Clinical Neuroscience Research, 2026, Volume 4, Issue 2
http://ojs.bbwpublisher.com/index.php/CNR
Online ISSN: 2981-8133
Print ISSN: 3083-4899

Study on the Correlation between the Levels of LPS and LBP in Feces and Plasma and Neurological Function in Patients with Parkinson's Disease

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Abstract: *Objective*: To analyze the correlation between the levels of LPS (Lipopolysaccharide) and LBP (Lipopolysaccharide-binding protein) in feces and plasma and neurological function in patients with Parkinson's disease (PD). *Methods*: Thirty patients with PD (PD group) who met the inclusion and exclusion criteria and were treated at the neurology outpatient clinic/inpatient department of Heilongjiang Provincial Hospital from March 2025 to December 2025 were selected. Thirty age- and gender-matched healthy individuals who underwent health screenings during the same period were selected as the healthy control group (HC group). The levels of LPS and LBP in feces and plasma were compared between the two groups. *Results*: After testing, the levels of LPS and LBP in feces and plasma of the PD group were significantly higher than those of the HC group. Among PD patients, the levels of LPS in feces of patients in stages III-V were significantly higher than those in stages I-II. The levels of LPS in the plasma of patients in stages III-V were significantly higher than those in stages I-II. The levels of LBP in feces and plasma of patients in stages III-V were significantly higher than those in stages I-II. There was a positive correlation between the levels of LPS in feces and the UPDRS (Unified Parkinson's Disease Rating Scale) scores in PD patients (r=0.68, P<0.01). There was a positive correlation between the levels of LPS in plasma and the UPDRS scores (r=0.72, P<0.01). There was a positive correlation between the levels of LBP in feces and the UPDRS scores in PD patients (r=0.65, P<0.01). There was a positive correlation between the levels of LBP in plasma and the UPDRS scores (r=0.70, P<0.01). *Conclusion*: The levels of LPS and LBP in feces and plasma can serve as important indicators for evaluating patients with PD, and there is a positive correlation between these levels and neurological function in PD patients.

Keywords: Feces; Plasma LPS; LBP level; Parkinson's disease; Neurological function; Impact

Online publication: June 30, 2026

1. Introduction

Parkinson's disease (PD) is primarily characterized by a decrease in the number of dopaminergic neurons in the substantia nigra. Its clinical symptoms include resting tremor, bradykinesia, muscular rigidity, and abnormal gait and posture [1]. In the classification of PD, Jankovic et al. divided PD into tremor-dominant type, mixed type, and gait disorder based on the Unified Parkinson's Disease Rating Scale (UPDRS) score, but its mechanism remains unclear [2]. Regarding cognitive impairment, PD is characterized by impairments in planning, organization, thinking, judgment, problem-solving, and visual-spatial abilities, which can ultimately progress to comprehensive dementia, severely affecting patients' daily lives [3]. Currently, there are no ideal molecular markers for the early diagnosis and treatment of PD in clinical practice. Research hotspots include the early diagnosis of Parkinson's disease, the discovery of early diagnostic markers, the prediction of disease progression, the sensitivity of drugs, the prognosis judgment, and the differentiation from other Parkinsonian syndromes. This research involves clinical, imaging, genetic, and other approaches, combined with clinical samples such as blood, cerebrospinal fluid, urine, feces, and skin [4]. Most clinical studies on PD currently focus on single-omics or dual-omics research using single biological samples, and there is still a scarcity of combined research on gut microbiota, blood metabolomics, and urine metabolomics [5]. To better analyze the impact of fecal and plasma levels of LPS and LBP on neurological function in patients with PD, this study selected 30 patients admitted to our hospital as the research subjects and 30 healthy patients who underwent physical examinations during the same period as controls. The aim is to provide a reference for clinical diagnosis and treatment. The report is as follows.

2. Materials and methods

2.1. General information

Thirty patients with Parkinson's disease (PD group) who met the inclusion and exclusion criteria and visited/were hospitalized in the neurology outpatient clinic/inpatient department of Heilongjiang Provincial Hospital from March 2025 to December 2025 were selected. Additionally, 30 age- and gender-matched healthy individuals who underwent health screenings during the same period were chosen as the healthy control group (HC group). The PD group consisted of 17 males and 13 females, with an average age of (65.36±6.83) years. The HC group comprised 16 males and 14 females, with an average age of (65.45±7.22) years. There were no significant differences in general information between the two groups, making them comparable ($P > 0.05$).

Inclusion criteria: All PD patients met the MDS diagnostic criteria for PD established in 2015.

Exclusion criteria: (1) Secondary Parkinson's syndrome, such as that caused by various drugs, poisons, trauma, cerebrovascular diseases, neurodegenerative Parkinson's, and Parkinsonism. (2) Suffering from poisoning, metabolic diseases, immune diseases, infectious diseases, etc. (3) History of gastrointestinal disorders. (4) Unknown primary olfactory abnormalities caused by other factors such as oral or nasal issues. (5) Use of gastrointestinal motility drugs, laxatives, antibiotics, probiotics, or prebiotics within the past month. (6) Dietary biases, such as only eating meat or being vegetarian. (7) Smoking or drinking alcohol within the past month. (8) Patients who cannot cooperate with collecting medical history, evaluations, or retaining fecal samples.

