SP019

Discerning ADHD from comorbid Austim Spectrum Disorder, and using the Child and Adolescent approach when diagnosing and treating adults with ADHD

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Abstract: Although Autism Spectrum Disorder (ASD) is frequently reported as a comorbidity in adults with Attention Deficit Hyperactivity Disorder (ADHD), diagnosing this additional condition in clinical practice remains challenging. Missing this diagnosis can significantly impact treatment and reduce the quality of life for these adults. In child and adolescent psychiatry, attention is also given to the source(s) that influence the symptoms and complaints of ASD and ADHD, both in diagnosis and treatment.
This lecture highlights the importance of identifying the source(s) of symptoms and complaints in adult patients, with or without ADHD, in an outpatient urban setting. Better mapping of these source(s) helps distinguish the symptoms of ASD from those of ADHD, refining both diagnosis and treatment. Unsurprisingly, ASD is often not even considered a potential comorbid condition in adults. In this lecture, we share practical experiences with adult outpatients and discuss the source(s) that frequently emerge, which aid in differentiating ASD symptoms from those of ADHD.

Disclosure of Interest: None Declared

SP020

Cognitive impairment in psychoses and affective disorders: addressing future challenges

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Abstract: Cognitive dysfunction is well documented in patients with schizophrenia and bipolar disorder, including impairments in attention, memory, executive function and social cognition. These impairments are associated with poor social functioning and reduced activities of daily living.
Various therapeutic interventions have targeted cognitive impairment in both schizophrenic and bipolar patients. Several trials and meta-analyses are currently available. In addition to psychopharmacology, cognitive remediation programmes are available and have been shown to be applicable in clinical practice.
In conclusion, as we move towards more integrative and personalised treatment strategies for schizophrenic and bipolar patients, new assessments and therapies are available to target cognitive dysfunction. Not least because there are currently no cognitive enhancers on the market, it is hoped that new pharmacological adjunctive strategies can improve cognition in schizophrenia and bipolar disorder.

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SP021

Functional dysconnectivity in Schizophrenia and Bipolar Disorder: associations with cognitive impairment

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Abstract: Background: Abnormal cerebellar functional connectivity (FC) has been independently implicated in the pathophysiology of schizophrenia (SCZ) and bipolar disorder (BD). However, the relationship between cerebellar dysconnectivity patterns in these two disorders and their association with cognitive functioning and clinical symptoms have not been fully clarified. In this study, we used the state-of-the-art functional atlas of the cerebellum to examine cerebellar FC changes in the SCZ–BD spectrum and their association with cognitive and clinical variables.
Methods: Resting-state functional magnetic resonance imaging (fMRI) data of 39 individuals with SCZ, 43 BD type I and 61 healthy controls were examined. The cerebellum was parcellated into ten functional systems and we calculated seed-based FC for each cerebellar system. Cognitive abilities were investigated with the Wechsler memory scale, the California Verbal Learning test, the Stroop test, the Attentional network task, the Continuous performance test, the Task Switch task and the Stop Signal task. Psychopathological evaluation was carried out using the Scale for the Assessment of Negative Symptoms and the Scale for the Assessment of Positive Symptoms. We used principal component analyses to reduce the dimensionality of the diagnosis-related FC and cognitive variables, respectively. Multiple regression analyses were conducted to assess the relationship between FC and cognitive and clinical data.
Results: We observed lower cerebellar FC with the frontal, temporal, occipital and thalamic areas in SCZ, and a more widespread decrease in cerebellar FC in BD, involving the frontal, cingulate, parietal, temporal, occipital and thalamic regions. SCZ presented increased within-cerebellum and cerebellar-frontal FC compared to BD. Higher cortico-cerebellar FC was positively associated with memory (p-FWE=0.036) and verbal learning (p-FWE=0.043). Exploratory analyses showed a negative correlation between cortico-cerebellar FC and positive symptoms (p-FWE=0.051).
Conclusions: These findings suggest a role for shared and distinct patterns of corticocerebellar dysconnectivity in the SCZ–BD spectrum that can result in cognitive impairment and psychotic symptoms. In addition, they highlight the potential role of cerebellar stimulation as a promising intervention for individuals with SCZ and BD-I that present cognitive impairment.

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