

Biological Mechanisms Linking Macroscale Structural Covariance Network Dysfunction With Neuropsychiatric Symptoms



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Jiwei Jiang¹, Wenyi Li¹, Kun Zhao², Huaguang Zheng¹, Yong Liu², Jun Xu¹

¹Beijing Tiantan Hospital, Capital Medical University, Beijing 100070, China

²School of Artificial Intelligence, Beijing University of Posts and Telecommunications, Beijing, 100876China

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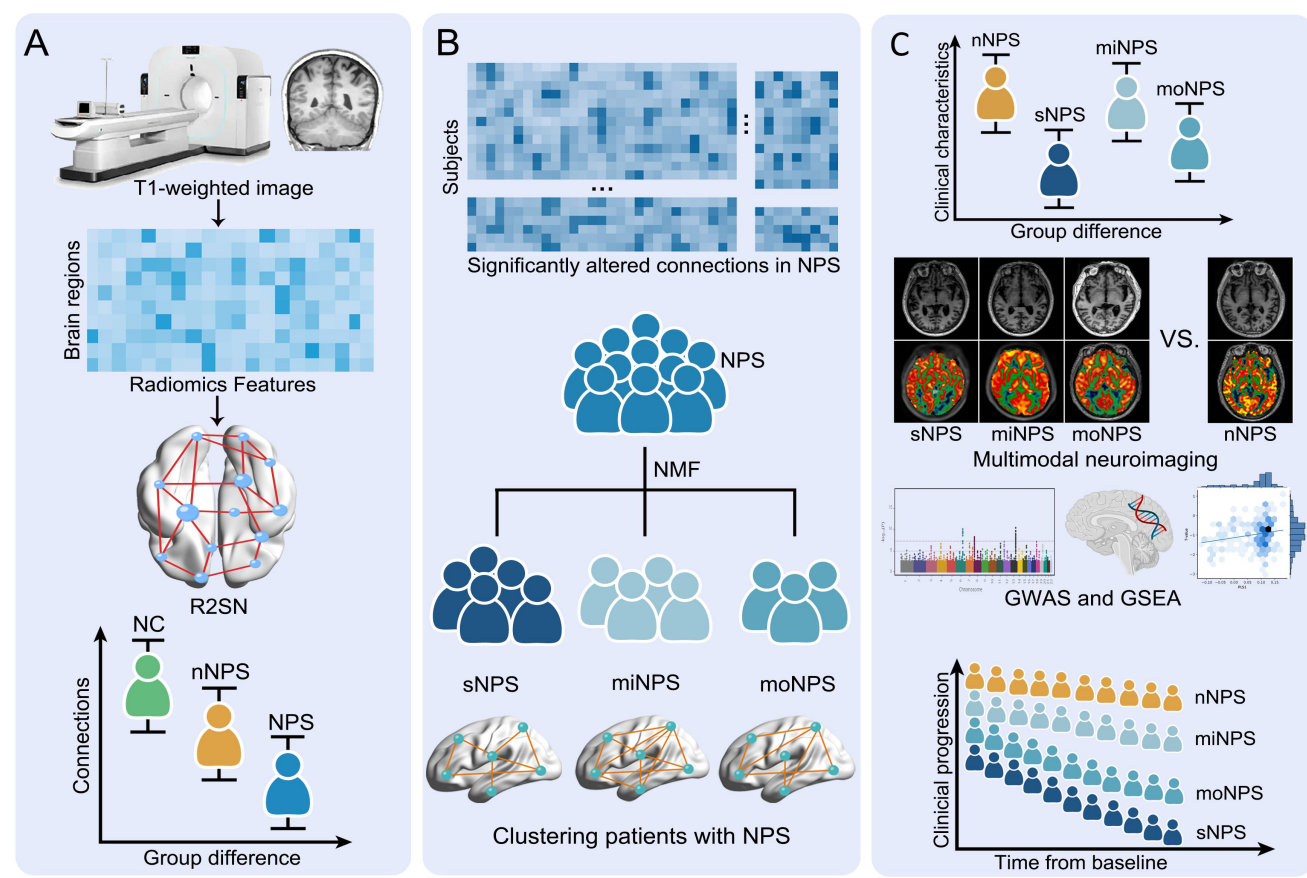
Professors Maria Vasileiadi and Sean Michael Nestor of the University of Toronto note that our findings represent an important step forward in understanding the pathophysiology of NPSs in AD and open the door for future precision medicine approach estailored to individual symptom profiles.

