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Original Article

Altered gut microbiota and inflammatory markers in depression: A cross-sectional study.

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Abstract

Background:

Depression is a multifactorial mental health disorder influenced by psychological, biological, and environmental factors. Emerging evidence highlights the role of the gut–brain axis, particularly gut microbiota and inflammatory responses, in the pathophysiology of depression.

Aim:

To investigate alterations in gut microbiota composition and their association with systemic inflammatory markers in individuals diagnosed with depression.

Methods:

A cross-sectional observational study was conducted over one year, involving 100 participants 50 with clinically diagnosed depression and 50 healthy controls. Stool and blood samples were collected to analyze gut microbiota via 16S rRNA sequencing and inflammatory markers (CRP, IL-6, TNF-α) via ELISA. Data were analyzed using SPSS version 23.0.

Results:

Participant characteristics included religion and educational status, with 68% of participants identifying as Hindu, 22% as Muslim, and 10% as others; 54% had completed secondary education, while 46% had higher education. Depressed individuals showed significantly elevated levels of CRP (6.2 ± 2.1 mg/L), IL-6 (11.4 ± 3.8 pg/mL), and TNF-α (19.7 ± 6.5 pg/mL) compared to controls ($p < 0.001$). Gut microbiota analysis revealed a reduction in beneficial bacteria (*Lactobacillus* and *Bifidobacterium*) and an increase in *Proteobacteria* and Firmicutes/Bacteroidetes ratio in the depression group. Significant negative correlations were observed between beneficial bacteria and inflammatory markers (*Lactobacillus* vs. CRP ($r = –0.52$, $p < 0.001$) and *Bifidobacterium* vs. TNF-α ($r = –0.44$, $p = 0.004$)), indicating a potential link between microbial dysbiosis and systemic inflammation in depression.

Conclusion:

The findings suggest that depression is associated with gut microbiota imbalance and elevated inflammatory markers. Dysbiosis may contribute to systemic inflammation reinforcing the role of the gut–brain–immune axis in depression's pathogenesis.

Recommendations:

Future longitudinal studies should explore microbiome-based therapeutic interventions, such as probiotics or dietary modulation, as adjunctive treatments for depression. Incorporating gut health screening in mental health assessments may enhance holistic management.

Keywords: *Depression, Gut Microbiota, Inflammatory Markers, Dysbiosis, Gut–Brain Axis*
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Introduction

Depression is a prevalent and debilitating mental health disorder that affects over 280 million people worldwide, making it a leading cause of disability globally [1]. Characterized by persistent sadness, loss of interest, and cognitive disturbances, depression has complex etiologies involving genetic, psychological, and environmental factors [2]. In recent years, growing evidence has suggested a significant role of the gut–brain axis in the pathophysiology of depression, with a particular focus on the role of gut microbiota in influencing mood, behavior, and neuroinflammation [3].

The gut microbiota comprises trillions of microorganisms that reside in the human gastrointestinal tract and play a crucial role in digestion, immune modulation, and neurochemical production. These microbes communicate with the central nervous system through neural, endocrine, and immune pathways—collectively referred to as the microbiota–gut–brain axis [4]. Alterations in the composition and diversity of gut microbiota, known as dysbiosis, have been linked to several neuropsychiatric conditions, including depression [5]. Patients with depression have been found to exhibit reduced levels of beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*, and increased abundance of pro-inflammatory taxa like *Proteobacteria* [6].

One proposed mechanism linking gut microbiota to depression involves systemic inflammation. Dysbiosis may disrupt gut barrier integrity, allowing translocation of bacterial endotoxins into the bloodstream, thereby triggering an inflammatory response [7]. Elevated levels of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been consistently reported in individuals with major depressive disorder [8]. These cytokines can influence neurotransmitter metabolism, hypothalamic–pituitary–adrenal (HPA) axis activity, and neuroplasticity, ultimately contributing to the development and persistence of depressive symptoms [9].

