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White matter structural connectivity abnormalities in subjects with Deficit Schizophrenia: a Diffusion Tensor Imaging Study

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INTRODUCTION

Since schizophrenia represents a clinically heterogeneous disorder, the identification of the neurobiological correlates of the disease is made difficult [1]. Deficit schizophrenia (DS) has been proposed as a separate disorder with respect to non-deficit schizophrenia (ND). It is characterized by the presence of primary, enduring negative symptoms and by different course, risk factors and clinical features [2].

AIMS

Our primary aims, using probabilistic analysis of diffusion tensor imaging data [3], were 1) to investigate differences of white matter connectivity within several brain areas in subjects with DS compared to ND and healthy controls (HCs); 2) to investigate the relationships between white matter connectivity and clinical variables.

METHODS

Forty-six subjects with chronic schizophrenia (SCZ) and 35 age- and gender-matched HCs were included (Table I). 9 SCZ were classified as DS, and 37 as ND using the Schedule for the Deficit Syndrome (Table II). Psychopathology was assessed with the Positive and Negative Syndrome Scale, and neurocognition with the MATRICS Consensus Cognitive Battery. Connectivity index [CI] (% of the probabilistic streamlines originating from a region that reach a second one) and Fractional Anisotropy (FA) of pathways connecting dorso-lateral prefrontal cortex (DLPFC), nucleus accumbens (NAcc), amygdala (AMY) and insular cortex (IC) were examined.

Table I

	SCZ (N=46)	HCs (N=35)	p
Demographic information			
Gender (M/F)	30/16	17/18	.136
Age	35.87±7.74	32.94±8.8	.116

Table II

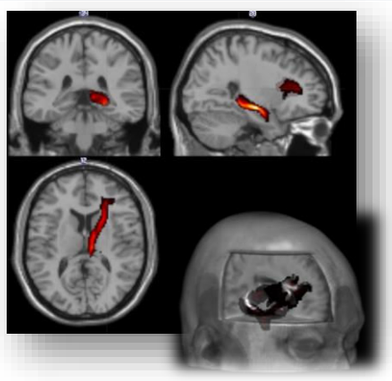
	ND (N=37)	DS (N=9)	p
Gender (M/F)	25/12	5/4	.508
Age	36.57±7.49	33±8.52	.219
SDS Avolition	4.75±3.43	7±3.58	.104
SDS Expressive Deficit	2.97±2.43	5.25±3.65	.035
PANSS Positive	8.09±4.21	6±2.44	.215
PANSS Disorganization	7.26±3.64	7.43±4.27	.917
PANSS Depression	2.49±0.84	1.42±0.49	.003

All authors report no conflict of interest

RESULTS

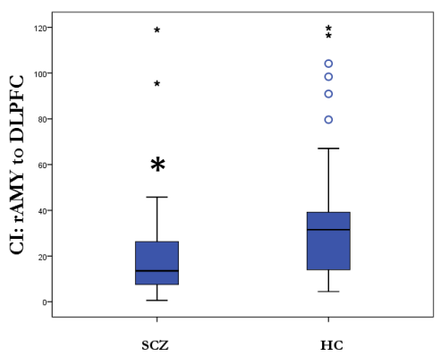
CI between right AMY and DLPFC (Fig. 1) was reduced in SCZ compared to HC (Fig. 2), but did not differ between DS and ND.

Fig. 1



3D rendering of the average distribution of the paths connecting the right AMY to the DLPFC in all the study subjects, superimposed onto a T1-weighted volume in the MNI space.

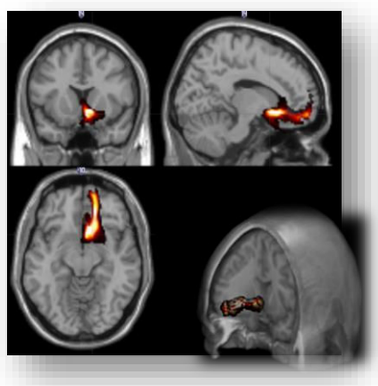
Fig. 2



* SCZ<HC Beta In 0.311; p=0.0044

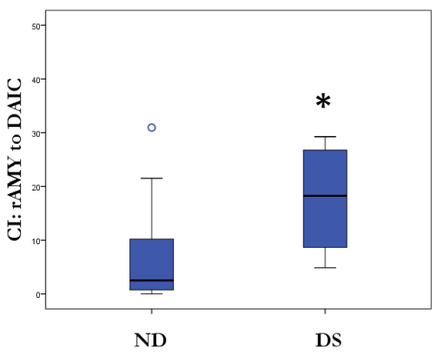
CI between right AMY and dorsal-anterior IC (Fig. 3) was increased in DS compared to ND (Fig. 4).

Fig. 3



3D rendering of the average distribution of the paths connecting the right AMY to the Dorsal-Anterior IC in all the study subjects, superimposed onto a T1-weighted volume in the MNI space.

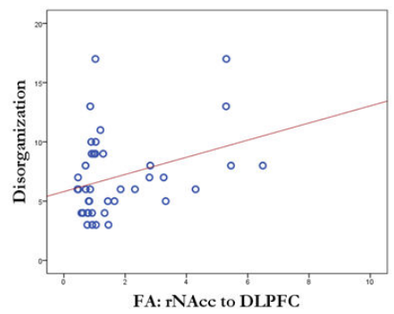
Fig. 4



* DS> ND Beta In 0.434; p=0.0036

In SCZ, PANSS disorganization was associated to the FA of right NAcc-DLPFC connections (Fig. 5).

Fig. 5



Beta In 0.441; p=0.0031

CONCLUSIONS

Our data are in line with previous results, showing that different symptom dimensions and clinical subtypes of schizophrenia are underpinned by distinct neurobiological substrates. In addition, our findings concerning the Connectivity between right amygdala and dorsal-anterior insular cortex in DS demonstrate that primary and persistent negative symptoms are related to connectivity abnormalities within brain regions involved in guiding goal-directed behavior based on experienced value [4-5].

REFERENCES

[1] Galderisi, S. et al. (2015). Eur Arch Psychiatry Clin Neurosci, 265:543-558.
[2] Mucci A. et al. (2017). Schizophr Res, S0920-9964(16)30238-9.
[3] Bracht, T. et al (2014). Schizophrenia Research, 153:136–142
[4] Harsay, H.A. et al. (2012). Front Hum Neurosci, 27-6:246.
[5] O'Doherty, J.P. (2016). Curr Topics Behav Neurosci, 27: 291–312.