which the genus is increased or beneficial (green arrows), such as metabolic and liver health, lower blood pressure and cholesterol, treatment response in oncology and enrichment by metformin, statins, prebiotics and probiotics, from settings in which it is decreased (red arrows) such as MASLD, hepatocellular carcinoma metastasis, polycystic ovary syndrome, glucocorticoid and antibiotic exposure and from the examples of contexts in which it is increased in disease (orange arrows), namely major depressive disorder and active swine dysentery. Some elements of the figure were created with BioRender.com. CAR, chimeric antigen receptor; MASLD, metabolic dysfunction-associated steatotic liver disease.

cognitive impairment was associated with increased *Dysosmobacter* as part of a dysbiotic signature; this alteration was reversed by the acetylated nobiletin derivative (5-AN).[50]

Dietary and supplement interventions have likewise been associated with changes in *Dysosmobacter* abundance. Increases were reported after turmeric root extract,[51] a postbiotic intervention in chronic diarrhoea,[52] supplementation with *Bifidobacterium bifidum* in obesity,[53] and administration of a seaweed prebiotic in a mouse model of LPS-induced inflammation,[54] whereas daily pomegranate intake was associated with a decrease[55] (figure 3). The *Dysosmobacter* genus was also increased after supplementation with *Mori Fructus* aqueous extract in a mouse model of renal injury,[56] by *Codonopsis pilosula* in an immunocompromised mouse model and by soy-milk kefir supplementation in healthy mice.[57] Higher baseline *D. welbionis* abundance was also associated with a better metabolic response to prebiotic treatment, suggesting that the bacterium may act not only as a marker of metabolic health but also as a potential predictor of dietary-intervention responsiveness. Taken together, these studies suggest that *Dysosmobacter* is a responsive member of the microbiota (biomarker) across several drug, prebiotic, probiotic and dietary interventions, and may in some settings contribute to their metabolic consequences (figure 3).

DYSOSMOBACTER ACROSS ANIMAL HOSTS AND EXPERIMENTAL MODELS

The genus is not confined to human beings, nor to mice. *D. acutus* was isolated from a monkey,[15] and *D. welbionis* or close relatives have been reported across a range of other animal hosts. In broiler chickens, soluble-fibre and fish-oil supplemented diets enriched butyrate-producing *D. welbionis* under an enteric challenge[58] and in healthy animals,[59] respectively. A chicken caecal metagenome-assembled genome also forms the basis of the proposed *Candidatus Dysosmobacter pullicola*,[16] which has been incorporated into metabolic reconstructions of the chicken caecal microbiome.[60] In the donkey hindgut, *Dysosmobacter* was identified as a microbial network connector implicated in complex carbohydrate degradation,[61] while

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in weaned goats, rumen-protected methionine increased its abundance in association with volatile fatty acid production and growth.[62] In pigs, *Dysosmobacter* sp BX15 increased during clinical swine dysentery and returned to baseline after recovery, indicating an association with active infection rather than health.[63] In rhesus macaques, genome-guided analysis identified the genus among putative butyrate producers,[64] and comparative genomic atlases of gut clostridia now routinely include *Dysosmobacter* among taxa with SCFA-producing potential.[65]

In mouse models of host physiology, *D. welbionis* and related taxa respond to sex, diet and exercise.[66] In a murine spaceflight study, *D. welbionis* was significantly enriched after both 29 and 56 days of spaceflight, although the authors concluded that whether this increase counteracts or contributes to spaceflight-associated metabolic pathology remains unclear.[67] In addition, *D. welbionis* abundance correlates with intestinal endocannabinoidome mediators in cancer cachexia[68] and with antioxidant metabolites in models of arsenic neurotoxicity.[69] Finally, the *Dysosmobacter* genus was also detected in the gut microbiota of *Drosophila melanogaster*.[70] This broad distribution across animal hosts and physiological contexts supports the view that *Dysosmobacter* is a metabolically specialised and environmentally responsive gut bacterium. Across studies, its associations are predominantly favourable, particularly in relation to SCFA production, metabolic function and resilience to physiological stress, although the direction of association remains dependent on host species and biological context.