INTRODUCTION

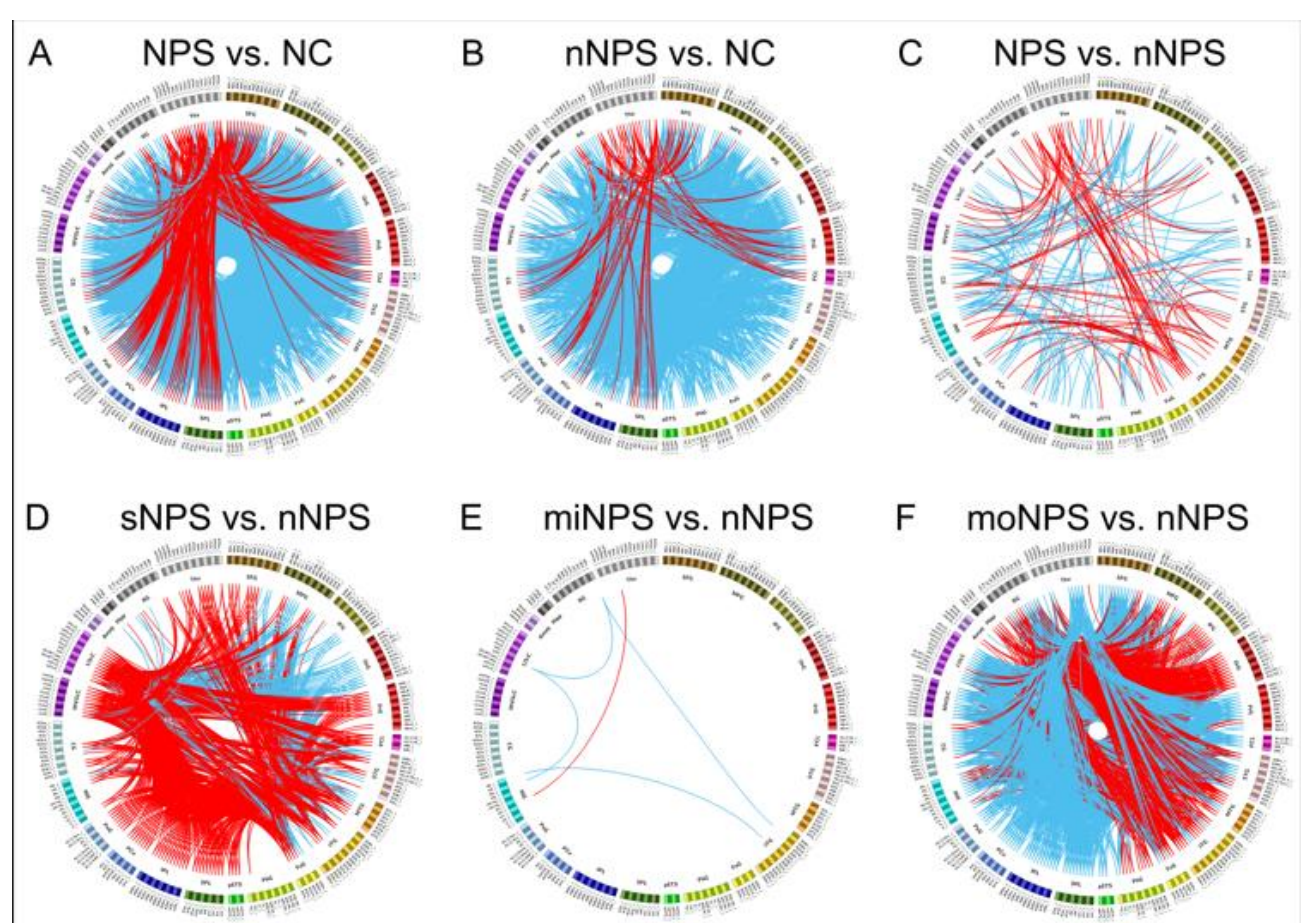
The high heterogeneity of neuropsychiatric symptoms (NPSs) hinders further exploration of their role in neurobiological mechanisms and Alzheimer’s disease (AD). We aimed to delineate NPS patterns based on brain macroscale connectomics to understand the biological mechanisms of NPSs on the AD continuum.

We constructed regional radiomics similarity networks for 550 participants (AD with NPSs [n = 376], AD without NPSs [n = 111], and normal control participants [n = 63]) from the CIBL (Chinese Imaging, Biomarkers, and Lifestyle) study.

METHODS

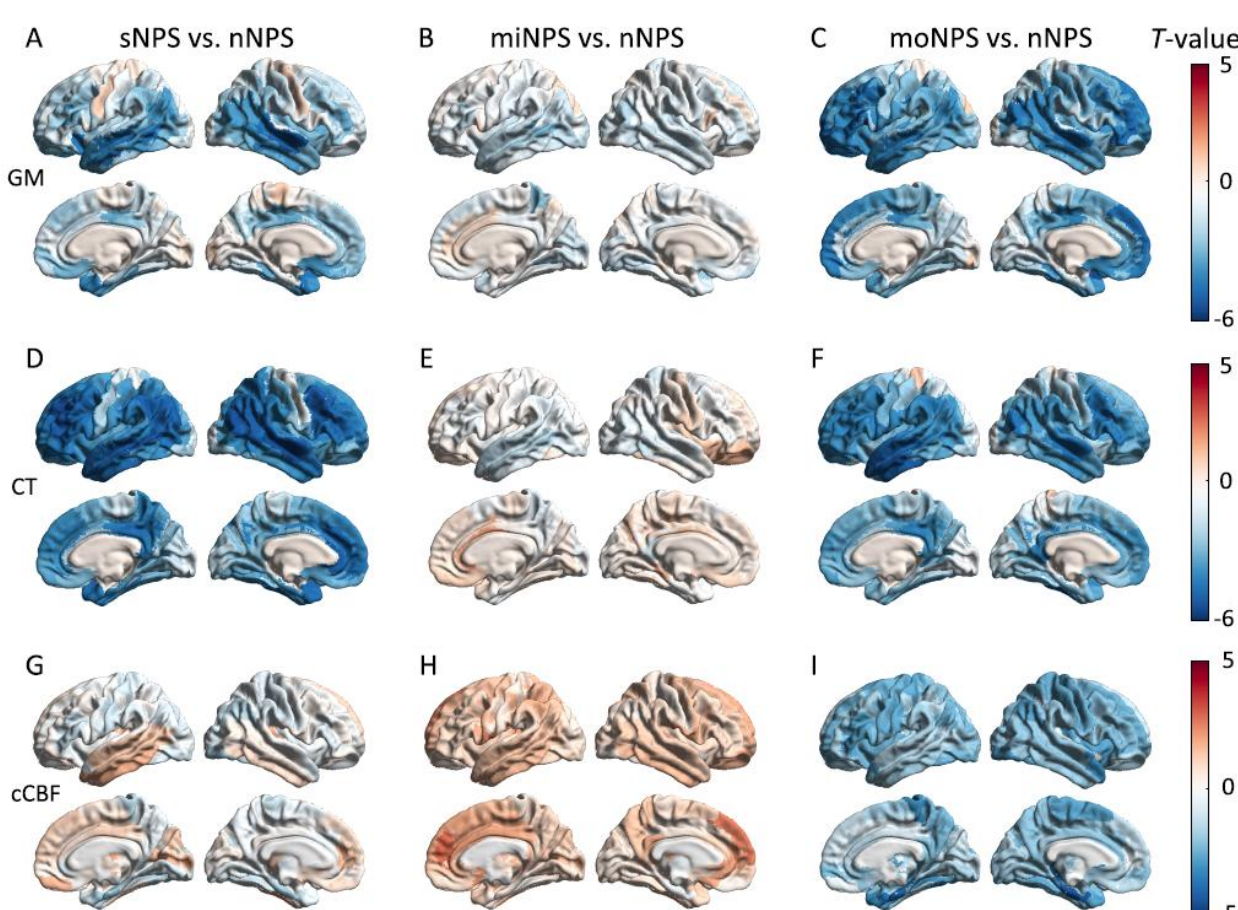


RESULTS

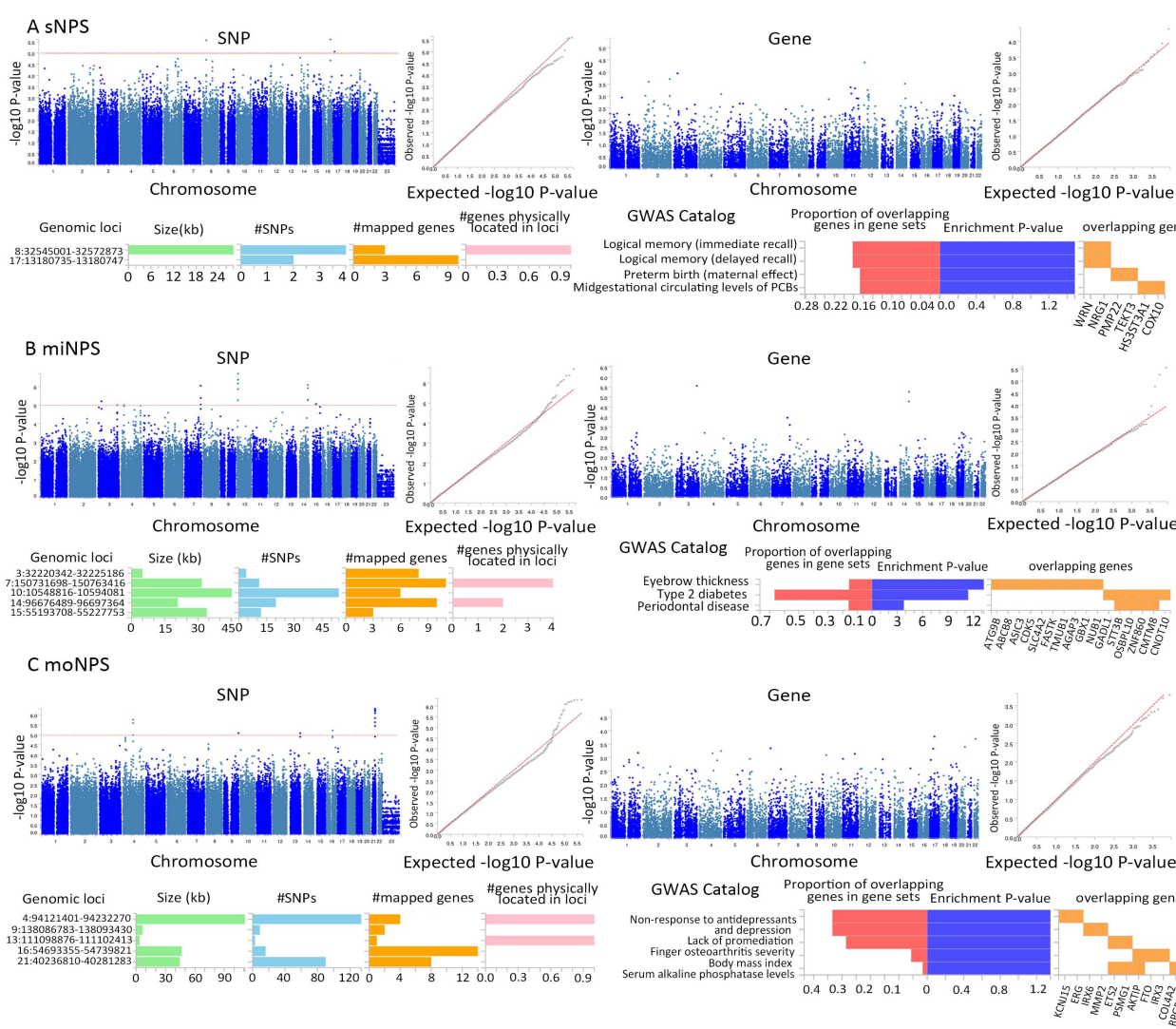


