S390 e-Poster Presentation

An analysis of environmental factors showed that children were brought up in conditions of insufficient attention, hypopedia. One third of the cases came from incomplete families. About a quarter of the children grew up in large families, were the youngest children of elderly parents.

In heredity, cases of manifest psychosis were not identified. However, an analysis of the personal qualities of parents speaks of schizotypical stigmatization; in almost every family, fathers or mothers had coronary heart disease and joint damage. Insufficient level of education of some parents.

Conclusions: In general, the mental state of children, we can conclude that it corresponds to dysontogenetic with a predominance of schizotypical stigmas in half of them, partial underdevelopment of the sensory and emotional-volitional spheres, similar to disorders in children from conditions of maternal deprivation.

Disclosure of Interest: None Declared

## **EPP0541**

Poor motor skills in childhood predict bully victimization across the lifespan: A study of adults with **Autism Spectrum Disorder** 

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**Introduction:** Children with autism spectrum disorder (ASD) are frequently clumsy and are more likely to be bullied compared to typically developing peers. The link between motor skills and bully victimization is poorly understood.

**Objectives:** The aim of the current study was to evaluate the effect of poor motor skills in childhood on bully victimization from early life to adulthood in those with ASD.

Methods: In this cross-sectional study, 182 adults diagnosed with ASD completed a questionnaire on their recollection of bully victimization at different stages of life and their performance in physical education (PE), as a proxy for motor skills, and academic skills at age 12. Prevalence rates of bully victimization (defined as bullied ≥twice monthly) were compared at different time periods between those with- and without a memory of poor motor skills by chi-square tests. Moreover, logistic regression evaluated the associations while adjusting for candidate covariates sex and academic skills.

Results: Out of the total sample of 182 adults (mean age=33 years, 48% female), 50% reported below average performance in PE. Prevalence rates of bully victimization were more common in those categorized as having poor motor skills as compared to those without poor motor skills in all measured time periods; 72% vs 28% p=.001 in nursery school, 69% vs 31%, p>.001 at 7-9 years, 61% vs 39%, p=.001 at 10-12 years, 64% vs 36%, p>.001 at 13-15 years, 73% vs 27%, p=.005 at 16-18 years and 73% vs 27%, p=.009 in working life. The statistically significant associations seen in the prevalence comparisons remained in the logistic regression models.

Conclusions: The present study adds to the small, but growing, body of literature supporting an association between poor motor skills and bully victimization amongst children and adolescents with ASD. Moreover, we showed that the effect of childhood clumsiness on bully victimization continues into adulthood. Possibly, poor motor skills and social deficits share the same biological pathways and contribute to the risk of being perceived as "different", and consequently bullied, by peers.

Disclosure of Interest: None Declared

## **EPP0542**

## Familial Autism Spectrum Disorder: A clinical study from South Tunisia

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Introduction: Autism Spectrum Disorder (ASD) is a multifactorial neurodevelopmental disorder, with both contribution of genetic and non-genetic factors. A collaboration of de novo mutations and prenatal with postnatal environmental factors are likely to play a role. ASD can be syndromic or non-syndromic. The etiology of non-syndromic ASD is still relatively undefined due to its genetic heterogeneity. Contrary to non-syndromic ASD, syndromic ASD is often associated with chromosomal abnormalities or monogenic alterations. Familial cases of ASD support the strong genetic component of ASD.

**Objectives:** To collect clinical arguments supporting a genetic cause of autism spectrum disorder.

Methods: We present a clinical study of familial cases of ASD. The families were recruited as part of a collaborative project between the department of Medical Genetic and the department of Child Psychiatry in Hedi Chaker Hospital, Sfax, Tunisia. The clinical and paraclinical data were collected retrospectively.

Results: Our study included 11 unrelated families from southern Tunisia, each with two ASD children, among them four couples of twins. Most families (80%) are consanguineous belonging to a middle socioeconomic class. None of the parents suffered from psychiatric disorder and a familial history of autism was reported in one family. Perinatal history, including advanced maternal or paternal age, fetal suffering and/or gestational problems, was found in 35% of cases.

The average age was 9.89  $\pm$  3.787 (3 to 17 years) with a balanced sex-ratio.

ASD was syndromic in seven out of 11 families: facial dysmorphism in half of cases (6/11 families) and/or another comorbidity in 25% of cases (celiac disease, congenital heart disease or idiopathic hydrocephaly). ASD was associated with other(s) neurodevelopmental disorder(s) in all children. Most of cases (14/22) had delayed psychomotor development and all of them had intellectual disability with various degrees. Epilepsy was identified in three cases belonging to unrelated families. Other behavioral problem was identified in 65% of cases.

When the autism spectrum disorder is syndromic and/or associated with other(s) neurodevelopmental(s) disorder(s), this points more towards a genetic origin.

Conclusions: Our study highlights the interest of clinical investigations to determine genetic risk factors of ASD. The identification of a genetic cause in familial cases would contribute not only to