DEVELOPMENT AS A NEXT-GENERATION BENEFICIAL MICROBE

The case for developing *Dysosmobacter* as a live biotherapeutic is unusually compelling among next-generation microbes because its effects extend beyond observational associations: causal evidence has been obtained in preclinical models and the underlying mechanisms are increasingly being defined (figure 3). However, several practical considerations follow from the biology described above. The beneficial effects require live, metabolically active bacteria, as pasteurisation abolishes them. This distinguishes *Dysosmobacter* from *A. muciniphila*, whose key effect can be retained even after heat treatment. Strain selection will also be critical. Although the type strain carries the mobile tetracycline resistance gene *tet(W)*, two newly isolated strains lacked predicted AMR genes and may therefore represent more suitable starting points for therapeutic development.[17] Nevertheless, the beneficial phenotype should be considered strain-supported rather than automatically species-wide until candidate AMR-free strains have been tested head-to-head with *D. welbionis* J115^T in the same models. *Dysosmobacter*'s transient aerotolerance and its marked preference for *myo*-inositol offer levers for scale-up and formulation. Its specialised nutrient niche also raises the possibility that future applications may require synbiotic strategies, using substrates or partner microbes that favour its activity rather than relying on bacterial administration alone.

Additionally, recent investigations into the perinatal safety of *D. welbionis* J115^T indicate that daily oral supplementation from early gestation through weaning is well tolerated by dams and does not adversely affect gestation or pup development. Interestingly, this perinatal exposure was associated with mild, sex-dependent metabolic effects in offspring, including reduced fat mass selectively in female offspring during early adulthood (submitted data). Plausible initial indications include hepatic steatosis and MASLD, impaired glycaemic control and dyslipidaemia, potentially through cholesterol-to-coprostanol conversion (figures 2 and 3). Applications involving the gut–brain axis and adjunctive oncology are more exploratory. Our team is extending the work to a further human-associated species isolated by phytate enrichment, provisionally named *D. phytatis*, and is exploring a possible role in breast cancer, both lines of work are currently unpublished. Whether future development follows the regulatory pathway of a live biotherapeutic product under the regulatory framework for medicinal products or that of a novel food will ultimately depend on the intended indication and the claims being made.

LIMITATIONS AND OPEN QUESTIONS

Despite the encouraging findings, important gaps remain, defining the key questions for the next stage of research. A first limitation relates to bioinformatic classification. *Dysosmobacter* is absent from some reference databases used to classify sequencing reads, while databases that include the genus often contain few reference genomes and lack species-level annotation. These limitations may impair the accurate detection and classification of *Dysosmobacter* in microbiome datasets. Moreover, although the genus was first described in 2020, its members are still frequently misclassified as *Oscillibacter*, further complicating their detection in sequencing analyses. Overcoming these analytical barriers will be essential to accurately determine the prevalence and diversity of *Dysosmobacter* species and clarify their associations with different pathophysiological conditions. The human evidence remains largely associative, and, apart from the mouse interventions, causation in humans has not been demonstrated. The causal evidence is strongest for diet-induced metabolic and hepatic phenotypes in mice, whereas the human data currently position *D. welbionis* as a consistent marker and mechanistic candidate rather than a proven therapeutic organism. The high-fat-diet mouse model reproduces early steatosis but not the ballooning and fibrosis of advanced human liver disease, so the protective liver data need to be re-examined in fibrosis-oriented models. The quantitative contribution of *Dysosmobacter* to the total colonic butyrate pool is likely to be modest compared with dominant producers such as *Faecalibacterium prausnitzii*, *Anaerobutyricum soehngenii* and *Roseburia*. The spatial biogeography of the organism

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along the gut, and the identity of the specific molecules, whether butyrate, particular bioactive lipids or secreted proteins, that mediate its host effects, are still unresolved.

The absence of genetic tools for this group of strict anaerobes means that the novel *myo*-inositol to butyrate pathway has not yet been validated by knockout, so its necessity for the phenotype is inferred rather than proven. The presence of *tet(W)* in the type strain is a real regulatory consideration, and the depression and swine-dysentery data are reminders that the organism may not be beneficial in every context. The IBD prevalence-only signal similarly suggests that loss of carriage may reflect ecological disruption rather than a simple linear relationship between abundance and disease severity. Sex-specific effects remain an important open question, since the gut–brain phenotype and response to *D. welbionis* J115^{T} were most evident in female high-fat-diet-fed mice, whereas comparable sex-dependent effects on metabolic or hepatic outcomes have not yet been formally tested. This need for sex-stratified research is strongly supported by recent findings demonstrating that host sex acts as a primary driver for *D. welbionis* strains, independently of the host's diet or exercise status.[66] Addressing these questions will require controlled human intervention trials, careful strain selection, spatially resolved sampling and, ideally, the development of genetic systems for the genus.

