BMI were smaller in DZ (0.20, 95%CI: 0.00 to 0.40), and null in MZ twins (0.07, 95%CI: -0.21 to 0.35).

Image 1:

	Variables	Scales/coding
Main analytic model	Child characteristics	
	Age at outcome	Age of the child when completed outcome
	Sex	0 females; 1 males
	Ethnicity	0 white; 1 ethnic minority
	Family characteristics	
	Composite family SES	5 different derived variables relating to parent qualifications and employment and mother's age at birth of first child
	Life events composite	The five significant life events experienced by the family at age 3
	Maternal depression	EPDS age 3 of the child
	BMI mother	BMI age 3
	BMI father	BMI age 3
	Primary confounders	
	BMI child	Standardised BMI age 14
	Puberty	Petersen Pubertal Development Scale age 12
	Autistic traits	The Autism-Spectrum Quotient (AQ) age 14
	Depressive symptoms	Short-Moods and Feelings Questionnaire (SMFQ) age 12
	Externalising behaviours	Strengths and Difficulties Questionnaire (SDQ) age 12
	Peer victimisation	Multidimensional Peer-Victimization Scale age 12
	Physical activity	Hours playing sport age 12
		Enjoyment while playing sport age 12
Sensitivity analyses	Secondary confounders	
	Chaos at home	CHAOS (Confusion, Hubbub and Order Scale) at home age 12
	Parenting	Parental discipline, derived from Deater-Deckard et al (1998) age 12
		Parental feelings questionnaire (PFQ) age 12
	Genetic	PRS for anorexia nervosa, PRS for schizophrenia + 10 principal components of genetic ancestry

Conclusions: Our findings suggest that greater body dissatisfaction might be a causal risk factor for eating disorders and depression in young people, as associations seen in the full sample persisted in co-twin control analyses. This indicates that body dissatisfaction could be a modifiable target to reduce the risk of these mental health problems in adolescents and young adults. Evidence of associations between body dissatisfaction and increased BMI was weaker in co-twin control analyses than in the full sample. This might be due to larger proportions of shared genetic risk and thus require larger sample sizes to detect.

Disclosure of Interest: None Declared

# **Prevention of Mental Disorders**

### **O122**

Paternal Smoking and Risk of Attention Deficit Hyperactivity Disorder in Children: A Systematic Review and Meta-Analysis

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**Introduction:** Attention Deficit Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental disorder that significantly affects

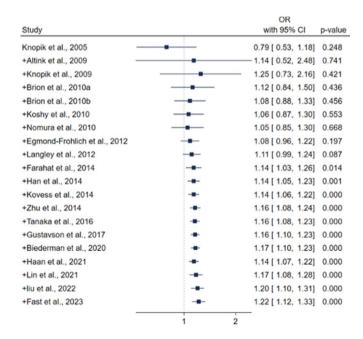
children's behaviour, attention, and academic performance. While the impact of maternal smoking during pregnancy on ADHD risk is well-established, emerging research suggests that paternal smoking may also contribute to this risk. However, the relationship between paternal tobacco use and ADHD remains underexplored, with existing studies presenting mixed results.

**Objectives:** This systematic review and meta-analysis aim to clarify the extent of this association and provide a comprehensive assessment of the evidence available.

**Methods:** All relevant studies in CINAHL, Embase, PsycINFO, PubMed, Scopus, and Web of Science databases were searched from inception until 15 March 2024. Both conventional and cumulative meta-analyses were conducted. Pooled odds ratios with 95% confidence intervals (CIs) were calculated using a random-effects model. The heterogeneity among studies was assessed using the I<sup>2</sup> test, and the presence of small study effects was evaluated using funnel plots and Egger's test. Sensitivity and subgroup analyses were also performed.

**Results:** Twenty observational studies involving over 294, 236 study participants from 16 different countries were included. We found that paternal smoking was associated with a 22% increased risk of ADHD in children (RR=1.22, 95% CI: 1.12, 1.33). The observed association has remained stable since 2014, with minimal fluctuations in effect sizes and their corresponding 95% CIs. Our subgroup analysis revealed that this association is only evident among studies that did not account for maternal smoking (OR=1.23, 95% CI: 1.10, 1.38, n=8), while no increased risk of ADHD was found in studies that adjusted for maternal smoking (OR=1.14, 95% CI: 0.98, 1.33), suggesting that maternal smoking may confound the observed association.

