Results: We included five studies with a total of 2,647,536 patients with ADHD (150,741) / ASD (63,726), of whom 271,126 were exposed to acetaminophen prenatally and 2,376,410 were not exposed. Prenatal acetaminophen exposure was associated with an increased risk of developing ADHD (OR 1.30; 95% CI 1.17 to 1.45; p<0.01; I2 = 73%; Figure 1) and ASD (OR 1.17; 95% CI 1.14 - 1.20; p<0.01; I2 = 0%; Figure 2). Sensitivity analyses revealed that acetaminophen exposure during the third trimester of pregnancy was associated with an increased risk of ADHD (HR 1.26; 95% CI 1.07 to 1.47; p<0.01; I2 = 0%; Figure 3), but not during first (HR 1.10; 95% CI 0.97 to 1.26; p=0.13; I2 = 0%; Figure 3) and second (HR 1.07; 95% CI 0.95 to 1.19; p=0.26; I2 = 0%; Figure 3) trimesters.

Image 1:

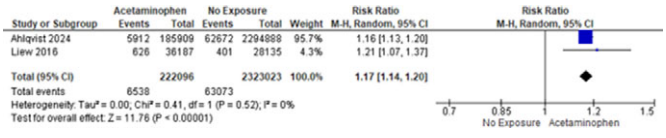
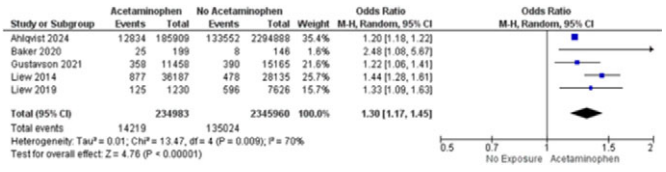


Image 2:



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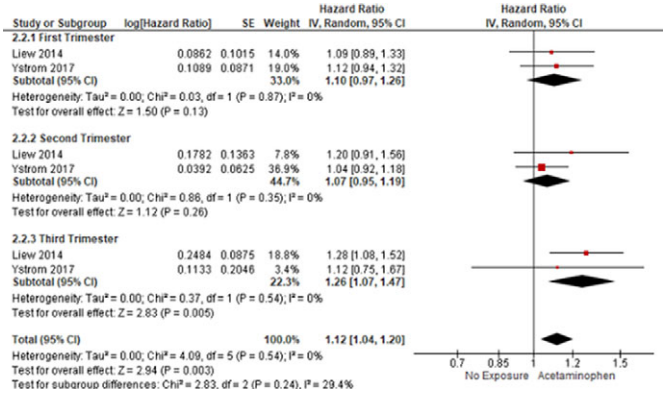

Conclusions: In this systematic review and meta-analysis, prenatal acetaminophen exposure was significantly associated with risk of developing ADHD and ASD, especially if exposure occurs in the third trimester of pregnancy.

Disclosure of Interest: None Declared

O121

The association between body dissatisfaction, depression, eating disorders and BMI: a prospective twin cohort study

I. Costantini[1]*, T. C. Eley[2], N. M. Davies[1], H. Bould[3], C. M. Bulik[4], G. Krebs[5], P. Diedrichs[6], G. Lewis[1], G. Lewis[1], C. Llewellyn[7], D. Nicholls[8], J.-B. Pingault[9] and F. Solmi[1]