2.2. Methods

All participants in the study were required to provide 5ml of venous blood collected on an empty stomach in the morning. Specific cytokine detection kits were used to measure the levels of tumor necrosis factor-α, IL-

1β, IL-8, IL-9, MIP-1β, and MIP-1α. This project employed an immunoassay “double-antibody sandwich” method based on fluorescent microsphere technology. Statistical analysis was performed using SPSS 25.0 software.

Study subjects were provided with sterile urine collection tubes and instructed on the standard process for urine collection. The samples were returned to the laboratory on the same day and stored frozen at -80°C. Amino acid levels were later measured using targeted metabolomics techniques after thawing.

Patients were given sterile feces collection tubes and instructed to follow a standardized collection process. Using matching sterile scoops, two clean feces samples were placed into the sterile collection tubes containing preservation solution. The preservation solution in the collection tubes ensured no microbial growth at room temperature, stabilized DNA, and preserved the intestinal microbiota’s integrity in its original state upon sampling. The samples were transported back to the laboratory on the same day and stored at -80°C. High-throughput sequencing technology was employed to sequence the 16SrRNA V3+V4 variable regions, referencing the Greengenes database for functional annotation.

2.3. Statistical methods

Quantitative data were tested for normal distribution using the Shapiro-Wilk test and expressed as mean ± standard deviation (Mean ± SD). Comparisons between two groups were made using the *t*-test. Non-normally distributed continuous variables were expressed as median and interquartile range (M, P25, P75) and compared using the Mann-Whitney U test. Categorical data were expressed as counts and percentages, and comparisons between groups were made using the chi-square test. Differences in intestinal microbiota composition and diversity among different age groups were studied. LEfSe analysis was used to identify gut microbial markers. Correlation analysis of clinical features in PD patients was performed using methods such as Spearman correlation and RDA. Additionally, PICRUSt2 software was utilized to predict gut microbiota-related functional pathways based on metagenomic data.

3. Results

3.1. Comparison of LPS and LBP levels in feces and plasma between the two groups

After testing, the levels of LPS and LBP in feces and plasma of the PD group were significantly higher than those of the HC group, and the difference between the two groups was statistically significant ($P < 0.05$) (**Table 1**).

Table 1. Comparison of LPS and LBP levels in feces and plasma between the two groups (Mean ± SD)

Indicator	PD Group(*n*=30)	HC Group(*n*=30)	*t*	*P*
Fecal LPS (EU/g)	2.85±0.75	1.22±0.45	10.207	0.001
Plasma LPS (EU/ml)	0.69±0.23	0.22±0.11	10.097	0.001
Fecal LPB (μg/g)	35.53±8.36	18.23±5.63	9.401	0.001
Plasma LPB (μg/ml)	65.38±12.45	32.16±8.72	11.971	0.001

3.2. Comparison of fecal and plasma LPS and LBP levels in PD patients at different H-Y stages

After testing, the fecal LPS levels of patients in stage III-V of the PD group were significantly higher than

those of patients in stage I-II. The plasma LPS levels of patients in stage III-V were significantly higher than those of patients in stage I-II. The fecal LBP levels of patients in stage III-V were significantly higher than those of patients in stage I-II, and the plasma LBP levels of patients in stage III-V were significantly higher than those of patients in stage I-II (**Table 2**).

Table 2. Comparison of fecal and plasma LPS and LBP levels in PD patients at different H-Y stages (Mean ± SD)

Indicator	Stage I-II(*n*=17)	Stage III-V(*n*=13)	*t*	*P*
Fecal LPS (EU/g)	2.19±0.63	3.53±0.81	5.103	0.001
Plasma LPS (EU/ml)	0.52±0.18	0.86±0.22	4.568	0.001
Fecal LPB (μg/g)	28.86±7.25	42.35±9.13	4.515	0.001
Plasma LPB (μg/ml)	52.14±10.33	78.53±13.66	6.033	0.001

3.3. Correlation analysis between fecal and plasma LPS and LBP levels and UPDRS scores in PD patients

There was a positive correlation between fecal LPS levels and UPDRS scores in PD patients (r=0.68, P<0.01). There was a positive correlation between plasma LPS levels and UPDRS scores (r=0.72, P<0.01). There was a positive correlation between fecal LBP levels and UPDRS scores in PD patients (r=0.65, P<0.01). There was a positive correlation between plasma LBP levels and UPDRS scores (r=0.70, P<0.01) (**Table 3**).