Recent studies have emphasized the bi-directional relationship between gut microbiota and inflammation in depression. For instance, gut microbial therapies such as probiotics and fecal microbiota transplantation have shown promise in alleviating depressive symptoms, possibly by reducing inflammation and restoring microbial balance [10,11]. However, despite growing interest, the exact microbial profiles and their associations with inflammatory markers in depression remain insufficiently characterized, especially in diverse and under-researched populations. This study aims to investigate alterations in gut microbiota composition and their association with systemic inflammatory markers in individuals diagnosed with depression.

Methodology

Study Design

This study was designed as a cross-sectional observational study aimed at investigating the alterations in gut microbiota among individuals diagnosed with depression and examining its association with inflammatory markers. The design was chosen to provide a snapshot of gut microbial patterns and inflammatory profiles in depressed individuals at a single point in time.

Study Setting

The study was conducted in Keonjhar district, Odisha, India. Participants were recruited from outpatient and inpatient departments of mental health and general medicine units in local hospitals and community health centres within Keonjhar. The study was conducted over a period of one year, from 15 January 2023 to 15 January 2024

Participants

A total of 100 participants were enrolled in the study. This included 50 individuals clinically diagnosed with depression according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria and 50 healthy controls matched for age and sex. All participants were between the ages of 18 and 60 years.

The sample size was calculated using the formula for comparing two independent means:

Where for a 95% confidence level, for 80% power, is the estimated standard deviation of the outcome measure, and is the minimum expected difference between groups. Based on previous studies, and, giving a required sample size of 50 participants per group, resulting in a total of 100 participants (50 depressed and 50 healthy controls).

Inclusion Criteria

- Individuals aged 18–60 years.
- Patients diagnosed with depression based on DSM-5 criteria (study group).
- Healthy individuals without any known psychiatric or chronic medical illnesses (control group).
- Willingness to provide informed consent and stool and blood samples.

Exclusion Criteria

- Individuals currently on antibiotics, probiotics, or prebiotics within the past month.
- Patients with chronic gastrointestinal disorders, autoimmune diseases, or metabolic syndromes.

- Pregnant or lactating women.
- Individuals with substance use disorders.

Bias

Selection bias was minimized by employing random sampling techniques within each group. Interviewer and observer biases were reduced by using standardized data collection tools and blinding the laboratory personnel analyzing the biological samples to the clinical status of participants. Confounding factors such as dietary habits and recent infections were documented and controlled during analysis.

Data Collection

Data were collected using structured questionnaires and clinical interviews to record demographic information, medical history, and depression severity (using the Hamilton Depression Rating Scale). Stool samples were collected in sterile containers and transported under cold chain conditions to the microbiology laboratory for gut microbiota analysis using 16S rRNA gene sequencing. Blood samples were collected to measure inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) using ELISA kits.

Procedure

Upon enrollment, each participant was briefed about the study purpose and procedures, and written informed consent was obtained. Stool and blood samples were collected from all participants under aseptic conditions. Samples were processed and stored according to standard microbiological and biochemical protocols. Gut microbiota composition was analyzed through DNA extraction followed by 16S rRNA sequencing, while serum inflammatory markers were quantified using standard ELISA techniques. Data were entered and coded for analysis.

Statistical Analysis

Statistical analysis was performed using SPSS software version 23.0. Descriptive statistics were used to summarize demographic and clinical characteristics. The chi-square test was used for categorical variables, and independent t-tests were used for continuous variables, depending on the data distribution. Correlation between gut microbiota alterations and inflammatory markers was assessed using Pearson correlation coefficients. A p-value of <0.05 was considered statistically significant.

Results

A total of 300 individuals were initially assessed for eligibility during the study period. Of these, 200 participants were excluded for not meeting the inclusion criteria, which included a prior history of psychiatric disorders other than depression, recent use of antibiotics or probiotics, chronic inflammatory or autoimmune conditions, or unwillingness to provide informed consent. The remaining 100 participants, comprising 50 patients with clinically diagnosed depression and 50 age- and sex-matched healthy controls, were included in the study and completed all assessments of inflammatory markers and gut microbiota composition.

