dopaminergic and serotoninergic neurotransmitter systems have also been linked to FTD.
Conclusions: Many areas of the brain have been implicated in the pathogenesis of FTD, though some more consistently than others. The superior temporal, middle temporal and inferior frontal gyri in particular have repeatedly revealed both structural and functional alterations in patients with FTD. A reversed lateralization has also been observed at both structural and functional levels. The different neurotransmitter systems have also been connected with FTD, with the glutamate system being the one more thoroughly explored. However, the direction of causality between changes in the brain and FTD, and the influence of potential mediators remain largely unknown.

Disclosure of Interest: None Declared

O0141

Transdiagnostic and Disorder-Specific Resting-State Functional Network Alterations in Alcohol Use Disorder, Schizophrenia, Bipolar Affective Disorder and Obsessive-Compulsive Disorder

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Introduction: The ICD and DSM diagnostic categories do not represent entirely distinct entities because several cognitive impairments are shared across psychiatric disorders. Such shared cognitive impairments are hypothesized to be caused by common neurobiological substrates, one of which is transdiagnostic alterations in functional network connectivity (FNC).
Objectives: To investigate and compare the within-network functional connectivity (WNFC) and between-network functional connectivity (BNFC) in alcohol use disorder (AUD), schizophrenia (SCZ), bipolar affective disorder (BPAD), obsessive-compulsive disorder (OCD) and healthy controls (HC) using resting-state fMRI employing a data-driven exploratory approach.
Methods: The current study was a secondary analysis of data from the ADBS project in India. After pre-processing of fMRI data, a spatially and temporally constrained group-independent component analysis in the GIFT toolbox was performed using the NeuroMark templates to generate 53 independent components (ICs). These components were divided into seven functional domains including subcortical (SC), auditory (AU), sensorimotor (SM), visual (VI), cognitive-control (CC), default-mode (DM), and cerebellar (CB). To investigate the FNC correlations associated with group status (patients or HC) univariate models were applied which were subjected to corrections for multiple comparisons at an alpha=0.05 significance level using the FDR.
Results: The overall sample size was 249 [AUD=35, SCZ=44, BPAD=48, OCD=53, and HC=69]. Transdiagnostic WNFC alterations largely involved dysconnectivity in the CC, DM, and SC domains, resulting in ICs with both increased and decreased WNFC. Transdiagnostic BNFC alterations were primarily in the form of increased connectivity of the SC domain with various cortical domains whereas reduced connectivity was noted between AU, VI, SM, and CB domains. There was AUD-specific hyperconnectivity in the CC domain and SCZ-specific hyperconnectivity in the DM domain, and dysconnectivity in the SC domain. BPAD-specific hyperconnectivity was identified in DM and SC domains in addition to increased connectivity between CB and SM domains and decreased connectivity between CB and SC domains. All results were significant at $p \leq 0.05$; [FDR] $q = 0.05$.
Conclusions: Our transdiagnostic WNFC alterations corresponded to the central executive network, default mode network, salience network, and CSTC loop, which provided transdiagnostic evidence for the triple network model of psychopathology and underlined the relevance of subcortical dysconnectivity in this model. Furthermore, our BNFC changes showed subcortical hyperconnectivity with many cortical networks, underscoring its relevance as a potential target for transdiagnostic therapeutic interventions.

Disclosure of Interest: None Declared

O0142

Alterations in brain myelination at early-stage schizophrenia detected by macromolecular proton fraction MRI

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Introduction: There is evidence that cerebral myelination is impaired in schizophrenia. The purpose of this study is to find the myelin content changes in the brain structures of patients with early-stage schizophrenia using the macromolecular proton fraction (MPF) method, and also to evaluate the differences in the myelination of these structures.
Objectives: To measure MPF in the brain structures of schizophrenia patients
Methods: Forty-five subjects, 22 controls (10m+12f, 31.6±9.7 y.o.) and 23 schizophrenia patients (F20.0, 11m+12f, 31.5±5.1 y.o.). Philips Achieva dStream 3T MRI scanner, standard head coil. The magnetization transfer (TR=20 ms, TE=4.60 ms, FA=10°), T1-weighted (TR=20 ms, TE=4.60 ms, FA=20°) and PD-weighted (TR=20 ms, TE=4.60 ms, FA=4°) were acquired. The MPF maps were reconstructed using home-made software. In FSL, non-brain structures were removed and MPF maps were registered to a standard MNI152 1 mm atlas. Harvard Oxford Cortical and Subcortical atlases were used to select areas of interest. T-test was used in search for between-group differences.
Results: A 3% decrease in myelination in schizophrenia was observed in whole cerebral cortex $p = 0.03$) and cerebral white matter (p=0.02). Trends to cortical demyelination were found: