Abnormal PCU-PCC network specific to auditory verbal hallucinations of schizophrenia

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Background: Abnormality of the default mode network (DMN) has been frequently reported in comparisons between schizophrenia and healthy controls, but the role of DMN in auditory verbal hallucinations (AVH) is still under debate. A remarkable portion of AVH studies did not report abnormality in DMN. Recently, an RSN that partially overlaps with DMN but plays different functional roles has been proposed by multiple studies, adding a new possibility in RSN dysfunctions in AVH. This RSN consists of dorsal precuneus and posterior cingulate cortex (referred to as PCN), and has often been viewed as a posterior DMN in some studies. PCN has been recently argued to act independently from DMN, as a memory retrieval system. This potential role of PCN suggests a novel hypothesis that AVH is actually related to PCN, because a main cognitive model of AVH has attributed this symptom to dysfunctions in memory system. We examined this hypothesis using a longitudinal design, where a group of AVH patients showed significantly reduced AVH scores between an initial scan and a follow-up scan.

Methods: 130 subjects were involved in this study, including three groups: (i) 65 healthy controls (HC); (ii) 36 schizophrenia patients without auditory verbal hallucinations (SZ); (iii) 29 schizophrenia patients with auditory verbal hallucinations (HAL), of which 35 HC, 15 SZ, and 13 HAL were re-scanned after 2 months. Independent component analysis was conducted to separate PCN from DMN. Voxel-wise component weights were compared across groups and correlated with AVH scores. Functional network connectivity was examined and correlated to AVH scores.

Results: We showed that the intra-PCN functional connectivity, instead of intra-DMN, was significantly different across the HC, SCZ, and HAL groups, and the intra-PCN functional connectivity was significantly correlated with AVH severity. The interaction between PCN and auditory network was also correlated with AVH severity, which links dysfunction in PCN to auditory-related symptoms in AVH. Our follow-up examination confirmed the specificity of PCN dysfunction to AVH by showing that with the remission of AVH symptoms, the functional connectivity difference in PCN between HAL and SZ patients diminished.

Conclusions: Our results support the hypothesis that abnormal PCN, instead of DMN, underlies the symptoms of AVH. The findings contribute to the memory dysfunction model of AVH by separating a memory retrieval-related brain network from DMN in schizophrenia. More importantly, this study reveals a functional connectivity network that exhibits specificity to AVH.