CONCLUSION

Dysosmobacter has covered a remarkable distance in a short time, from an accidental isolate recovered while chasing a different genus to one of the best-supported candidate next-generation beneficial microbes in the human gut. It is prevalent, partly heritable and metabolically distinctive. Unusually, for a taxon first identified through human microbiome associations, supplementation of the live type strain reproduces its favourable epidemiological signature in animal models through mechanisms that touch brown adipose tissue thermogenesis, adipocyte biology, insulin secretion, bioactive-lipid signalling, cholesterol metabolism and the gut–brain axis. The clinical literature now connects it to metabolic disease, liver disease, cardiovascular health, oncology and drug responses, with a small number of instructive exceptions (figure 3). The central task ahead is to move from association to causation in humans, to identify the molecules that carry its effects and to select and manufacture the right strain for the right indication. If those steps succeed, *Dysosmobacter* may become one of the first members of its ecological guild to reach the clinic.

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Competing interests PDC is inventor on patent applications dealing with the use of gut bacteria and their components in the treatment of diseases. PDC was a co-founder of Enterosys. PDC is an Associate Editor of *eGastroenterology*.

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REFERENCES

1 Tseng C-H, Wong S, Yu J, *et al*. Development of live biotherapeutic products: a position statement of Asia-Pacific Microbiota Consortium. *Gut* 2025;74:706–13.
2 Everard A, Belzer C, Geurts L, *et al*. Cross-talk between Akkermansia muciniphila and intestinal epithelium controls diet-induced obesity. *Proc Natl Acad Sci USA* 2013;110:9066–71.
3 Plovier H, Everard A, DruartC, *et al*. A purified membrane protein from Akkermansia muciniphila or the pasteurized bacterium improves metabolism in obese and diabetic mice. *Nat Med* 2017;23:107–13.
4 Depommier C, Everard A, Druart C, *et al*. Supplementation with Akkermansia muciniphila in overweight and obese human volunteers: a proof-of-concept exploratory study. *Nat Med* 2019;25:1096–103.
5 Cani PD, Depommier C, Derrien M, *et al*. Akkermansia muciniphila: paradigm for next-generation beneficial microorganisms. *Nat Rev Gastroenterol Hepatol* 2022;19:625–37.
6 Van Hul M, Cani PD. From microbiome to metabolism: Bridging a two-decade translational gap. *Cell Metab* 2026;38:14–32.
7 Everard A, Lazarevic V, Derrien M, *et al*. Responses of gut microbiota and glucose and lipid metabolism to prebiotics in genetic obese and diet-induced leptin-resistant mice. *Diabetes* 2011;60:2775–86.
8 Forslund K, Hildebrand F, Nielsen T, *et al*. Disentangling type 2 diabetes and metformin treatment signatures in the human gut microbiota. *Nature* 2015;528:262–6.
9 Zhang X, Fang Z, Zhang C, *et al*. Effects of Acarbose on the Gut Microbiota of Prediabetic Patients: A Randomized, Double-blind, Controlled Crossover Trial. *Diabetes Ther* 2017;8:293–307.
10 Bajaj JS, Heuman DM, Hylemon PB, *et al*. Altered profile of human gut microbiome is associated with cirrhosis and its complications. *J Hepatol* 2014;60:940–7.
11 Qin N, Yang F, Li A, *et al*. Alterations of the human gut microbiome in liver cirrhosis. *Nature* 2014;513:59–64.
12 Van Hul M, Le Roy T, Prifti E, *et al*. From correlation to causality: the case of Subdoligranulum. *Gut Microbes* 2020;12:1–13.
13 Le Roy T, Van der SmissenP, PaquotA, *et al*. Dysosmobacter welbionis gen. nov., sp. nov., isolated from human faeces and emended description of the genus Oscillibacter. *Int J Syst Evol Microbiol* 2020;70.

