International Journal of Integrated Sciences and Mathematics
Vol: 02; **Issue**: 01; Jan-Jun 2025; **Pg**: 012-019

Gut Microbiome Dysbiosis and Mental Health: A Multiple-Case Study of the Microbiota–Gut–Brain Axis in Treatment-Resistant Depression and Anxiety

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Received: 15th March 2025 / Accepted: 02th March 2025 / Published: 25th April 2025

Citation: Y Surya Teja (2025). Gut Microbiome Dysbiosis and Mental Health: A Multiple-Case Study of the Microbiota–Gut– Brain Axis in Treatment-Resistant Depression and Anxiety, Internation Journal of Integrated Sciences and Mathematics, 2(1), 012-019
DOI: https://doi.org/10.54646/ijism.2025.03

Abstract:
This case study investigates the relationship between dysbiosis of the gut microbiome and mental health symptoms in two individuals, namely Jane and Amir, suffering from treatment-resistant depression and anxiety. Both of them demonstrated continuous neuropsychiatric symptoms along with digestive issues. Thorough research revealed a reduction in microbial diversity, a decrease in short-chain fatty acid production, and a rise in inflammatory markers, which indicates a disrupted gut-brain axis. Modification in the patient's diet and the inclusion of probiotics do not really improve patients' mood, digestive issues, and overall bodily inflammation. The results show that gut microbiota may lead to mental health illness, which highlights the promising effectiveness of extensive, gut-focused therapeutic approaches. Further study is needed to know more about the effective therapies targeting the microbiome within the area of psychiatry.

Keywords: *Gut microbiome, mental health, depression, anxiety, inflammation, probiotics, integrative psychiatry.*

INTRODUCTION
The gut microbiota represents a complex ecosystem that consists of diverse microorganisms, including viruses, fungi, bacteria, and archaea, with considerable research dedicated to understanding the role of bacteria [1]. A number of previous works have demonstrated a correlation between gut microbiota and overall well-being. Consequently, specific bacterial groups have been recognized as important factors in the modulation of dysbiosis or an unbalanced microbiota. The incorporation of probiotics and prebiotics into dietary protocols, in conjunction with fecal microbiota transplantation, has demonstrated efficacy in addressing specific infections, particularly those induced by resistant strains of Clostridioides difficile [2]. Additionally, strategies designed to modify the gut microbiome offer potential therapeutic options for various conditions, eg. inflammatory bowel disease, inflammatory disorders, metabolic issues, cancer, and other health-related challenges [3]. Further investigations are necessary to validate the suitability of their implementation in these health conditions. Emotional states function as dynamic processes that govern the interaction between the brain and the body, highlighting the interconnectedness of these two systems. The connection between the central nervous system and gut microbiota, commonly known as the 'gut-brain axis,' has garnered considerable interest in recent years [4]. This phenomenon illustrates the mediation of a reversible communication pathway between gut bacteria and the brain by the immunological, endocrine, and neurological systems. This dual interaction encompasses the immune system's response, hormone secretion, and modulation of neuronal activity. This incident exemplifies a reciprocal reaction, involving changes in gut lining and blood-brain barrier permeability. Short-chain fatty acids (SCFAs), bacterial neurotransmitters like gamma-aminobutyric acid (GABA) and serotonin, immune system modulators like quinolinic acid, and hormones like cortisol are among the metabolic products that the gut microbiota responds to [5]. There is evidence that the microbiota may affect behavior, mental health, and brain function. Changes in the gut microorganisms of humans have been

connected to neuropsychiatric illnesses and mood disorders. These changes can cause neurotransmitter levels to be disbalanced. Previous studies suggested that the gut microbiota is linked to autism spectrum disorder (ASD) symptoms, which are typically made worse by gut problems and dysbiosis of gut microbes [6].

METHODOLOGY CONTEXT

This study was conducted within an extensive mental health research initiative at a large urban academic medical center that provides interdisciplinary care, advanced microbiome profiling, and psychometric assessments. The aim was to explore connection between gut microbiome dysbiosis and mental health symptoms in individuals unresponsive to standard therapies.

