Results: In our preliminary analyses, patients show significantly lower MSCEIT scores. Furthermore, MSCEIT scores are directly related to FA values in the tracts connecting prefrontal cortex to anterior cingulate and superior temporal gyrus in the patients. Discussion: Social cognition impairments seem to be associated with altered structural connectivity in the patients.

S175. AMOTIVATION IS ASSOCIATED WITH SMALLER VENTRAL STRIATUM VOLUMES IN OLDER PATIENTS WITH SCHIZOPHRENIA

Fernando Caravaggio1, Gagan Fervaha2, Yusuke Iwata3, Eric Piltman1, Jun Ku Chung1, Shinichiro Nakajima1, Wanna Maa1, Philip Gerretsen1, Julia Kim3, Mallar Chakravarty4, Benoît Mulsant1, Bruce Pollock1, David Mamo1, Gary Remington1, Ariel Graff-Guerrero1
1Centre for Addiction and Mental Health, University of Toronto; 2Queen’s University Faculty of Medicine; 3Centre for Addiction and Mental Health; 4McGill University

Background: Motivational deficits are prevalent in patients with schizophrenia, persist despite antipsychotic treatment, and predict long-term outcomes. Evidence suggests that patients with greater amotivation have smaller ventral striatum (VS) volumes. We wished to replicate this finding in a sample of older, chronically medicated patients with schizophrenia. Using structural imaging and positron emission tomography, we examined whether amotivation uniquely predicted VS volumes beyond the effects of striatal dopamine D2/3 receptor (D2/3R) blockade by antipsychotics.

Methods: Data from 41 older schizophrenia patients (mean age: 60.2 ± 6.7; 11 female) were reanalysed from previously published imaging data. We constructed multivariate linear stepwise regression models with VS volumes as the dependent variable and various sociodemographic and clinical variables as the initial predictors: age, gender, total brain volume, and antipsychotic striatal D2/3R occupancy. Amotivation was included as a subsequent step to determine any unique relationships with VS volumes beyond the contribution of the covariates. In a reduced sample (n = 36), general cognition was also included as a covariate.

Results: Amotivation uniquely explained 8% and 6% of the variance in right and left VS volumes, respectively (right: β = .38, t = 2.48, P = .01; left: β = .31, t = 2.17, P = .03). Considering cognition, amotivation levels uniquely explained 9% of the variance in right VS volumes (β = .43, t = 0.26, P = .03).

Discussion: We replicate and extend the finding of reduced VS volumes with greater amotivation. We demonstrate this relationship uniquely beyond the potential contributions of striatal D2/3R blockade by antipsychotics. Evocating the structural correlates of amotivation in schizophrenia may help develop treatments for this presently irremediable deficit.

S176. SYSTEMATIC REVIEW AND META-ANALYSIS OF MAGNETIC RESONANCE IMAGING FINDINGS IN 22Q11.2 DELETION SYNDROME

Guido Maria Lattanzio1, Cristina Scarpazza2, Fabio Di Fabio3, Philip McGuire1, Giuseppe Sartori1, Simon B. Eickhoff1, Stefania Tognin1
1Institute of Psychiatry, Psychology & Neuroscience, King’s College London; 2Institute of Psychiatry, Psychology & Neuroscience, King’s College London, University of Padua; 3Sapienza University of Rome; 4University of Padua; 5Institute for Systems Neuroscience Heinrich-Heine University Düsseldorf

Background: Since the 22q11.2 Deletion Syndrome (22q11.2 DS) is the most important genetic model for psychotic disorders, an increasing interest in its brain structural and functional abnormalities has arisen in the last decade. However, studies so far have reported inconsistent findings. Therefore, the aims of the present study are 1) to systematic review the literature on structural and functional brain abnormalities associated to 22q11.2 DS and 2) to identify the most consistently reported abnormalities through a meta-analysis of structural (sMRI) and functional magnetic resonance imaging (fMRI) studies.

Methods: The following electronic databases were systematically searched: PubMed, ETHOS, Kings Open Portal, EMBASE, MEDLINE, PsyCINFO and CINHAL. Studies were included if they presented original data, were written in English, had a sample size larger than 5, had a healthy control comparison group, and if they reported results from a whole brain analysis. As we were interested in identifying abnormalities in both brain structure and function, in the systematic review we included studies that used different imaging techniques (i.e. sMRI, fMRI and diffusion tensor imaging (DTI)). The meta-analysis was performed with studies reporting results in standardised-space coordinates (e.g. Talairach), using the Activation Likelihood Estimation (ALE) method. Results were corrected at cluster level with family wise error correction (p<0.01) and 1000 permutations.

Results: Seventy-three original articles were included in the systematic review, 25 of these were also included in the meta-analysis. Forty-two sMRI, 23 fMRI and 11 DTI articles were retrieved. Only one study performed a direct comparison between 22q11.2 DS individuals with and without psychosis.

The systematic review revealed that the most affected areas were the frontal middle gyri bilaterally, the posterior cingulum bilaterally, the right cuneus, the precuneus bilaterally, the right superior temporal gyrus, the left parietal inferior gyrus and the left side of the cerebellum. The meta-analysis revealed consistent abnormalities in a cluster located in the inferior parietal lobe (4936 voxels, peak of activation in the coordinate 44 -52 48) and extending to the superior temporal gyrus, supramarginal gyrus and precuneus. A second cluster of consistent activation is found in the posterior cingulate cortex (3104 voxels, peak of activation in the coordinate 6 -50 16).

Discussion: The systematic review revealed widespread abnormalities throughout the brain, mainly within areas involved in visual and speech processing, language, and within association areas. The meta-analysis of structural and functional studies revealed consistent abnormalities in the inferior parietal lobe, an area consistently found affected in psychosis.

Only few studies on 22q11.2 DS individuals with psychosis were available and most studies included young individuals (mean age 15.12) rather than adults. 22q11.2 DS is one of the most compelling genetic models of schizophrenia, however most imaging studies do not provide clinical data on psychotic symptoms. This could partially be explained by the relatively low mean age of the overall sample; some participants could have been too young to manifest psychotic symptoms. Finally, the present study does not allow to make inferences on brain changes overtime as longitudinal studies were scarce.

Future studies should adopt a longitudinal design and investigate brain abnormalities in adults with 22q11.2 DS displaying symptoms of psychosis. This would help to clarify the brain structural and functional features associated with this particular form of psychosis and their longitudinal course.

S177. FRONTAL CORTICAL PLASTICITY IN SCHIZOPHRENIA PATIENTS EXAMINED BY LTP-INDUCING ANODAL TDCS AND REPETITIVE EEG

Benjamin Pross1, Melina Siamouli1, Oliver Pogarell1, Peter Falkai1, Alkomet Hasan1, Wolfgang Strube1
1Ludwig Maximilians University Munich