Poster

2025年9月27日(土) 11:00 ~ 12:10 Poster Session (6F Meeting Room 4-6)

Poster 36

[P-36-05] Habenular Abnormalities in Bipolar Disorder and Their Molecular Correlates: A Multimodal Imaging Study

*Meng xuan Qiao[1], Hua Yu[1], Tao Li[1] (1. Affiliated Mental Health Center & Hangzhou Seventh People's Hospital and School of Brain Science and Brain Medicine, Zhejiang University School of Medicine (China))

キーワード：habenula、bipolar disorder、Magnetic Resonance Imaging

Bipolar disorder (BD) is a chronic psychiatric condition marked by alternating manic and depressive episodes and a high degree of heritability. Growing evidence implicates the habenula—a central hub regulating dopaminergic and serotonergic signaling—in the pathophysiology of BD.This study integrated structural and functional neuroimaging with transcriptomic analyses to characterize habenular abnormalities in BD. Structural MRI and resting-state fMRI data from 78 BD patients and 102 healthy controls were analyzed. Habenular volumes were manually segmented and compared via ANCOVA. Resting-state functional connectivity (rs-FC) was assessed using DPABI-SURF, with the habenula as the seed region. Spatial transcriptomic associations were examined using partial least squares correlation with the Allen Human Brain Atlas, followed by gene enrichment and polygenic risk score (PRS) analyses.We observed significantly reduced bilateral habenular volumes in BD patients, particularly during depressive episodes. Habenular volumes were negatively correlated with depressive symptoms and positively associated with manic symptoms. Functional analysis revealed increased rs-FC between the bilateral habenula and the right precentral gyrus during manic states.Transcriptomic analysis indicated that altered habenular rs-FC was associated with genes enriched in synaptic structure and neurotransmission pathways, several of which overlapped with BD risk loci identified in genome-wide association studies. PRS analysis further revealed that habenula–precentral gyrus connectivity was negatively correlated with PRS for G-protein-coupled serotonin receptor signaling, suggesting a genetic basis for these functional alterations.These findings provide multimodal evidence linking structural and functional abnormalities of the habenula to the molecular and genetic architecture of BD. This integrative approach offers novel insights into the neurobiological mechanisms underlying BD and highlights potential targets for individualized therapeutic strategies.