A total of 100 participants were included in the study, comprising 50 patients with clinically diagnosed depression (depression group) and 50 age- and sex-matched healthy individuals (control group). The mean age of participants in the depression group was 36.4 ± 10.2 years, while in the control group it was 35.8 ± 9.5 years. There was no significant difference in age (p = 0.73) or gender distribution (p = 0.84) between the two groups.

Table 1. Demographic Characteristics of Participants

Variable	Depression Group (n = 50)	Control Group (n = 50)	p-value	Test statistic
Mean Age (years)	36.4 ± 10.2	35.8 ± 9.5	0.73	$t = 0.35$
Gender (M/F)	28 / 22	27 / 23	0.84	$\chi^2 = 0.04$
BMI (kg/m²)	24.1 ± 3.4	23.6 ± 3.1	0.48	$t = 0.70$
Urban/Rural (%)	60 / 40	58 / 42	0.82	$\chi^2 = 0.05$

Explanation

The chi-square analysis confirms that there were no significant differences between the groups for categorical variables, supporting good baseline comparability. Continuous variables were analyzed using t-tests as reported previously.

Inflammatory Marker Levels

Serum levels of inflammatory markers were significantly elevated in the depression group compared to controls. The mean CRP level was 6.2 ± 2.1 mg/L in the depression group versus 2.9 ± 1.3 mg/L in controls ($p < 0.001$). Similarly, IL-6 and TNF-α levels were significantly higher in the depression group.

Table 2. Inflammatory Marker Levels in Study Groups

Inflammatory Marker	Depression Group (Mean ± SD)	Control Group (Mean ± SD)	p-value
C-reactive protein (mg/L)	6.2 ± 2.1	2.9 ± 1.3	<0.001
Interleukin-6 (pg/mL)	11.4 ± 3.8	5.2 ± 2.1	<0.001
TNF-alpha (pg/mL)	19.7 ± 6.5	9.3 ± 3.4	<0.001

All three inflammatory markers—CRP, IL-6, and TNF-α—were significantly elevated in the depression group, suggesting a systemic inflammatory response in individuals with depression.

Gut Microbiota Composition

Analysis of gut microbiota revealed a marked dysbiosis in the depression group. There was a significant decrease in *Lactobacillus* and *Bifidobacterium* genera, while *Firmicutes/Bacteroidetes* ratio and abundance of *Proteobacteria* were significantly higher.

Table 3. Gut Microbiota Profile of Study Groups (Mean Relative Abundance %)

Bacterial Genus/Phylum	Depression Group	Control Group	p-value
Lactobacillus	3.2 ± 1.1	7.6 ± 2.3	<0.001
Bifidobacterium	4.8 ± 1.5	9.2 ± 2.1	<0.001
Firmicutes	58.6 ± 7.3	52.1 ± 6.9	0.002
Bacteroidetes	22.4 ± 5.5	32.6 ± 6.2	<0.001
Proteobacteria	10.3 ± 3.8	3.6 ± 2.2	<0.001
Firmicutes/Bacteroidetes Ratio	2.61 ± 0.9	1.60 ± 0.7	<0.001

Explanation

The depression group exhibited a higher *Firmicutes/Bacteroidetes* ratio and increased *Proteobacteria*, which are commonly associated with dysbiosis and inflammatory states. The reduction in beneficial bacteria such as *Lactobacillus* and *Bifidobacterium* further suggests a disrupted gut microbial balance.

Correlation Between Gut Microbiota and Inflammatory Markers

Pearson correlation analysis showed a significant negative correlation between *Lactobacillus* levels and CRP ($r = -0.52$, $p < 0.001$), and a positive correlation between *Proteobacteria* and IL-6 ($r = 0.47$, $p = 0.002$).

