

Changes in functional connectivity of ventral striatum subregions in parkinson's disease patients with depression

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ABSTRACT

Despite the fact that several studies have found a high prevalence of depression among Parkinson's Disease (PD) patients, the pathophysiological mechanism of depression in PD remains unknown. The Ventral Striatum (VS), the reward network's core region, is crucial in the occurrence and progression of DPD. The purpose of this study was to look into the altered Functional Connectivity (FC) of VS sub regions in DPD. We enrolled 20 DPD patients, 37 NDPD patients, and 41 Healthy Controls (HC) who were age, gender, and years of education matched. The

patients were diagnosed with Parkinson's disease for the first time. The FC differences of VS sub regions were then detected using resting-state functional magnetic resonance imaging.

Key Words: *Depression*

INTRODUCTION

NDPD patients had significantly higher FCs between the bilateral ventromedial putamen and the left paracentral lobule, the right ventromedial putamen and the right precentral gyrus, and the left precuneus than HC patients. This study sheds new light on the neural mechanisms of depression in the early stages of Parkinson's disease and contributes to the investigation of potential neuroimaging markers for DPD. Parkinson's Disease (PD) is the second-most common neurodegenerative disease, characterized by both motor and non-motor symptoms. Depression is one of the first non-motor symptoms to appear in Parkinson's disease and can persist throughout the disease's course, reducing patient-ability and quality of life while increasing caregiver burden. The strong clinical overlap of depression and Parkinson's disease symptoms, such as psychomotor slowing, loss of expression, and sleep disturbances, complicates distinguishing and managing Depression in Parkinson's Disease (DPD) at the onset of the disease. Thus, identifying measurable indicators for early DPD detection could have significant research implications as well as potential clinical applications. Notably, advanced neuroimaging technology is a promising tool for this endeavor. Neuroimaging studies of depression have conceptualized reward and medial prefrontal-limbic networks as important neural systems for understanding the pathophysiologic processes of the disease.

The former involves reward processes and is influenced by dopamine neurotransmission, while the latter is influenced by serotonin neurotransmission. The classic pathological injury of Parkinson's disease is neurodegeneration of dopaminergic neurons. Furthermore, decreased dopamine levels in the Cerebral Spinal Fluid (CSF) of DPD patients are negatively correlated with depression severity. Dopamine is most likely more important than 5-hydroxytryptamine in DPD. In other words, the reward network is more likely to be involved in the pathological mechanism of DPD. Many studies have been conducted Using Functional Magnetic Resonance Imaging (fMRI) to investigate the functional connectivity (FC) pattern of the VS in depression. Patients suffering from major depression had lower FC of the Nucleus Accumbens (NAc) with the rostral anterior cingulate and the NAc subregions with the frontal gyrus. The FC between the NAc and the middle temporal gyrus, as well as the left ventral caudate and the postcentral gyrus, was reduced in adolescents with major depressive disorder. Overall, the VS, especially the representative NAc, plays an important role in the pathophysiology of depression. Before delving into these findings, several factors must be considered. To begin, both anti-and Parkinson's antidepressant medications alter FC between brain regions. Although the majority of the studies required subjects to abstain from drugs for a period of time prior to the trials, the chronic effects of drugs on the results cannot be completely ruled out.

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To avoid the potential impact of drugs on the results, this study included de-novo, untreated Parkinson's disease patients. Second, we tracked patient's PD progression and medication efficacy for at least a year to rule out other parkinsonian syndromes. It is recognized; however, that only long-term follow-up of de novo PD patients can definitively confirm the diagnosis. Finally, the overlap of symptoms between depression and Parkinson's disease complicates the operational DPD diagnostic criteria. As a result, this study was useful for analyzing the FC of the VS sub. This study, however, had several limitations. For starters, the sample size was small due to the stringent inclusion criteria. To reduce diagnostic bias, we screened de novo untreated Parkinson's disease patients and used the double conditions for DPD diagnosis. Second, while the MMSE scale excluded patients with dementia, patients with mild cognitive impairment could not be completely excluded. However, no statistically significant difference in MMSE scores was found between DPD and NDPD patients. MMSE was used as a covariable in statistical analysis, however, to eliminate the potential impact of cognitive impairment on the results. Third, because depression is frequently accompanied by anxiety, we excluded subjects who met the criteria for other mental disorders, including anxiety disorders. However, further research into DPD neuroimaging markers is required to clarify the potential

confounding role of anxiety symptoms. Furthermore, these study only used one imaging technique to investigate the potential relationship between the FCs of the VS sub regions and DPD. Thus, future research should include longitudinal follow-up observation and multimodal MRI technology to uncover the neuroimaging markers of DPD as well as the mechanism of DPD occurrence and progression. The primary role of the VS in the pathophysiological mechanism of DPD was confirmed by resting-state FC analysis in this study. We discovered that its effect on Parkinson's disease motor and depressive symptoms varies by region. The hyper connectivity between the vCa L and the MOG.L, which was found to be negatively correlated with the severity of depression in DPD patients, could be used to investigate potential neuroimaging markers to aid clinicians in the early stages of DPD diagnosis. Furthermore, the negative correlation suggested a potential compensatory mechanism of the visual network for depression in early-stage Parkinson's disease. The NAc, on the other hand, showed no relationship with DPD. The role of vCa in the pathological mechanism of DPD is likely to be greater than that of NAc. The findings are largely speculative and must be supported by future multimodal imaging studies. This study sheds new light on the disease's neural mechanism from a visual standpoint, and it suggests potential biomarkers for clinical early DPD diagnosis.