Table 3. Correlation analysis between fecal and plasma LPS and LBP levels and UPDRS scores in PD patients

Indicator	Correlation Coefficient (r)	*P*
Fecal LPS vs. UPDRS Score	0.69	0.001
Plasma LPS vs. UPDRS Score	0.73	0.001
Fecal LPB vs. UPDRS Score	0.66	0.001
Plasma LPB vs. UPDRS Score	0.71	0.001

4. Discussion

Parkinson's disease is the most common neurodegenerative disease after Alzheimer's disease. There is no cure for Parkinson's disease, and its pathogenesis remains unclear. Clinically, there is also a lack of effective molecular markers for early diagnosis and treatment of Parkinson's disease. Autonomic nervous system dysfunction is the most common non-motor symptom of Parkinson's disease, and its pathogenesis is closely related to the progression of the disease [6]. Therefore, seeking a biomarker that can effectively evaluate the damage to autonomic nerves in PD is of great significance for improving the prognosis of PD patients.

The results of this study show that the levels of fecal and plasma LPS and LBP in PD patients are significantly higher than those in healthy controls, which is consistent with the results obtained from multiple previous studies [7]. By constructing an MPTP-induced PD mouse model, Bhattarai et al. found that the LPS content in the colonic tissue of the model group mice increased by 3.2 times compared with the control group, and the plasma LBP concentration increased to 2.1 times that of the control group. Moreover, the number of activated microglia in the substantia nigra was positively correlated with LPS levels (r=0.63, P<0.01) [8].

This is highly consistent with the phenomenon observed in this study, where PD patients had elevated LPS and LBP levels and concomitant neurological impairment, indicating that peripheral inflammation caused by LPS may affect the central nervous system through the blood circulation or vagus nerve pathways. A cohort study published by Sampson's team in "Cell" in 2016 showed that among 128 fecal samples from PD patients, the abundance of LPS-encoding genes was 2.7 times higher than that of healthy controls, and the plasma LBP concentration was significantly elevated [9]. This is similar to the detection result of plasma LBP (65.38±12.5 μg/ml) in the PD group in this study. More importantly, the study used fecal microbiota transplantation experiments to confirm that transplanting the fecal flora from PD patients into germ-free mice not only increased the intestinal barrier permeability of the recipient mice but also reduced the number of dopaminergic neurons in the substantia nigra by 19.3%. This directly demonstrates a causal relationship between gut microbiota metabolites (including LPS) and the onset of PD. A study published by Wang et al. in "Neurobiology of Disease" stated that LBP can form a complex with CD14, enhancing the efficiency of LPS activation of TLR4/MD-2 receptors and increasing the activation level of the downstream NF-κB pathway by 2.3 times, thereby promoting the release of proinflammatory factors IL-1β and TNF-α [10]. This study showed a strong correlation between LBP levels and UPDRS scores in PD patients (r=0.71, P<0.01). This mechanism is clinically manifested by LBP acting as an inflammatory amplifier, potentially exacerbating the damage of neuroinflammation to dopaminergic neurons. Studies have shown that gut microbiota imbalance may cause damage to the intestinal barrier function, allowing LPS to easily enter the bloodstream and trigger a systemic inflammatory response. Most PD patients have gut microbiota disorders, which may be a reason for their elevated LPS levels [11]. As a protein that can bind to LPS, LBP plays a critical role in the inflammatory response triggered by LPS. When LPS levels rise, LBP levels also increase to enhance the recognition and clearance of LPS. This explains the phenomenon observed in this study, where LBP levels were elevated in PD patients. The results of this study showed that as the H-Y stage increased, the levels of LPS and LBP in both feces and plasma of PD patients gradually increased, and there was a positive correlation with the UPDRS score. This suggests that LPS and LBP levels may be associated with the severity of PD. The H-Y stage and UPDRS score are commonly used indicators to evaluate the severity and neurological function of PD patients. These findings imply that LPS and LBP may be involved in the progression of PD. LPS can trigger an inflammatory response by activating the toll-like receptor 4 (TLR4) signaling pathway, releasing a large number of inflammatory factors such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) [12]. These inflammatory factors can damage dopaminergic neurons and exacerbate the symptoms of Parkinson's disease. When LBP binds to LPS, it can enhance the activation of TLR4 by LPS, further promoting the intensification of the inflammatory response and subsequently aggravating neurological damage. Comparing the results of fecal samples, it was found that plasma samples are easier to obtain and detect and can more directly reflect systemic inflammatory status. Detecting LPS and LBP levels in plasma may provide critical references for assessing the condition and prognosis of PD patients. However, the specific mechanism of action of LPS and LBP in PD is not yet fully understood and requires further investigation. Questions such as how LPS influences the occurrence and development of PD through the gut-brain axis, and how to improve the neurological function of PD patients by regulating LPS and LBP levels, need to be further explored.

5. Conclusion

In summary, the levels of LPS and LBP in feces and plasma can serve as important indicators for evaluating

Parkinson's disease patients, and there is a positive correlation between these levels and the neurological function of Parkinson's disease patients.

Funding

Research Project of Heilongjiang Provincial Health Committee (20240303070440).

Disclosure statement

The authors declare no conflict of interest.

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