

for family psychoeducation, psycho-social interventions, and cognitive-behavioural education treatment approaches in individuals with CdLS.

Disclosure of Interest: None Declared

EPP0804

TRAPPC9 deficiency's implication in "secondary" autism spectrum disorders

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Introduction: Autism spectrum disorder (ASD) is a highly heterogeneous neurodevelopmental disorder with many contributing risk genes. Multiple intellectual disability (ID) susceptibility genes have been identified in ASDs to date. The trafficking protein particle complex subunit 9 *TRAPPC9* (OMIM#611966) in an autosomal recessive intellectual disability (ID) gene associated with not fully penetrant phenotype combining secondary microcephaly, dysmorphic facial features, obesity, autism spectrum disorder (ASD) and attention-deficit hyperactivity disorder (ADHD).

Objectives: The aim of this study is to consider *TRAPPC9* deficiency in autosomal recessive ID with ASD.

Methods: We present the observation of two siblings, born to Tunisian consanguineous and healthy parents, followed up for syndromic intellectual disability (ID) associated ASD and microcephaly. A clinical exome sequencing was performed to one child using a Trusight One kit of Illumina. We used sanger sequencing to validate the suspected variant for the other child and to specify the parental segregation.

Results: A homozygous pathogenic variant in the *TRAPPC9* (NM_001160372.4) gene, c.1414C > T (p. Arg472Ter) were identified in one child. Sanger sequencing confirmed the homozygosity profile of this variant for the other child while the parents were both heterozygous carriers.

Conclusions: Repetitive behaviours, especially hand-flapping, were the mean ASD feature in our patients. The current variant is known in the Tunisian population. It is described to lead to the creation of a premature stop codon and a truncating protein causing a *TRAPPC9* deficiency. The impairing neuronal NFκB signalling due to *TRAPPC9* deficiency has been suggested to be implicated in ASD. Due to the profound ID seen in both patients, we suggest the classification of ASD related to *TRAPPC9* deficiency as "secondary" rather than "primary".

Our results support the implication of *TRAPPC9* in secondary ASD and shed the light on the possibility of screening p. Arg472Ter in Tunisian patients with this form of ASD as it is a recurrent mutation in the Tunisian population.

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EPP0805

Bipolar Disorder and Parkinson disease: a 123I-FP-CIT SPECT study

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Introduction: Bipolar Disorder (BD) has been suggested to be a risk factor for development of Parkinson Disease. Psychiatric drugs used as standard treatment of BD includes many drugs that are known to induce drug-induced parkinsonism (DIP).

Objectives: Clinical differentiation between PD and DIP is a clinical and scientific crucial result. It might be aided by functional neuroimaging of the dopaminergic nigrostriatal pathway.

Methods: Twenty consecutive BD patients with parkinsonism were clinically assessed and underwent ¹²³I-ioflupane dopamine transporter SPECT. Imaging data of BD patients with pathological nigrostriatal pathway were further compared to a population of *de-novo* PD patients.

Results: Four BD patients had abnormal scans; they had higher putaminal binding ratio and putamen-to-caudate ratios than PD patients, despite similar motor symptom burden.

Conclusions: in our initial results, up to 20% of BD patients with parkinsonism might have an underlying dopaminergic deficit, which is higher than expected in the general population. This evidence supports that BD represents a risk factor for subsequent development of neurodegenerative parkinsonism.

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EPP0806

Chronic obstructive pulmonary disease and comorbid psychiatric disorders: preliminary results of an 8-year retrospective study using real data

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Introduction: Chronic obstructive pulmonary disease (COPD) is the third leading cause of mortality worldwide. In Portugal, it is estimated to afflict 14.2% of the population over the age of 45 and is one of the most common causes of morbidity, with a significant