

Can the Gut Predict Parkinson's Disease? The Emerging Role of Microbiome Screening Years Before Symptoms Appear

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ABSTRACT

Background: Parkinson's disease is increasingly understood as a multisystem disorder in which gastrointestinal dysfunction, inflammation, microbial dysbiosis and altered host-microbe metabolism may appear before classical motor symptoms.

Objective: This review synthesizes recent evidence published on gut microbial signatures, short-chain fatty acids, inflammatory mechanisms, microbial functional pathways, machine-learning classifiers and clinical translation challenges.

Methods: This narrative review considered recent studies published mainly from 2021 to 2026, searching concepts included: "Parkinson's disease," "gut microbiome," "prodromal Parkinson's," "GBA1," "REM sleep behavior disorder," "metagenomics," "short-chain fatty acids," "microbial biomarkers," "early diagnosis," and "microbiota-gut-brain axis."

Results: The strongest recent evidence comes from asymptomatic GBA1 variant carriers, whose microbiome profiles resemble an intermediate state between healthy controls and diagnosed Parkinson's disease.

Conclusion: A combined screening framework using stool metagenomics, microbial metabolites, prodromal symptoms and genetic risk could eventually allow earlier identification of individuals most likely to benefit from monitoring or preventive intervention.

Keywords: parkinson's disease, gut microbiome, microbiota-gut-brain axis; gba1, rem sleep behavior disorder

1. INTRODUCTION

Parkinson's disease is the second most common neurodegenerative disorder and a growing public health burden. Global estimates show that more than 8.5 million people were living with Parkinson's disease in 2019, and prevalence has doubled over the past 25 years [1]. The clinical diagnosis is still mainly based on motor symptoms such as bradykinesia, rigidity, tremor and postural instability. Yet by the time motor features are apparent, neurodegeneration is already well established. This creates a

major therapeutic problem: disease-modifying treatments are most likely to work before irreversible dopaminergic neuronal loss becomes advanced [2]. The gut has become one of the most important biological windows into early Parkinson’s disease. Constipation, altered intestinal motility and other gastrointestinal symptoms can precede motor diagnosis by years. Recent microbiome studies suggest that the gut is not merely affected by Parkinson’s disease, but may participate in disease initiation, progression, immune activation and α -synuclein-related pathology [3]. Large metagenomic studies have reported widespread dysbiosis in Parkinson’s disease, involving altered microbial species, genes and functional pathways. More importantly, newer evidence indicates that Parkinson’s-like microbial signatures may already be detectable in prodromal or genetically at-risk individuals before clinical diagnosis. This review focused on the hypothesis that gut microbiome screens could identify Parkinson’s disease risk years before symptoms appear [4]. It evaluates the current evidence, biological plausibility, biomarker candidates, limitations and translational requirements needed before such screening can enter routine clinical or population-level use.

2. MATERIALS & METHODS

This narrative review considered recent studies published mainly from 2021 to 2026, prioritizing human studies, systematic reviews, metagenomic analyses, multi-cohort studies, machine-learning biomarker studies and translational reviews. Search concepts included: “Parkinson’s disease,” “gut microbiome,” “prodromal Parkinson’s,” “GBA1,” “REM sleep behavior disorder,” “metagenomics,” “short-chain fatty acids,” “microbial biomarkers,” “early diagnosis,” and “microbiota–gut–brain axis [5].” Emphasis was placed on studies with large cohorts, independent validation, functional profiling or clinical relevance.