Image 1:



**Conclusions:** Paternal smoking may increase the risk of ADHD in children. Future studies should focus on maternal and paternal

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comparisons to disentangle the independent and combined effects of parental smoking on ADHD risk in children.

Disclosure of Interest: None Declared

## **Child and Adolescent Psychiatry**

#### 0124

# Tobacco Smoke Exposure in Children and Adolescents: Prevalence, Risk Factors and Co-Morbid Neuropsychiatric Conditions in a US Nationwide Study

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**Introduction:** Tobacco smoke exposure(TSE) is a significant public health issue, children and adolescents, who are often involuntarily exposed through secondhand or thirdhand smoke. This exposure has been linked to a range of neuropsychiatric conditions and can negatively impact mental and physical well-being. In the U.S., TSE is prevalent among certain sociodemographic groups, including those with lower income and specific racial backgrounds. The goal of this study was to evaluate the prevalence of TSE and its association with neuropsychiatric comorbidities.

**Objectives:** Assess TSE prevalence among U.S. children and adolescents

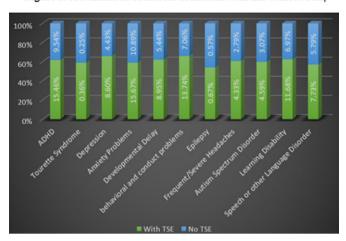
Examine sociodemographic factors influencing TSE

Analyze the link between TSE and neuropsychiatric conditions' prevalence and severity

Methods: This cross-sectional study used data from the 2020-2021 National Survey of Children's Health (NSCH), a survey conducted by the U.S. Census Bureau.Parent-proxy responses were collected in English and Spanish through mail and web-based surveys. A total of 91,404 children aged 0-17 were included for the analysis of TSE prevalence, while 79,182 children aged 3-17 were analyzed for neuropsychiatric comorbidities. The primary measures were TSE, assessed through household smoking behavior, and the presence of neuropsychiatric conditions. Statistical analyses included t-tests, Chi-Square tests, and multivariate regression models to identify associations between TSE, socio-demographic factors, and neuropsychiatric comorbidities, providing adjusted odds ratios and confidence intervals. Statistical analyses were carried out using Stata version 17. Results: TSE was identified in 12.9% of the sample population. The likelihood of TSE was higher among males and adolescents aged 11-17 years. Children from lower-income households and American Indian/Alaska Native backgrounds had a greater risk of TSE. Among children exposed to tobacco smoke, 36.4% had at least one neuropsychiatric comorbidity, with anxiety (15.7%), ADHD (15.5%), and conduct problems (13.7%) being the most common. Females had lower odds of anxiety and autism spectrum disorder(ASD)compared to males, and Asian children exhibited lower odds of ADHD and other conditions. TSE was associated with increased severity of neuropsychiatric conditions. Figure 1.

#### Image 1:

Figure 1. Prevalence of co-morbid conditions with and without TSE



Conclusions: TSE is a significant concern among U.S. children and adolescents, particularly affecting males and those from lower-income families. Exposure to tobacco smoke notably elevates the risk and severity of neuropsychiatric conditions, with ADHD, behavioral and conduct problems, and learning disabilities being the most common co-occurring issues. These findings highlight the importance of early screening and intervention for youth exposed to tobacco smoke. Addressing sociodemographic disparities in exposure and implementing prevention strategies could play a critical role in reducing these negative health outcomes.

Disclosure of Interest: None Declared

### **O125**

Time trends in incidence, comorbidities and sociodemographic risk factors for diagnosed autism spectrum disorder among children and adolescents in Finland

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**Introduction:** ASD is a neurodevelopmental disorder with 1% prevalence among children and adolescents, with high comorbidity with Intellectual Disability. ASD imposes a significant disease burden and is a leading cause of disability among children and adolescents. Its aetiology is multifactorial, including both genetic and environmental factors. Although prior studies have explored the incidence, comorbidities and several prenatal risk factors, research especifically focusing on ASD categorization by ID is limited.