Characteristics of altered morphological connectivity in the R2SN profiles

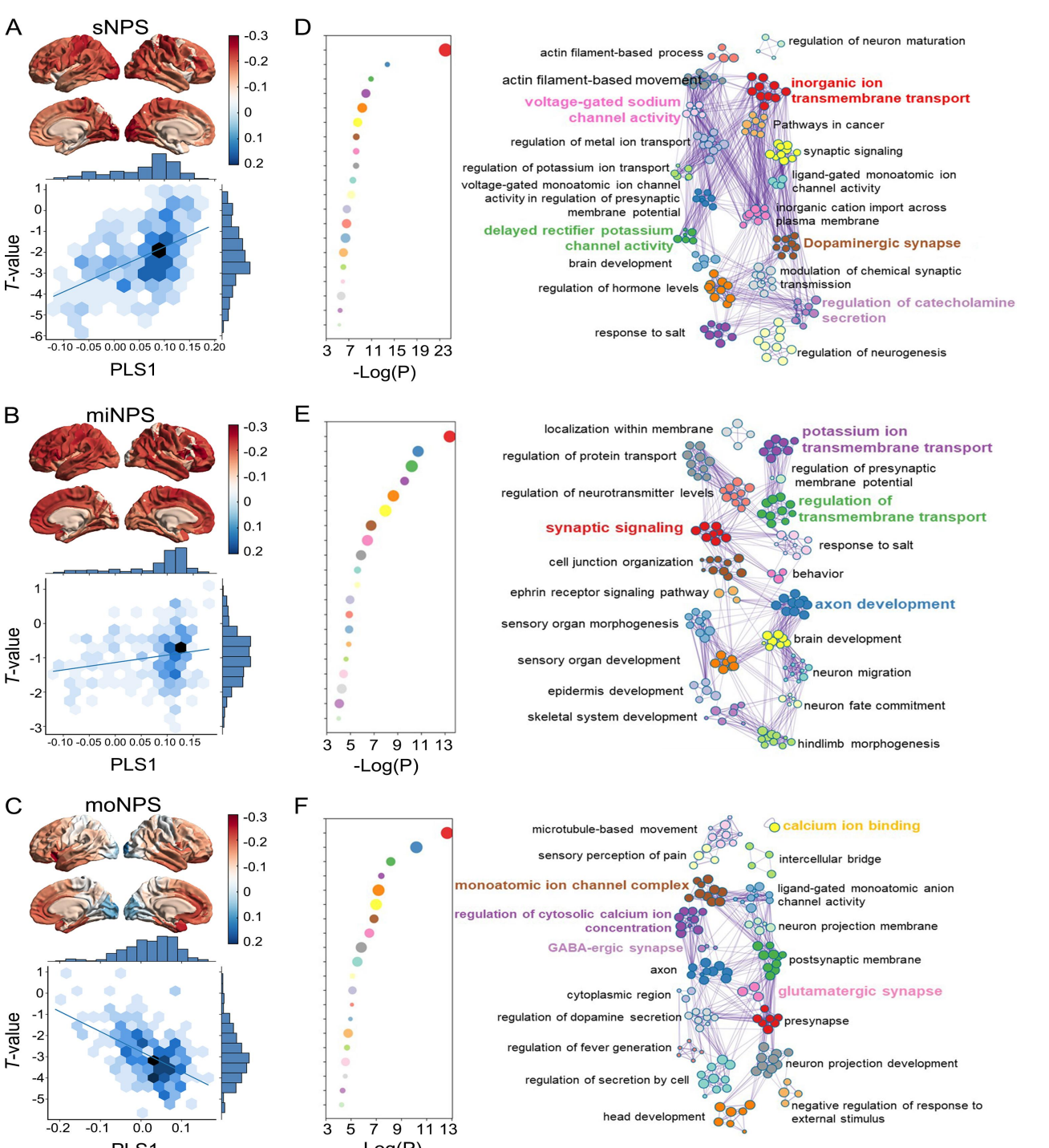
RESULTS



Differences in multimodal neuroimaging burden between three NPS subtypes and nNPS in the AD continuum



Severe and moderate NPS patterns were associated with significant cognitive impairment, increased plasma p-tau181 levels, extensive decreased brain volume and cortical thickness, and accelerated cognitive decline.



CONCLUSIONS

This study identified and validated three distinct NPS brain connectivity damage patterns, underscoring the role of NPSs in neurobiological mechanisms and progression of the AD continuum

CONTACT



Wechat: jjw126728

Email: jjwwxd126128@163.com