3. CURRENT EVIDENCE LINKING GUT MICROBIOME ALTERATION WITH PARKINSON’S DISEASE

Recent evidence strongly supports the presence of altered gut microbiota in Parkinson’s disease. A large shotgun metagenomic study by Wallen et al. enrolled 490 Parkinson’s disease patients and 234 controls and found that more than 30% of tested species, genes and pathways differed between groups, suggesting broad dysbiosis rather than a single-microbe abnormality [6]. The reported changes included organisms and pathways linked to inflammation, neuroactive metabolites, xenobiotic metabolism and microbial ecology. A particularly important development came from studies of prodromal Parkinson’s disease. Huang et al. studied early Parkinson’s disease, REM sleep behavior disorder, first-degree relatives of REM sleep behavior disorder patients and healthy controls. The authors reported that gut microbiota composition was significantly altered in early Parkinson’s disease and REM sleep behavior disorder, with depletion of butyrate-producing bacteria and enrichment of pro-inflammatory taxa such as *Collinsella*. Random forest modelling identified 12 microbial markers that distinguished REM sleep behavior disorder from controls [7]. This supports the idea that gut dysbiosis may emerge before classical Parkinsonian motor symptoms. The strongest recent support for preclinical screening comes from Menozzi et al. in 2026. The study compared gut microbiomes from 271 Parkinson’s disease patients, 43 disease-free GBA1 variant carriers and 150 healthy controls. GBA1 carriers without Parkinson’s disease showed an intermediate microbiome pattern between healthy controls and diagnosed Parkinson’s disease, and the authors identified 176 species differing between Parkinson’s disease and healthy controls. They also proposed a 16-species Parkinson’s Disease Microbiome Score, called PDMS-16, as a proof-of-concept microbial risk score. Together, these studies suggest that gut microbiome screening is moving from descriptive association toward risk prediction. Still, the field remains early. Most studies are cross-sectional, and few have confirmed whether microbiome-positive but symptom-free individuals later convert to Parkinson’s disease.

Table 01: Recent Evidence Supporting Gut Microbiome Screening in Parkinson’s Disease

Study / Year	Design and Population	Main Finding	Screening Relevance
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Wallen et al., 2022	Shotgun metagenomics; 490 PD, 234 controls	More than 30% of tested species, genes and pathways differed in PD	Supports broad microbial dysbiosis as a disease-associated signature
Huang et al., 2023	Early PD, RBD, RBD first-degree relatives, controls	RBD and early PD showed altered microbiota; butyrate producers depleted; Collinsella enriched	Supports microbiome changes in prodromal stages
Palacios et al., 2023	Metagenomic study of PD and prodromal features	PD and prodromal PD features showed similar microbiome changes	Supports early biomarker potential
Zhao et al., 2024	Network-based meta-analysis; 550 PD, 456 controls	Faecalibacterium, Roseburia and Coprococcus_2 reduced; Akkermansia and Bilophila increased	Supports reproducible microbial biomarker discovery
Menozzi et al., 2026	271 PD, 43 asymptomatic GBA1 carriers, 150 controls	GBA1 carriers showed intermediate PD-like microbiome signature	Strongest evidence for pre-symptomatic microbial risk signal
Zhang et al., 2026	Drug-naïve early PD, essential tremor and controls	Microbiota + SCFA model distinguished early PD from controls and essential tremor	Supports stool-based differential diagnostic models

The gut microbiome may contribute to Parkinson’s disease through several overlapping pathways. No single pathway fully explains Parkinson’s disease, but the convergence of gut barrier dysfunction, microbial metabolites, neuroinflammation and α -synuclein biology make the gut a plausible early detection site.

4. INTESTINAL BARRIER DYSFUNCTION AND INFLAMMATION

Gut dysbiosis can reduce mucosal integrity and increase intestinal permeability. When barrier function weakens, bacterial products such as lipopolysaccharide may interact with immune cells and promote local and systemic inflammation. An author [8] reported altered short-chain fatty acids, calprotectin and gut barrier-related markers in Parkinson’s disease, supporting a link between dysbiosis, inflammation and gut barrier biology. Chronic immune activation may then contribute to neuroinflammatory processes relevant to Parkinson’s disease progression.

SHORT-CHAIN FATTY ACIDS AND BUTYRATE-PRODUCING BACTERIA

Short-chain fatty acids, especially butyrate, help maintain epithelial barrier function, regulate inflammation and influence gut–brain signaling. Several studies report reductions in butyrate-producing taxa such as Faecalibacterium, Roseburia and Coprococcus-related organisms in Parkinson’s disease. An author [9] found that butyrate-producing genera including Faecalibacterium, Roseburia and Coprococcus_2 were reduced in Parkinson’s disease, while Akkermansia and Bilophila were increased.

This pattern supports the idea that loss of anti-inflammatory microbial functions may be more important than the presence or absence of any single organism.

α-SYNUCLEIN, ENTERIC NERVOUS SYSTEM AND BODY-FIRST PARKINSON'S DISEASE

The “body-first” Parkinson’s disease model proposes that pathology may begin outside the brain, including in the gut or autonomic nervous system, before spreading centrally. REM sleep behavior disorder, constipation and autonomic dysfunction are clinically consistent with this model [10]. The finding that REM sleep behavior disorder patients already show Parkinson’s-like gut dysbiosis strengthens the hypothesis that microbiome changes may precede motor diagnosis.

MICROBIAL METABOLISM OF NEUROACTIVE COMPOUNDS

The gut microbiome can influence bile acids, amino acid metabolites, neurotransmitter-related pathways and xenobiotic metabolism. In the 2026 Nature Medicine study, Parkinson’s disease microbiomes showed altered functional modules involving neurotransmitter metabolism, amino acid degradation, p-cresol degradation, riboflavin biosynthesis and carbohydrate degradation. These functional shifts may be highly relevant because clinical screening based only on microbial names may miss disease-relevant metabolic activity [11].

5. CANDIDATE MICROBIAL AND METABOLIC BIOMARKERS

The most reproducible Parkinson’s disease microbiome finding is not one organism, but a pattern: reduction of fiber-fermenting, SCFA-producing and anti-inflammatory taxa, combined with enrichment of taxa linked to mucin degradation, inflammation or altered bile acid and amino acid metabolism. An author [12] added an important metabolite layer by combining gut microbiota and fecal short-chain fatty acids. In drug-naïve early Parkinson’s disease, the combined model distinguished Parkinson’s disease from healthy controls with a validation AUC of 0.864 and distinguished Parkinson’s disease from essential tremor with a validation AUC of 0.825. These findings are clinically useful because essential tremor is a common diagnostic mimic of tremor-dominant Parkinson’s disease.

Table 02: Candidate Gut Microbiome Biomarkers for Parkinson’s Disease Risk Screening

Biomarker Class	Examples Reported in Recent Studies	Direction in PD / Risk State	Possible Biological Meaning	Clinical Potential
Butyrate-producing bacteria	Faecalibacterium, Roseburia, Coprococcus-related taxa	Often decreased	Reduced SCFA production, weaker gut barrier, more inflammation	Useful as part of risk panel
Pro-inflammatory or mucin-associated taxa	Collinsella, Akkermansia, Bilophila	Often increased in some studies	Mucosal stress, inflammation, bile acid changes	Needs population-specific validation
Bifidobacterium-related taxa	Bifidobacterium longum, B. dentium	Increased in some PD metagenomic studies	May reflect medication, diet, host metabolism or disease ecology	Interpret carefully; not always protective
SCFAs	Butyrate, acetate, propionate; branched-chain SCFAs such as	Altered	Metabolic readout of microbial function	Stronger when combined with taxa

	isovaleric/isobutyric acids			
Functional modules	Neurotransmitter metabolism, riboflavin biosynthesis, carbohydrate degradation, p-cresol metabolism	Altered	Captures microbial activity, not just composition	High value for future precision screening
Composite microbial scores	PDMS-16	Higher PD-like score in risk profile	Species-based risk stratification	Promising but needs longitudinal validation

6. THE CASE FOR SCREENING BEFORE SYMPTOMS

A gut microbiome screen has several theoretical advantages. It is non-invasive, repeatable, relatively acceptable to patients and capable of capturing environmental, dietary, immune and metabolic signals. Unlike single genetic tests, microbiome profiles are dynamic and may reflect modifiable risk pathways [13]. The 2026 GBA1-carrier study is especially important because it directly addresses risk before clinical symptoms. The authors found that healthy GBA1 carriers without Parkinson's disease had microbiome alterations in the same direction as Parkinson's disease patients, although generally less severe, suggesting an intermediate biological state. In addition, the proposed PDMS-16 score identified a subgroup of healthy controls with clinical profiles closer to Parkinson's disease, including more depression, anxiety, autonomic dysfunction and constipation. However, a screening test must do more than distinguish diagnosed Parkinson's disease from controls [14]. It must predict future conversion in asymptomatic individuals. This requires longitudinal studies with repeated stool sampling, standardized diet data, medication history, prodromal clinical assessment, genetic risk stratification and eventual conversion outcomes.