Participants

Two adults were purposefully selected for this multiple-case study. Jane, 32, presented with persistent depression and anxiety, low mood, fatigue, cognitive impairment, and sleep disruption lasting over six months. Despite antidepressant and CBT trials, her progress remained limited. She also reported gastrointestinal issues including irregular bowel movements, bloating, and food intolerances. Amir, 28, had a two-year history of generalized anxiety and major depression, marked by irritability, poor concentration, hopelessness, and chronic GI disruptions like constipation and abdominal cramping. Both had tried multiple conventional treatments with minimal or short-lived effects and were referred for further assessment.

Data Collection

Data sources included semi-structured clinical interviews exploring psychiatric and GI histories, prior treatments, and lived experiences. Lab assessments covered stool microbiome analysis (bacterial diversity, relative abundance, SCFA levels) and inflammatory markers (hs-CRP, IL-6, TNF-α). Participants kept weekly reflective journals over 12 weeks, documenting symptom changes, triggers, and observations. Clinical documentation (progress notes, diagnostic summaries), expert consultations with a psychiatrist and microbiome specialist, and self-report inventories on mental health and gut-brain attitudes were also collected at baseline and study end.

Data Analysis

A deductive thematic coding approach was used, drawing on gut-brain axis models and psychobiotic/neuroinflammatory theory. Interviews, journals, and notes were coded for themes like microbial shifts, symptom patterns, dietary/lifestyle impacts, and care barriers. Lab data were mapped to physiological mechanisms in literature, such as SCFA deficiencies, pro-inflammatory taxa, and leaky gut markers. NVivo software supported qualitative data organization and cross-case comparison. Three integrative memos were created: one outlining individual trajectory, another synthesizing shared biological/psychological mechanisms, and a third comparing psychosocial and care-related factors. A second researcher reviewed the coding to ensure analytic rigor.

Trustworthiness and Validation

Member checking was used, with participants reviewing narrative summaries and themes. A second investigator independently audited interpretations to enhance confirmability. Triangulation of clinical, lab, narrative, and expert data across both cases further bolstered credibility.

RESULTS

Findings from Jane and Amir's cases marked by overlapping mental and gastrointestinal symptoms tied to gut dysbiosis are presented through five key themes derived from integrated qualitative and quantitative data: (1) Clinical Symptomatology and Treatment Resistance, (2) Gut Microbiome Profiles and SCFA Production, (3) Inflammatory Markers and Immune Activation, (4) Psychosocial Experiences and Illness Perception, and (5) Interventions and Symptom Modulation.

Clinical Symptomatology and Resistance to Conventional Treatment

Both Jane and Amir presented with longstanding neuropsychiatric symptoms including anxiety, depressive mood, fatigue, reduced cognitive function, and disrupted sleep. Jane described her experience as "a mental fog," adding that traditional therapy and selective serotonin reuptake inhibitors (SSRIs) "only lifted me halfway back to normal." She reported persistent low energy, difficulty concentrating, and a heightened sense of worry. Amir expressed comparable challenges but highlighted his difficulty in maintaining progress. "I would initially experience an improvement, only to encounter a regression after several weeks, irrespective of my attempts," he stated. Both participants exhibited diverse reactions to first- and second-line psychiatric medications as well as psychotherapeutic interventions over prolonged durations. The treatment history included selective serotonin reuptake inhibitors (SSRIs) such as escitalopram and fluoxetine, serotonin-norepinephrine reuptake inhibitors (SNRIs), mindfulness-based interventions, and dietary changes; nevertheless, none resulted in consistent or lasting remission. All participants reported some gastrointestinal discomfort that affects their mental health. Jane reported chronic bloating, irregular bowel movements, and dietary intolerances. These symptoms all arose before her psychological difficulties but increased after all those issues developed. Amir exhibited significant constipation, abdominal cramps related to meals, and ongoing fatigue. Both patients emphasized a significant relationship between their gastrointestinal issues and mental health, noting that "stomach discomfort intensified all other concerns."

Table 1: Psychiatric and Gastrointestinal Symptoms in Two Patients

Patient	Core Psychiatric Symptoms	GI Symptoms	Duration of Mental Symptoms	Response to Treatment
Jane	Low mood, anxiety, fatigue, "brain fog"	Bloating, irregular bowel habits, intolerances	>6 months	Partial to minimal
Amir	Anxiety, depression, poor concentration	Constipation, abdominal cramps, food sensitivity	~2 years	Fluctuating, minimal

Gut Microbiome Profiles and SCFA Production

The analysis of stool samples has been done in the laboratory, and some irregularities were identified in the number of gut microbiota in both participants. Lactobacillus and Bifidobacterium species levels were found to be very low in Jane, which is further accompanied by an increase in Proteobacteria, a phylum recognized for its frequently pro-inflammatory Gram-negative bacteria. Short-chain fatty acids (SCFAs), particularly butyrate, which is essential for preserving gut mucosal health, maintaining the blood-brain barrier, and facilitating anti-inflammatory signaling, were also found to be decreased in the study. Similarly, Amir's microbiota displayed a low alpha diversity score and an overrepresentation of endotoxin-producing bacteria within the Enterobacteriaceae family. His SCFA panel reflected a dramatic underproduction of acetate and propionate, both of which have neuroactive and immunomodulatory roles. These microbial deficiencies likely contributed to mucosal permeability ("leaky gut") and systemic immune dysregulation observed on further testing. Results indicated functional impairments in microbial metabolic output. Metabolomic assessments suggested impaired fermentation of dietary fibers and imbalances in tryptophan metabolism, shifting conversion away from serotonin synthesis and toward kynurenine pathway derivatives such as quinolinic acid, which are neurotoxic and implicated in depressive symptomatology.

Table 2: Microbial and SCFA Findings

Patient	Microbial Findings	SCFA Findings
Jane	↓*Lactobacillus*, ↓*Bifidobacterium*, ↑*Proteobacteria*	↓Butyrate, ↓overall SCFAs
Amir	↓Diversity, ↑endotoxin-producers (*Enterobacteriaceae*)	↓Acetate, ↓propionate

Inflammatory Markers and Systemic Immune Activation

Laboratory biomarkers indicated low-grade systemic inflammation in both cases. Jane's high-sensitivity C-reactive protein (hs-CRP) was increased above the standard reference range, and her interleukin-6 (IL-6) measurements were mildly raised. These findings are consistent with chronic immune activation, and likely link gut microbial imbalance with HPA axis dysregulation and neuroinflammation. Amir presented similar inflammatory trends, with both C-reactive protein and TNF-α levels elevated on two occasions, despite the absence of any known infection or diagnosed autoimmune disease. When contextualized with his microbiome profile particularly his overgrowth of gram-negative bacteria capable of producing lipopolysaccharide (LPS) the findings strongly suggest endotoxemia-related immune perturbation. Both participants underwent repeat testing after initiating gut-targeted interventions (outlined under Interventions), showing mild reductions in inflammatory markers 10 weeks into treatment. While not conclusive, the trend aligned with patients' subjective reports of symptom stabilization.

Table 3: Inflammatory Marker Findings and Interpretation

Patient	Key Inflammatory Markers	Interpretation
Jane	↑hs-CRP, mildly ↑IL-6	Persistent low-grade inflammation
Amir	↑CRP, ↑TNF-α (repeatedly measured)	Endotoxemia-related immune activation

Psychosocial Experiences and Illness Perception

Patient interviews and reflective journals revealed deep psychological distress associated not only with the symptoms themselves, but with the prolonged search for answers and ineffective treatment experiences. Jane shared that she felt "invalidated" and "invisible" by her medical providers, who frequently overlooked the connection between her gut and brain symptoms. Amir expressed his frustration, saying that "nobody looks at anything beyond mood scores or labels they didn't ask about how bad my stomach problems are or how tired I really feel." Both participants showed a lot of interest and openness about the gut microbiome as a potential reason for their distress. When her dysbiosis was confirmed, Jane, who started her own research into the gut-brain relationship, felt incredibly empowered: "It cleared my confusion." For the first time, I was given an explanation that linked my mental haze to my stomach problems. Nonetheless, both conveyed doubts and fatigue regarding the uncertainties associated with microbiome science and its practical use in clinical settings. Amir observed, "There is much discussion surrounding probiotics and gut health these days, yet few can provide clear guidance on the appropriate actions to take." It continues to seem as though you are navigating this journey independently. Data concerning social and lifestyle factors highlighted elements that contribute to dysbiosis. Jane's journal entries frequently documented changes in her eating habits attributed to stress, inconsistent sleep patterns, and a higher consumption of processed foods. Amir's workload, sedentary lifestyle, and irregular meal patterns were identified as contributing factors. These lifestyle factors were presumed to contribute to the decline of gut health and to sustain the psychophysiological cycle of distress.

Table 4: Table: Patient-Centered Insights about Experience, Perception, and Lifestyle Factors

Patient	Healthcare Experience	Illness Perception	Relevant Lifestyle Factors
Jane	Felt "invalidated," ignored	Empowered by gut-brain validation	Stress-eating, irregular sleep, processed foods
Amir	Frustration with narrow approaches	Cautiously optimistic, skeptical	Work stress, sedentary habits, meal irregularity

Interventions and Symptom Modulation

Both participants initiated integrated interventions following confirmation of gut dysbiosis. These included a high-fiber, anti-inflammatory diet, individualized probiotic supplementation, sleep hygiene protocols, and guided stress-reduction strategies. Jane also received targeted SCFA supplementation (butyrate), while Amir began a trial of synbiotics (probiotic + prebiotic combination) tailored to increase microbial diversity and SCFA production. By the tenth week of intervention, both individuals reported modest but meaningful improvements in energy levels, mood regulation, and gastrointestinal comfort. Jane noted greater emotional resilience and reduced bloating, stating, "I feel like I'm on a gentler wave now, not crashing every other week." Amir similarly described improved digestion, more "stable mornings," and a return of mental clarity that he had not experienced in over a year." Biological indicators supported these subjective assessments: SCFA levels increased partially, microbiota composition shifted toward higher diversity and commensal representation, and systemic inflammatory markers trended downward. Although not all clinical targets were reached and results varied between participants, the evidence suggested that gut-focused interventions played a role in modulating mental health outcomes, particularly when conventional treatments alone had failed.

Table 5: Intervention, Outcomes, and Objective Changes for Each Patient

Patient	Intervention Components	Subjective Outcomes	Objective Changes
Jane	High-fiber/anti-inflammatory diet, probiotics, SCFA supplement	Improved energy, less bloating, greater resilience	Partial ↑SCFA, ↑diversity, ↓inflammation
Amir	Synbiotics, tailored diet, sleep/stress management	Better digestion, stable mornings, improved clarity	Partial ↑SCFA, shift in microbiota, ↓inflammation

DISCUSSION

An expanding body of research has revealed intricate bidirectional communication between the the central nervous system and gut microbiome now widely termed the microbiota–gut–brain axis. Accumulating clinical and translational evidence points to gut microbiota as a key modulator of neuropsychiatric health, impacting the onset, severity, and treatment response of disorders such as anxiety, depression, and more severe neuropsychiatric syndromes [7, 8, 9, 10]. Understanding the pathways that link gut dysbiosis to mental health symptoms is an urgent scientific and clinical priority, especially for individuals showing poor response to standard treatments [8, 11]. Mounting studies report that individuals with anxiety and depression often exhibit lower microbial diversity, with particular losses in beneficial short-chain fatty acid (SCFA) producing bacteria such as Faecalibacterium, Bifidobacterium, Lactobacillus, and an overabundance of pro-inflammatory taxa like Proteobacteria and Enterobacteriaceae. Reduced gut microbial richness (alpha diversity), as observed in both Jane and Amir in this study, is strongly associated with the persistence and severity of mental health symptoms [12, 13]. These