[1]Division of Psychiatry, University College London; [2]Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College, London; [3]Population Health Sciences, University of Bristol, Bristol, United Kingdom; [4]Department of Psychiatry, University of North Carolina, Chapel Hill, United States; [5]Research Department of Clinical, Educational and Health Psychology, University College London, London; [6]Centre for Appearance Research, University of the West of England, Bristol; [7]Department of Behavioural Science & Health, University College London; [8]Division of Psychiatry, Imperial College London and [9]Department of Clinical, Educational and Health Psychology, Division of Psychology and Language Sciences, University College London, London, United Kingdom
*Corresponding author.
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Introduction: Body dissatisfaction is becoming more common among adolescents and is a putative risk factor for adverse mental (e.g., eating disorder and depressive symptoms) and physical health outcomes (e.g., excessive weight gain), both of which have also been increasing. Targeting body dissatisfaction through preventative interventions might improve these outcomes, however robust evidence of causal associations is limited.
Objectives: To investigate the association between body dissatisfaction at age 16 and eating disorder symptoms at age 21, as well as depressive symptoms and BMI at ages 21 and 26 using a co-twin control design.
Methods: We used data from the Twins Early Development Study (TEDS) and validated self-report measures. We fitted univariable and multivariable linear mixed effect models adjusting for a comprehensive list of confounding factors (Figure 1) to investigate the association between body dissatisfaction, eating disorder and depressive symptoms, and BMI in the full twin sample. We then repeated these analyses using a co-twin control design, which allows to fully and partially control for genetic confounding in monozygotic (MZ) and dizygotic (DZ) twins, respectively, and for any shared measured and unmeasured environmental factors. We conducted primary analyses in imputed datasets for participants with complete exposure data.
Results: The analytical sample included 2,183 twins (60.2% females, 61.7% DZ twins). In the full twin sample, one unit increase in body dissatisfaction at age 16 was associated with: (i) a 1.80-point increase in eating disorder symptoms at age 21 (95%CI: 1.49 to 2.11), (ii) a 0.59-point increase in depressive symptoms (95%CI: 0.46 to 0.72), and (iii) a 0.20-point BMI increase (95%CI: 0.08 to 0.32) across age 21 and 26 years. In co-twin control analyses, the association between body dissatisfaction and eating disorder symptoms was larger in DZ twins (N=661; 1.72, 95%CI: 1.11 to 2.33) than in MZ twins (N=414, 0.96, 95%CI: 0.15 to 1.77), whereas effect sizes for depressive symptoms were comparable [DZ: (0.56, 95%CI: 0.33 to 0.78); MZ: (0.50, 95%CI: 0.15 to 0.85)]. Associations with

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

B. Dachew[1]*, Y. Damtie[1], G. Ayano[1] and R. Alati[1,2]

[1]School of Population Health, Curtin University, Perth and [2]Institute for Social Sciences Research, The University of Queensland, Brisbane, Australia

*Corresponding author.

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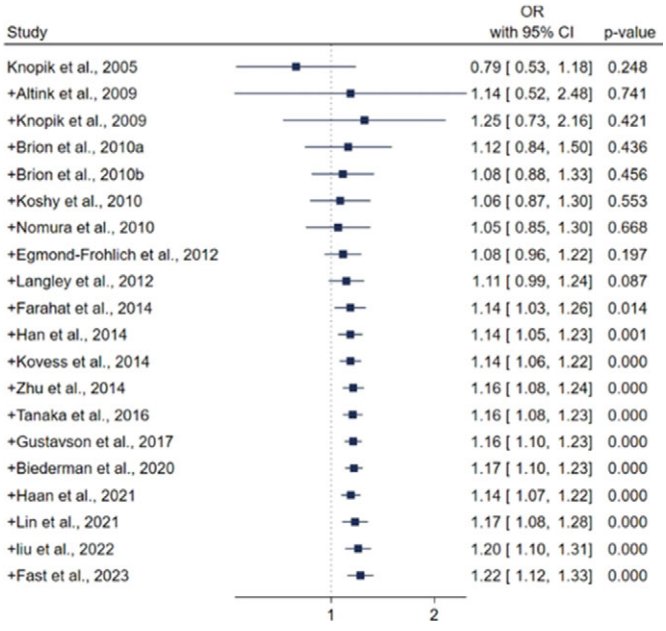
Introduction: Attention Deficit Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental disorder that significantly affects children's behaviour, attention, and academic performance. While the impact of maternal smoking during pregnancy on ADHD risk is well-established, emerging research suggests 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^2 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