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 the Positive and Negative Syndrome Scale (PANSS) and were administered the MATRICS consensus assessments 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 and demographic information.

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).

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].

REFERENCES