Table 4. Correlation Between Gut Microbiota and Inflammatory Markers

Variables Compared	Correlation Coefficient (r)	p-value
Lactobacillus vs. CRP	–0.52	<0.001
Bifidobacterium vs. TNF-α	–0.44	0.004
Proteobacteria vs. IL-6	0.47	0.002
Firmicutes/Bacteroidetes vs. CRP	0.41	0.007

These findings suggest that gut microbiota imbalances are not only associated with depression but are also correlated with systemic inflammation, indicating a potential gut–brain–immune axis dysregulation.

Summary of Key Findings

- Depression was significantly associated with elevated inflammatory markers (CRP, IL-6, TNF-α).
- Gut microbiota analysis revealed reduced beneficial bacteria and increased pro-inflammatory microbes.
- Statistically significant correlations were found between altered microbiota and elevated inflammatory markers.

Discussion

This study investigated the association between altered gut microbiota and inflammatory markers in individuals with depression compared to healthy controls. A total of

100 participants were included, with 50 clinically diagnosed cases of depression and 50 age- and sex-matched healthy controls. The demographic data showed no significant differences between the two groups in terms of age, gender distribution, BMI, or residential background, indicating a well-matched comparison.

The analysis of inflammatory markers revealed significantly elevated levels of C-reactive protein (CRP: 6.2 ± 2.1 mg/L vs. 2.9 ± 1.3 mg/L in controls, $p < 0.001$), interleukin-6 (IL-6: 11.4 ± 3.8 pg/mL vs. 5.2 ± 2.1 pg/mL, $p < 0.001$), and tumor necrosis factor-alpha (TNF-α: 19.7 ± 6.5 pg/mL vs. 9.3 ± 3.4 pg/mL, $p < 0.001$) in the depression group compared to controls. This supports the growing body of evidence suggesting that depression is associated with a pro-inflammatory state. Elevated cytokine levels may contribute to neuroinflammation, potentially influencing mood regulation and contributing to depressive symptoms.

Gut microbiota profiling showed distinct differences between the two groups. Individuals with depression exhibited a notable reduction in beneficial bacterial genera such as Lactobacillus (3.2 ± 1.1% vs. 7.6 ± 2.3%, $p < 0.001$) and Bifidobacterium (4.8 ± 1.5% vs. 9.2 ± 2.1%, $p < 0.001$), which are known to support gut health and have anti-inflammatory properties. Conversely, there was a significant increase in the relative abundance of Proteobacteria (10.3 ± 3.8% vs. 3.6 ± 2.2%, $p < 0.001$) and a higher Firmicutes/Bacteroidetes (F/B) ratio (2.61 ± 0.9 vs. 1.60 ± 0.7, $p < 0.001$) in the depression group. These findings are indicative of dysbiosis, a state of microbial imbalance often associated with systemic inflammation and impaired gut barrier function.

Importantly, correlation analysis demonstrated a strong inverse relationship between beneficial microbes and inflammatory markers, and a positive relationship between pro-inflammatory bacteria and cytokine levels. For instance, Lactobacillus showed a significant negative correlation with CRP ($r = –0.52$, $p < 0.001$), Bifidobacterium was negatively correlated with TNF-α ($r = –0.44$, $p = 0.004$), while Proteobacteria was positively correlated with IL-6 ($r = 0.47$, $p = 0.002$). These findings suggest that alterations in gut microbiota may not only be a consequence of depression but could actively contribute to its pathophysiology through immune system activation and inflammation.

Overall, the results support the hypothesis that gut microbiota dysbiosis and inflammation are closely linked in depression. The presence of systemic inflammation alongside microbial imbalance points toward a disrupted gut-brain-immune axis. These findings underscore the potential role of gut microbiota modulation, through probiotics, prebiotics, or dietary interventions—as a complementary approach in managing depressive disorders.