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14 Liu C, Du M-X, Abuduaini R, *et al*. Enlightening the taxonomy darkness of human gut microbiomes with a cultured biobank. *Microbiome* 2021;9:119.
15 Li D-H, Abuduaini R, Du M-X, *et al*. Alkaliphilus flagellatus sp. nov., Butyricicoccus intestinisimiae sp. nov., Clostridium mobile sp. nov., Clostridium simiarum sp. nov., Dysosmobacter acutus sp. nov., Paenibacillus brevis sp. nov., Peptoniphilus ovalis sp. nov. and Tissierella simiarum sp. nov., isolated from monkey faeces. *Int J Syst Evol Microbiol* 2022;72.
16 Gilroy R, Ravi A, Getino M, *et al*. Extensive microbial diversity within the chicken gut microbiome revealed by metagenomics and culture. *PeerJ* 2021;9:e10941.
17 Lee C-H, Bui TPN, Petitfils C, *et al*. Novel myo-inositol to butyrate fermentation pathway in the prevalent human gut species Dysosmobacter welbionis, a bacterium associated with improved metabolic and liver health. *Gut* 2026;75:1338–52.
18 Kenny DJ, Plichta DR, Shungin D, *et al*. Cholesterol Metabolism by Uncultured Human Gut Bacteria Influences Host Cholesterol Level. *Cell Host Microbe* 2020;28:245–57.
19 Li C, Stražar M, Mohamed AMT, *et al*. Gut microbiome and metabolome profiling in Framingham heart study reveals cholesterol-metabolizing bacteria. *Cell* 2024;187:1834–52.
20 da Silva AC, Lapkin J, Yin Q, *et al*. Meta-analysis of the uncultured gut microbiome across 11,115 global metagenomes reveals a candidate signature of health. *Cell Host Microbe* 2026;34:379–92.
21 Le Roy T, Moens de Hase E, Van Hul M, *et al*. *Dysosmobacter welbionis* is a newly isolated human commensal bacterium preventing diet-induced obesity and metabolic disorders in mice. *Gut* 2022;71:534–43.
22 Hoskinson C, Dai DLY, Petersen C, *et al*. Saccharomycetes and Malassezia fungi associate with early-life gut maturation and allergic disease risk in childhood. *Nat Commun* 2026;17:5500.
23 Alderete TL, Holzhausen EA, Liang D, *et al*. Early-Life Air Pollution Exposure Is Associated with the Infant Gut Microbiome and Fecal Metabolome in the First Two Years of Life. *Res Rep Health Eff Inst* 2026;2026:1–58.
24 Jagare L, Rozenberga M, Silamikelis I, *et al*. Metatranscriptome analysis of blood in healthy individuals and irritable bowel syndrome patients. *J Med Microbiol* 2023;72.
25 Kwak S, Usyk M, Beggs D, *et al*. Sociobiome - Individual and neighborhood socioeconomic status influence the gut microbiome in a multi-ethnic population in the US. *NPJ Biofilms Microbiomes* 2024;10:19.
26 Mutoni J d, Van Hul M, Uwimana A, *et al*. Gut microbiota composition differences are associated with geographic location and age in malaria-endemic regions of Rwanda. *PLoS One* 2025;20.
27 Maghini DG, Oduaran OH, Olubayo LAI, *et al*. Expanding the human gut microbiome atlas of Africa. *Nature* 2025;638:718–28.
28 Manghi P, Bhosle A, Wang K, *et al*. Coffee consumption is associated with intestinal Lawsonibacter asaccharolyticus abundance and prevalence across multiple cohorts. *Nat Microbiol* 2024;9:3120–34.
29 Huang KD, Müller M, Sivapornnukul P, *et al*. Dietary selective effects manifest in the human gut microbiota from species composition to strain genetic makeup. *Cell Rep* 2024;43:115067.
30 Singh R, McDonald D, Hernandez AR, *et al*. Temporal nutrition analysis associates dietary regularity and quality with gut microbiome diversity: insights from the Food & You digital cohort. *Nat Commun* 2025;16:8635.
31 Petitfils C, Depommier C, Delzenne NM, *et al*. Fecal Dysosmobacter spp. concentration is linked to plasma lipidome in insulin-resistant individuals with overweight, obesity and metabolic syndrome. *Lipids Health Dis* 2025;24:203.
32 Carrizales-Sánchez AK, Tamez-Rivera O, García-Gamboa R, *et al*. Gut microbial composition and functionality of school-age Mexican population with metabolic syndrome and type-2 diabetes mellitus using shotgun metagenomic sequencing. *Front Pediatr* 2023;11.
33 Teede HJ, Khomami MB, Morman R, *et al*. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. *The Lancet* 2026;407:2329–39.
34 Yang Y, Cheng J, Liu C, *et al*. Gut microbiota in women with polycystic ovary syndrome: an individual based analysis of publicly available data. *eClinicalMedicine* 2024;77:102884.
35 Lin Y-T, Sayols-Baixeras S, Baldanzi G, *et al*. The association between the gut microbiome and 24-h blood pressure measurements in the SCAPIS study. *Commun Med (Lond)* 2025;5:276.
36 Deng Z, Mei S, Ouyang Z, *et al*. Dysregulation of gut microbiota stimulates NETs-driven HCC intrahepatic metastasis: therapeutic implications of healthy faecal microbiota transplantation. *Gut Microbes* 2025;17.
