Cortical folding abnormalities in patients with schizophrenia who have persistent auditory verbal hallucinations

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Abstract
In schizophrenia temporal cortical volume loss differs between patients presenting with persistent auditory verbal hallucinations (pAVH) in contrast to those without hallucinatory symptoms (nAVH). However, it is unknown whether this deﬁcit re‡ects a neural signature of neurodevelopmental origin or if abnormal temporal cortical volume is re‡ective of factors which may be relevant at later stages of the disorder. Here, we tested the hypothesis that local gyriﬁcation index (LGI) in regions of the temporal cortex differs between patients with pAVH (n=10) and healthy controls (n=14), and that abnormal temporal LGI discriminates between pAVH and nAVH (n=10). Structural magnetic resonance imaging at 3T along with surface-based data analysis methods was used. Contrary to our expectations, patients with pAVH showed lower LGI in Broca’s region compared to both healthy persons and nAVH. Compared to nAVH, those individuals presenting with pAVH also showed lower LGI in right Broca’s homologue and right superior middle frontal cortex, together with increased LGI in the precuneus and superior parietal cortex. Regions with abnormal LGI common to both patient samples were found in anterior cingulate and superior frontal areas. Inferior cortical regions exhibiting abnormal LGI...
1. Introduction

Auditory verbal hallucinations (AVH) are a highly relevant clinical feature of schizophrenia that has attracted extensive clinical, phenomenological and neurobiological interest. Yet treating these symptoms, especially in persons suffering from persistent AVH (pAVH) which do either not or not sufficiently respond to psychopharmacotherapy, is still a major clinical challenge. AVH are a core symptom of schizophrenia and related spectrum-disorders, but they also frequently occur in other psychiatric entities and in the non-psychiatric population (Diederen et al., 2013). The clinical features of AVH may also characterize distinct nosological entities within the schizophrenia-spectrum (Mauri et al., 2008), which could be more prone to treatment-resistance (Gonzalez et al., 2006), and which may differ from patients without AVH in terms of brain volume abnormalities (Gaser et al., 2004; Shapleske et al., 2002; van Swam et al., 2013).

Across neuroimaging studies temporal cortical volume loss is one of the most consistent finding in patients with pAVH in schizophrenia (Allen et al., 2007, 2008). Most structural data derived from voxel-based morphometry (VBM) studies, which reported an association between AVH severity and bilateral superior temporal gray matter volume loss (reviewed by Modinos et al. (2013)). In a previous report, using a multivariate statistical technique for structural data analysis, we have shown lower volume of a network including medial/inferior frontal and bilateral superior temporal regions in pAVH patients compared to both controls and to non-hallucinating patients with schizophrenia (nAVH) (Kubera et al., 2014). These data also supported a specific role of this structural network in the expression of specific symptom characteristics, such as symptom duration or symptom frequency and intensity (Kubera et al., 2014). Yet it is unclear so far whether disrupted temporal cortical integrity reflects a stable trait of the disorder, or if may evolve over time during the course of the illness.

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