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Avolition and microstructural brain abnormalities in Schizophrenia: reduced fractional anisotropy in pathways connecting amygdala and insular cortex

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INTRODUCTION

The pathophysiology of the avolition/apathy domain of negative symptoms in schizophrenia is probably related to the dysfunctions of the motivation-reward system[1]. Although the nucleus accumbens (NAcc) and the ventral tegmental area (VTA) dopamine pathways appear to be central nodes of this circuit, structural and functional abnormalities have been reported in further key regions, including the orbito-frontal cortex (OFC), the amygdala (AMY) and the insular cortex (IC)[2].

AIMS

Our aim was to investigate the white matter connectivity patterns and their alterations within the above-mentioned regions in subjects with schizophrenia (SCZ) with respect to healthy controls (HC), using probabilistic analysis of diffusion tensor imaging (DTI) data. Furthermore, we examined the eventual associations between abnormal structural connectivity and clinical indices in patients.

METHODS

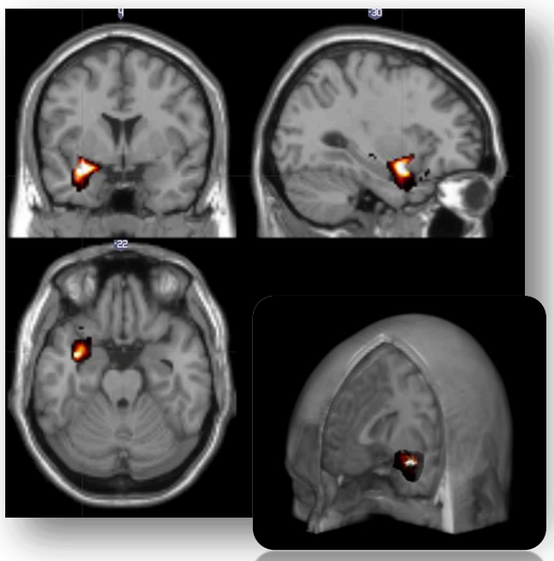
Thirty male SCZ and 17 age-matched male HC, underwent DTI (Table I). SCZ were evaluated clinically with the Schedule for Deficit Syndrome (SDS), the Positive and Negative Syndrome Scale (PANSS) and were administered the MATRICS consensus cognitive battery (MCCB). Probabilistic tractography was used to assess bilaterally the connectivity strength and structural integrity of the pathways connecting AMY and NAcc with OFC and IC. Statistical analysis was carried out by linear regression analysis and General Linear Model was fitted separately for each measure to assess differences between groups and correlations with clinical scores [3].

Table I	SCZ (N=30)	HC (N=17)	p
Demographic information			
Age	37.0±7.90	32.18±8.26	0.054
Paternal education (years)	8.57±5.04	12.88±4.92	0.007
Maternal education (years)	7.87±4.39	10.71±4.81	0.046
SDS Avolition	5.96±3.51		
SDS Expressive Deficit	4.04±2.95		
PANSS Positive	8.31±4.39		
PANSS Disorganization	8.27±4.06		
PANSS Depression	2.54±0.85		

RESULTS

In pathways connecting left AMY (lAMY) and ventral anterior IC (VAIC) (Fig. 1) a reduced fractional anisotropy (FA) was observed in SCZ compared to controls (Fig. 2A). In the same pathway, FA was negatively correlated with avolition/apathy but not with expressive deficit scores (Fig. 2B).