Recent research continues to confirm that changes in the gut microbiota are closely linked to systemic and neuroinflammatory responses in patients with depression. A 2024 study on inflammatory depression reported that patients showed higher levels of Bacteroides and lower levels of Clostridium in the gut, along with increased short-chain fatty acid (SCFA)-producing species with dysregulated butanoate metabolism. Fecal microbiota transplantation (FMT) from these patients induced depressive-like behaviors and elevated inflammatory markers in mice, while administration of *Clostridium butyricum* reversed these effects, suggesting a causal role of gut-derived inflammation in depressive pathology [12]. Another review highlighted the contribution of Th17 cells in gut-brain immune crosstalk, showing that gut microbiota can induce homeostatic Th17 responses which, under pathological conditions, shift to inflammatory phenotypes, potentially contributing to treatment-resistant depression [13]. Similarly, an observational study on late-life depression (LLD) found dysbiosis marked by reduced abundance of *Akkermansia* and *Verrucomicrobia*, coupled with elevated levels of IL-6 and IFN-γ. These changes were significantly correlated with depression severity and proposed as potential biomarkers for LLD [14].

A large-scale cross-sectional study of treatment-naive MDD patients showed that these individuals had elevated high-sensitivity C-reactive protein (hs-CRP) and a distinct microbial signature, including higher levels of *Bifidobacterium* and *Haemophilus* and lower levels of *Bacteroides*, *Faecalibacterium*, and *Roseburia*. These microbiota changes were negatively correlated with hs-CRP and clinical depression scores, indicating a strong relationship between inflammation and microbial dysregulation in MDD [15].

In an animal model, chronic unpredictable mild stress (CUMS) altered the microbiota and activated the NLRP3 inflammasome in both brain and colon tissues. These changes were associated with reduced gut barrier integrity and elevated inflammatory cytokines. However, probiotic supplementation mitigated these effects, suggesting that modulation of gut flora can suppress inflammasome activation and inflammation-driven depression [16].

Comprehensive reviews have corroborated these findings, emphasizing that microbial metabolites like lipopolysaccharides and SCFAs can trigger cytokine release and modulate the kynurenine pathway, both of which are implicated in neuroinflammation and depressive symptoms [17]. Another review further noted that gut inflammation plays a pivotal mediating role in the bidirectional communication between the microbiota and the brain, with therapeutic implications drawn from probiotic interventions [18].

Clinical trials have also shown promising results for probiotics. A review of randomized controlled trials concluded that probiotics significantly reduced depressive symptoms in mild to moderate cases and lowered inflammatory markers such as IL-6 and kynurenine. These findings support the potential of probiotics as adjunct therapy to conventional antidepressants [19].

Generalizability

The findings may apply to similar adult populations with depression in tertiary care settings but should be validated in larger, multi-center studies.

Conclusion

This study highlights a significant association between altered gut microbiota and elevated inflammatory markers in individuals with depression. The findings support the role of the gut–brain–immune axis in the pathophysiology of depression and suggest that targeting gut microbiota may offer novel therapeutic approaches for improving mental health outcomes.

Limitations

The study was limited by its cross-sectional design, relatively small sample size, and lack of longitudinal follow-up to establish causality.

Recommendation

Gut microbiota modulation through probiotics, prebiotics, or dietary interventions may be considered as an adjunctive strategy in managing depressive disorders.

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Conflict of Interest

The authors declare that there is no conflict of interest related to this study.

List of Abbreviations

- CRP – C-reactive Protein
- IL-6 – Interleukin-6
- TNF-α – Tumor Necrosis Factor-alpha
- F/B Ratio – Firmicutes/Bacteroidetes Ratio
- MDD – Major Depressive Disorder
- LLD – Late-Life Depression
- SCFA – Short-Chain Fatty Acid
- FMT – Fecal Microbiota Transplantation

Author Biography

The authors are clinicians and researchers specializing in psychiatry, immunology, and microbiology with experience in studying the gut–brain–immune axis and inflammatory mechanisms in depression.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

All authors contributed equally to the study.

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