37 Saha S, Rehman L, Rehman A, *et al*. Longitudinal analysis of gut microbiome and metabolome correlates of response and toxicity with idecabtagene vicleucel. *Blood Adv* 2025;9:3429–40.
38 Lukas RV, Zhai L, Lauing KL, *et al*. Phase I Evaluation of Patients with Newly Diagnosed Glioblastoma Treated with Radiation, Nivolumab, and IDO1 Enzyme Inhibitor BMS-986205. *Clin Cancer Res* 2026;32:3476–92.
39 De Greef J, Nguyen KN, Van Hul M, *et al*. Associations between weight gain, integrase inhibitors antiretroviral agents, and gut microbiome in people living with HIV: a cross-sectional study. *Sci Rep* 2025;15.
40 Nickols WA, Kuntz T, Shen J, *et al*. MaAsLin 3: refining and extending generalized multivariable linear models for meta-omic association discovery. *Nat Methods* 2026;23:554–64.
41 Wang X, Cao D, Zhang H, *et al*. Utilizing metagenomic profiling and machine learning model to identify bacterial biomarkers for major depressive disorder. *Front Psychiatry* 2025;16:1539596.
42 Vilar Geraldi M, Dwibedi C, Jaiswal R, *et al*. Gut microbiota associates with frailty in older women. *Nat Commun* 2026;17:5925.
43 Moens de Hase E, Petitfils C, Alhouayek M, *et al*. Dysosmobacter welbionis effects on glucose, lipid, and energy metabolism are associated with specific bioactive lipids. *J Lipid Res* 2023;64.
44 Moens de Hase E, Neyrinck AM, Rodriguez J, *et al*. Impact of metformin and Dysosmobacter welbionis on diet-induced obesity and diabetes: from clinical observation to preclinical intervention. *Diabetologia* 2024;67:333–45.
45 Wong GC, Delzenne NM, Van Hul M, *et al*. Dysosmobacter welbionis J115T reduces stress-like phenotype in high-fat diet-induced obese female mice. *Benef Microbes* 2025;17:153–64.
46 Wang G, Luo Y, Song S, *et al*. Role of the Gut Microbiome in Modulating Interactions Between β-Lactam Antibiotics and Statins in Coronary Heart Disease. *J Cardiovasc Pharmacol* 2026;87:299–309.
47 Ren P, Wen X, Lin X, *et al*. Regulatory effects of leflunomide on gut microecology during IgA nephropathy treatment. *Ren Fail* 2026;48:2650023.
48 Lyu L, Fan Y, Bryrup T, *et al*. Glucocorticoid-induced changes of the gut microbiota and metabolic markers in healthy young men: Outcome of a randomized controlled trial. *Cell Rep Med* 2025;6.
49 Su J, Zhao K, Zhou X, *et al*. Early-life exposure to linezolid caused gut microbiota dysbiosis can be inherited from parents to offspring. *Chem Biol Interact* 2026;424:111863.
50 Tsai HY, Chang CI, Yeh YT, *et al*. An acetylated nobiletin derivative alleviates methylglyoxal-induced cognitive impairment and modulates gut microbiota. *J Sci Food Agric* 2026.
51 Mahalak KK, Narrowe AB, Liu L, *et al*. The ex vivo effects of ethanolic extractions of black cumin seed, turmeric root, and Ceylon cinnamon bark on the human gut microbiota. *PLoS One* 2025;20.
52 Guo S, Ma T, Kwok L-Y, *et al*. Effects of postbiotics on chronic diarrhea in young adults: a randomized, double-blind, placebo-controlled crossover trial assessing clinical symptoms, gut microbiota, and metabolite profiles. *Gut Microbes* 2024;16.
53 Burakova I, Smirnova Y, Morozova P, *et al*. The effect of short-term consumption of Bifidobacterium bifidum on the gut microbiome of obese individuals. *Exp Biol Med* 2026;251:10894.
54 Khitrov AA, Burakova IY, Smirnova YD, *et al*. Effect of a Combination of Prebiotic Supplements Based on Fucus and Kelp on the Gut Microbiome of Mice with Induced Inflammation. *Microorganisms* 2026;14:592.
55 Bandow B, Shaaban ES, Rajakaruna S, *et al*. Diet Supplementation with Pomegranate Fruit Alters Distal Gut Microbiota of Healthy Female College Students. *Microorganisms* 2025;13:305.
56 Wei Y-Y, Zhang Y-N, Wang H, *et al*. Mori fructus aqueous extracts attenuate carbon tetrachloride-induced renal injury via the Nrf2 pathway and intestinal flora. *Ecotoxicol Environ Saf* 2022;245.
57 Rong X, Shu Q. Modulating butyric acid-producing bacterial community abundance and structure in the intestine of immunocompromised mice with neutral polysaccharides extracted from Codonopsis pilosula. *Int J Biol Macromol* 2024;278.
58 Tabish RW, Lin Y, Rochell SJ, *et al*. Jejunal histopathology, metagenome, and mucosal transcriptome of broilers after an enteric challenge and fed diets with different fiber types and concentrations. *Poult Sci* 2026;105:107215.
59 Jadhav VV, Fasina Y, Omaliko PC, *et al*. Effect of dietary fat source on the composition of the cecal microbiome in maturing broiler chicken. *Front Microbiol* 2024;15:1462757.
60 Utkina I, Fan Y, Willing BP, *et al*. Metabolic modeling of microbial communities in the chicken ceca reveals a landscape of competition and co-operation. *Microbiome* 2025;13:248.
61 Kou X, Liu Y, Xiang F, *et al*. Insights into the Donkey Hindgut Microbiome Using Metagenome-Assembled Genomes. *Animals (Basel)* 2024;14:3625.