7. VALUE-ADDED PROPOSAL: A MULTI-LAYER PARKINSON'S MICROBIOME RISK INDEX

A clinically useful microbiome screen should not rely only on bacterial abundance. The following value-added model is proposed: Parkinson's Microbiome Risk Index = Taxonomic Dysbiosis + Functional Dysbiosis + Metabolite Dysbiosis + Prodromal Clinical Load + Genetic Risk Modifier. This model would classify individuals into low-, moderate- and high-risk categories. Each component adds different information:

- Taxonomic dysbiosis: species or genera associated with Parkinson's disease.
- Functional dysbiosis: microbial genes and pathways linked to inflammation, neurotransmitter metabolism, bile acids, amino acid metabolism and carbohydrate fermentation.
- Metabolite dysbiosis: SCFAs, bile acids, p-cresol, amino acid derivatives and inflammatory stool markers.
- Clinical prodromal load: constipation, REM sleep behavior disorder, depression, hyposmia, autonomic dysfunction and subtle motor changes.
- Genetic risk modifier: GBA1, LRRK2 and family history [15].

This framework could reduce false positives by requiring convergence across microbial, metabolic and clinical signals rather than relying on one stool result.

Table 03: Proposed Parkinson's Microbiome Risk Index

Component	Suggested Input	Weighting Principle	Reason for Inclusion
Taxonomic signature	PD-associated species/genera; PDMS-like score	Moderate	Captures microbial composition
Functional pathway signature	KEGG/GMM/GBM modules; metagenomic pathways	High	More stable than taxonomy across populations
Metabolite signature	SCFAs, bile acids, amino acid metabolites	High	Reflects biological activity
Inflammation/barrier markers	Calprotectin, zonulin, LPS-binding markers	Moderate	Links gut dysbiosis to host response
Prodromal clinical score	RBD, constipation, hyposmia, autonomic dysfunction	High	Improves disease specificity
Genetic risk	GBA1, LRRK2, family history	Contextual modifier	Identifies high-risk populations
Longitudinal change	Repeat testing over 12–36 months	Very high	Distinguishes stable variation from disease trajectory

8. CLINICAL TRANSLATION CHALLENGES

Despite strong promise, gut microbiome screening for Parkinson’s disease is not ready for routine clinical use. Several challenges must be solved. First, most studies are cross-sectional. Cross-sectional data can show association but cannot prove that microbiome changes predict conversion to Parkinson’s disease. Second, the gut microbiome is strongly affected by diet, geography, age, sex, constipation, antibiotics, probiotics, bowel transit time and medications. These variables can create false biomarker signals [16]. Third, sequencing methods vary across studies, including 16S rRNA sequencing, shotgun metagenomics, different DNA extraction methods and different bioinformatics pipelines. An author [17] emphasized that replicable biomarkers across populations remain difficult because of dataset and batch effects. Fourth, ethical issues are significant. A person said that their stool profile suggests elevated Parkinson’s disease risk may experience anxiety, insurance concerns or unnecessary medical testing, especially when no proven preventive drug exists. Therefore, microbiome screening should initially be used in research settings or specialist high-risk clinics rather than direct-to-consumer testing.