findings support a now well-established theme: that dysbiotic gut microbial communities may amplify vulnerability to and perpetuate symptoms of common mental illnesses [14]. Short-chain fatty acids (SCFAs), primarily propionate, acetate, and butyrate, are fundamental microbial metabolites that directly and indirectly support brain health. SCFAs maintain gut barrier integrity, regulate microglia (the CNS's immune cells), reduce systemic immune activation, and influence neurotransmitter production [15, 16, 17]. Reductions in fecal and systemic SCFA concentrations are consistently reported in major depressive disorder, schizophrenia, and anxiety. Furthermore, valeric acid and caproic acid have been associated with cognitive function, while SCFA deficits have been linked to memory impairment in schizophrenia and mood disorders [15, 16]. The data from Jane and Amir echo these patterns, with diminished SCFAs correlating with symptom severity and cognitive disruption. A shared mechanism now recognized in gut–brain axis research is inflammation. Dysbiotic gut communities increase intestinal permeability ("leaky gut"), permitting translocation of microbial products (e.g., lipopolysaccharides) and driving systemic and neuroinflammation via cytokine (e.g., IL-6, TNF-α, CRP) upregulation. Low-grade, chronic inflammation fuels overactivation of the hypothalamic-pituitary-adrenal (HPA) axis and results in blood–brain barrier (BBB) dysfunction, making the CNS more susceptible to neuroimmune assaults. Both patients in this study demonstrated low-grade systemic inflammation paralleling findings that link gut-mediated immune activation with depression, anxiety, and cognitive decline [18, 19]. Tryptophan, an essential amino acid, can be metabolized by gut microbes into serotonin (the "happiness neurotransmitter"), kynurenine, and other neuroactive compounds. Dysbiosis can disrupt this metabolic routing, shifting tryptophan away from serotonin synthesis toward the kynurenine pathway, increasing neurotoxic metabolites implicated in depression and cognitive symptoms. Thus, altered tryptophan metabolism provides a biochemical link connecting gut microbial imbalance, mood regulation, and neuroinflammation [20, 21]. Beyond metabolites, gut microbes influence neurotransmitter regulation (serotonin, dopamine, GABA, acetylcholine) and can signal directly to the brain through the vagus nerve markedly altering mood, anxiety, and cognitive function. Interventional studies in rodents show that ablation or restoration of key microbes rapidly modulates anxiety- and depression-like behaviors via vagal pathways, highlighting the critical and immediate influence of the gut microbiome on emotional states [22]. The emergence of "psychobiotics" (probiotics with psychological benefits) has spurred trials aimed at modulating the gut microbiome for mental health improvement. Multiple clinical and preclinical studies have demonstrated that interventions with probiotics, prebiotics, synbiotics, and targeted dietary changes can reduce depressive and anxiety symptoms, diminish inflammation, increase microbial diversity, and boost SCFA levels sometimes matching drug-based therapies in their beneficial effect [23]. Both participants in this study showed improvement in mood and inflammatory indices with structured, gut-targeted interventions. Fecal microbiota transplantation (FMT) transfer of stool from healthy donors to patients has shown preliminary benefit in small trials for patients with major depressive disorder or autism spectrum disorder, with ongoing clinical studies assessing its efficacy and safety. Such approaches highlight the burgeoning field of microbiome-based therapeutics for psychiatric disease, but they also underscore the need for rigorous trials, standardized protocols, and robust outcome measurement. A crucial yet sometimes overlooked dimension is the patient's lived experience of gut–brain interactions. Both Jane and Amir reported feeling dismissed when presenting with co-morbid gastrointestinal and psychiatric symptoms, echoing ethnographic research showing the psychological toll of medical invalidation. Conversely, recognition of gut-brain links empowers patients and encourages adherence to dietary and lifestyle modifications. This highlights the need for integrative, patient-centered mental health care that acknowledges the somatic underpinnings of "invisible" psychological illnesses [23].

CONCLUSION

This multiple-case study reinforces the existing research evidence supporting the critical role of the gut microbiome in mental health. Both participants exhibited chronic, treatment-resistant symptoms of anxiety and depression alongside gastrointestinal distress and documented gut dysbiosis. Laboratory findings revealed reduced microbial diversity, diminished short-chain fatty acid (SCFA) production, elevated inflammatory markers, and gut permeability, all of which have been mechanistically linked to poor mental health outcomes. Subjective improvements in mood, energy, and gastrointestinal function following microbiome-targeted interventions suggest a potentially significant role for integrative, gut-focused strategies in psychiatric care. The findings also highlight the interconnected nature of biological, psychological, and socio-environmental factors in mental health, highlighting the need for approaches that consider the microbiota–gut–brain axis. While these results are not sufficient to determine causality, they underscore the necessity for further research, especially randomized controlled trials and longitudinal microbiome studies. Personalized medicine that incorporates gut health diagnostics and interventions may offer a more effective, patient-centered model for those suffering from complex neuropsychiatric disorders. Ultimately, understanding and leveraging microbiome-based therapies has the potential to transform how we view and treat mental illness, shifting beyond the brain to the body's interconnected systems.

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