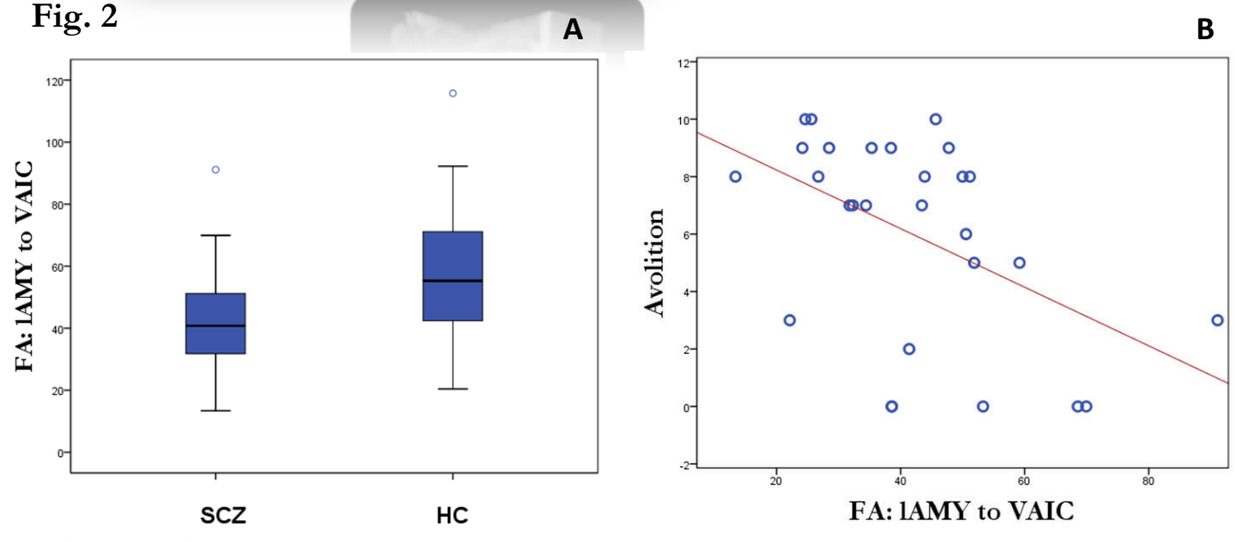
Fig.1



3D rendering of the average distribution of the paths connecting the left amygdala to the Ventral-Anterior Insular Cortex in all the study subjects, superimposed onto a T1-weighted volume in the MNI space.

Beside age, mean head movement was entered as nuisance covariates in the analysis, as patients moved significantly more than HC during the scan (0.42±0.12mm vs. 0.35±0.10mm, p=0.041).

Fig. 2

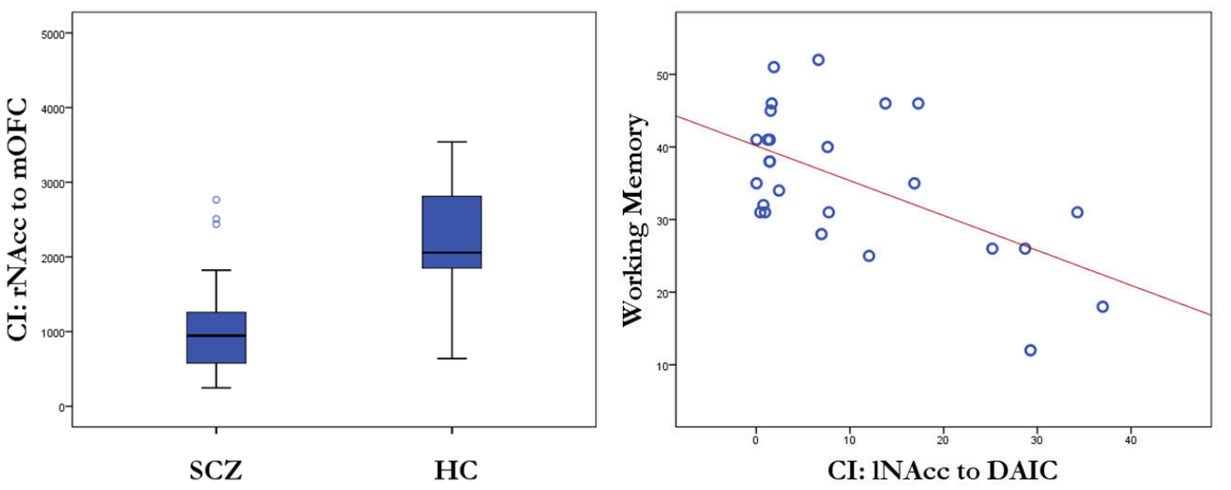


* HC>SCZ, *Beta In 0.390, p=0.0048*

Avolition showed a significant negative correlation with altered structural integrity (FA) between AMY and IC, regions involved in encoding and retrieval of experienced value [4].

A reduced connectivity indices (CI, % of the probabilistic streamlines originating from a region that reach a second one) between right NAcc (rNAcc) and medial OFC (mOFC) was found in SCZ as compared to controls (Fig. 3A). The CI between left (l) NAcc-dorsal anterior IC (DAIC) was negatively correlated with working memory scores (Fig. 3B).

Fig. 3



 # HC>SCZ: βIn 0.524, p=0.0001

CONCLUSIONS

According to our findings, the avolition/apathy but not the expressive deficit domain is related to the reward system dysfunction [4]. Finally, distinct alterations seem to underlie cognitive impairment and avolition/apathy [5].

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All authors report no conflict of interest