Table 04: Barriers to Clinical Implementation and Proposed Solutions

Barrier	Why It Matters	Proposed Solution
Cross-sectional evidence	Cannot prove prediction of future PD	Longitudinal cohorts with conversion outcomes
Population variability	Diet/geography alter microbiome	Multi-country reference ranges and local calibration
Medication confounding	Levodopa, laxatives, and antibiotics affect microbes	Include drug-naïve and medication-adjusted analyses
Sequencing variation	Results differ by lab and method	Standardized stool collection, extraction and pipelines
False positives	PD-like microbiome may not mean future PD	Combine microbiome with clinical and genetic risk

Ethical risk disclosure	No confirmed preventive cure yet	Genetic-style counseling and risk communication
Cost and access	Sequencing may be expensive	Develop tiered screening: clinical score → stool test → advanced omics
Regulatory uncertainty	Biomarker tests need validation	Prospective diagnostic accuracy and utility trials

9. MICROBIOME-BASED PREVENTION AND INTERVENTION POSSIBILITIES

If gut microbiome signatures do identify early Parkinson's disease risk, prevention could move beyond detection. Potential interventions include dietary fiber optimization, Mediterranean-style dietary patterns, prebiotics, probiotics, symbiotic, postbiotics, anti-inflammatory nutrition and fecal microbiota transplantation. Current reviews describe microbiome-targeted therapies as promising but not yet established as disease-modifying treatments [18]. At present, diet and lifestyle modification are the safest translational options. The 2026 GBA1-carrier study reported that individuals with healthier diet profiles were less likely to show high-risk microbiome patterns, although this remains associative rather than causal. Future prevention trials should test whether modifying the gut microbiome changes prodromal markers, inflammatory markers, α -synuclein biomarkers or conversion risk [19].

10. FUTURE RESEARCH AGENDA

The next stage of research should focus on prediction, causality and clinical utility. Priority areas include:

- Longitudinal high-risk cohorts: Follow GBA1 carriers, LRRK2 carriers and idiopathic REM sleep behavior disorder patients for 5–10 years.
- Repeated stool sampling: Capture microbiome trajectory rather than single time-point variation.
- Multi-omics integration: Combine metagenomics, metatranscriptomics, metabolomics, inflammatory markers and host genetics.
- Global validation: Include Asian, African, European, Middle Eastern and Latin American cohorts.
- Standardized bioinformatics: Use reproducible open-source pipelines with batch-effect correction.
- Clinical utility trials: Test whether microbiome screening improves early referral, diagnosis, monitoring or prevention.
- Mechanistic studies: Identify whether microbes drive pathology or reflect early disease physiology.
- Ethical frameworks: Develop guidelines for communicating preclinical neurodegenerative risk.

11. CONCLUSION

Gut microbiome screening is one of the most promising non-invasive strategies for identifying Parkinson's disease risk before motor symptoms appear. Recent studies show that Parkinson's disease is associated with broad microbial dysbiosis, altered short-chain fatty acid biology, inflammatory gut signatures and functional microbial pathway disruption. The strongest recent evidence comes from asymptomatic GBA1 variant carriers, whose microbiome profiles resemble an intermediate state between healthy controls and diagnosed Parkinson's disease. This suggests that gut microbial changes may be detectable during a preclinical window. However, microbiome screening should not yet be treated as a stand-alone diagnostic test. Its future clinical value will depend on longitudinal validation, standardized sequencing, multi-omics integration, careful adjustment for confounders and ethical risk communication. A combined screening framework using stool metagenomics, microbial metabolites,

prodromal symptoms and genetic risk could eventually allow earlier identification of individuals most likely to benefit from monitoring or preventive intervention. The field is now ready to move from association studies toward predictive, mechanistic and intervention-based research.

AUTHOR CONTRIBUTION STATEMENT

Li Wei & Zhang Min: Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work, Drafting the work or reviewing it critically for important intellectual content; AND Final approval of the version to be published, and Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data Availability: The datasets and information used in this study are publicly available from the sources cited within the article. No proprietary or restricted data were used.

Trial Registration: Trial registration is not applicable to this study, as it does not involve clinical trials, human participants, or experimental interventions.

Generative AI Disclosure: Generative artificial intelligence tools were not used for generating research data, conducting analyses, or producing scientific conclusions in this study. The authors take full responsibility for the content and accuracy of the manuscript.

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SUPPLEMENTAL MATERIAL

No supplementary files, datasets, figures, or additional materials are associated with this study. Any information necessary to understand and evaluate the work is included in the main article.