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62 Wang Y, Liu W, Li A, *et al*. Changes in the growth performance, serum biochemistry, rumen fermentation, rumen microbiota community, and intestinal development in weaned goats during rumen-protected methionine treatment. *Front Vet Sci* 2024;11:1482235.
63 Pérez-Pérez L, Arguello H, Cobo-Díaz JF, *et al*. From predisposition to recovery: field evidence of interactions between the gut microbiota and Brachyspira hyodysenteriae infection. *Vet Res* 2026;57:25.
64 Maaskant A, Lee D, Ngo H, *et al*. AI for rapid identification of major butyrate-producing bacteria in rhesus macaques (Macaca mulatta). *Anim Microbiome* 2025;7:39.
65 Sola L, Candeliere F, Busi E, *et al*. A genomic atlas of gut clostridia: phylogeny, butyrate, and propionate production. *Front Microbiol* 2026;17:1761627.
66 Dowden RA, Kerkhof LJ, Wisniewski PJ, *et al*. Temporal changes in the mouse gut bacteriota influenced by host sex, diet, and exercise. *J Appl Physiol* 2024;137:1374–88.
67 Gonzalez E, Lee MD, Tierney BT, *et al*. Spaceflight alters host-gut microbiota interactions. *NPJ Biofilms Microbiomes* 2024;10:71.
68 Degraeve AL, Cutignano A, Piscitelli F, *et al*. Changes in levels of endocannabinoidome mediators in mice with cancer cachexia: links with steatosis and gut microbial dysbiosis. *BJC Rep* 2026;4:7.
69 Zhang H, Qiao W, Tai D, *et al*. Taurine attenuates arsenic-induced neurotoxicity through modulation of gut microbiota structure. *Psychopharmacology (Berl)* 2026.
70 Chen L, Wang S, Cai C, *et al*. Exposing Drosophila to wheat-derived microbes enhances its immunity and survival under simulated microgravity. *Life Sci Space Res (Amst)* 2026;48:88–98.

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