

Child and Adolescent Psychiatry

O120

Efficacy and Safety of Cannabidiol Cannabis Extracts for Children and Adolescents with Autism Spectrum Disorder: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

L. Cappelletti Beneti Branco^{1*}, I. Borja De Oliveira², G. A. M. Alves³, M. Teranishi⁴, D. Xavier⁵, E. Sávio Schneider Motta⁶, F. Wagner⁷, M. L. Geremias⁸, A. V. de Vasconcelos⁹, A. C. Gonçalves Tames Zambrana¹⁰ and J. P. De Aquino^{11,12,13}

¹São Camilo University Center; ²School of Medicine, University of São Paulo, São Paulo, Brazil; ³Humanitas University; ⁴Università degli Studi di Milano, Milan, Italy; ⁵ECPE - PPCR Program, Harvard T. H. Chan School of Public Health, Boston, United States; ⁶Federal University of Espírito Santo, Espírito Santo; ⁷School of Medicine, Pontifical Catholic University of Rio Grande do Sul, Porto Alegre; ⁸University of the Joinville Region, Joinville; ⁹Afya College of Medical Sciences of Santa Inês, Santa Inês; ¹⁰Zambrana Clinic, Itajubá, Brazil; ¹¹Department of Psychiatry, Yale University School of Medicine, New Haven; ¹²VA Connecticut Healthcare System, West Haven and ¹³Clinical Neuroscience Research Unit, Connecticut Mental Health Center, New Haven, United States

*Corresponding author.

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Introduction: Autism Spectrum Disorder (ASD) affects approximately 3% of children and adolescents in the U.S. This condition is increasingly prevalent worldwide and presents significant treatment challenges. Preliminary evidence suggests that cannabidiol (CBD) cannabis extracts may help manage ASD symptoms, but their efficacy and potential harms have not yet been systematically investigated.

Objectives: To systematically review and meta-analyze the evidence from clinical trials investigating the efficacy and safety of CBD cannabis extracts in alleviating symptoms of ASD in children and adolescents.

Methods: We conducted a comprehensive search in MEDLINE, Embase, PsycINFO, and the Cochrane Central Register of Controlled Trials using MeSH terms including "Autism Spectrum Disorder," "Cannabidiol," "Cannabis," "Child," and "Adolescents." No language or publication date restrictions were applied. The search was last updated on September 8, 2024. We included randomized, placebo-controlled trials on the efficacy or safety of CBD cannabis extracts in children and adolescents with ASD. For outcomes with limited study data, we used a fixed-effects model. The risk of bias in the included studies was evaluated using the Risk of Bias 2 tool.

Results: Three studies met our criteria, comprising 276 participants (78.3% male; mean age 10.5 years, range 5 to 21). Interventions included orally administered CBD cannabis extracts, with tetrahydrocannabinol (THC) present in minimal amounts or in ratios of 9:1 to 20:1 CBD to THC. Dosages of CBD started at 1 mg/kg per day and were titrated up to 10 mg/kg per day. CBD cannabis extracts significantly enhanced social responsiveness (SMD = -0.75 [-1.08, -0.43], $p < 0.01$, $I^2 = 17\%$), reduced disruptive behavior (SMD = -0.36 [-0.67, -0.06], $p = 0.02$, $I^2 = 0\%$), and alleviated anxiety (SMD

= -0.33 [-0.63, -0.03], $p = 0.03$, $I^2 = 59\%$). CBD cannabis extracts also improved sleep quality, without reaching statistical significance (SMD = -0.19 [-0.49, 0.11], $p = 0.21$, $I^2 = 0\%$). There was no significant difference in adverse effects between interventions and placebo (odds ratio = 2.11 [1.00, 4.46], $p = 0.05$, $I^2 = 38\%$).

Conclusions: CBD cannabis extracts appear to provide meaningful benefits for children and adolescents with ASD, showing moderate improvements in social responsiveness and small yet notable reductions in disruptive behaviors and anxiety. They do not seem to significantly increase adverse effects compared to placebo, suggesting a favorable safety profile. These findings support the potential consideration of CBD cannabis extracts in ASD treatment plans. However, the review's limitations include a small number of studies, limited sample sizes, and significant heterogeneity. Future research with larger, robust trials is needed to clarify the efficacy and safety of CBD cannabis extracts in managing ASD.

Disclosure of Interest: None Declared

O120

Association of Prenatal Acetaminophen Exposure With Risk of ADHD and ASD in Offspring: A systematic review and meta-analysis

D. Carrazzoni Godoi^{1*}, K. Guedes Amorim², R. Góes de Oliveira Galvão³, A. C. Putini Vieira⁴, L. Bussiki Corrêa da Costa Kotecki⁵, D. Abraham Batista da Hora⁶ and S. A. de Souza Júnior, M.D.^{7,8}

¹School of Psychology, Cardiff University, Cardiff, United Kingdom;

²Medical Sciences Center, Federal University of Paraíba, João Pessoa;

³Escola Superior de Ciências da Saúde, Brasília; ⁴Universidade Santo

Amaro, Santo Amaro; ⁵Centro Universitário de Várzea Grande, Várzea

Grande; ⁶Universidade Federal do Amazonas, Manaus; ⁷Centro de

Ciências da Saúde, Curso de Medicina, Universidade de Fortaleza

and ⁸Departament of Physiology and Pharmacology, Faculty of Medicine, Federal University of Ceara, Fortaleza, Brazil

*Corresponding author.

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Introduction: The association between prenatal acetaminophen exposure and the development of Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) remains a subject of considerable debate. Despite extensive research, the evidence regarding this relationship is conflicting.

Objectives: To perform a systematic review and meta-analysis of studies comparing the incidence of ADHD and ASD in patients that were either exposed or not exposed to acetaminophen prenatally.

Methods: We systematically searched Pubmed, Embase and Cochrane Central for eligible studies up until August 2024. Only studies which included participants with a medical diagnosis of ADHD/ASD and reported acetaminophen exposure as a binary measure were included. Available summary data was extracted from published reports and pooled with a random-effects model using odds ratios (OR) with 95% confidence intervals (CI). Hazard ratios (HR) adjusted for potential confounding factors were used for sensitivity analyses. All statistical analyses were conducted utilizing Review Manager 5.4.1. PROSPERO ID:CRD42024587662.

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; $p < 0.01$; $I^2 = 73\%$; Figure 1) and ASD (OR 1.17; 95% CI 1.14 - 1.20; $p < 0.01$; $I^2 = 0\%$; 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; $p < 0.01$; $I^2 = 0\%$; Figure 3), but not during first (HR 1.10; 95% CI 0.97 to 1.26; $p = 0.13$; $I^2 = 0\%$; Figure 3) and second (HR 1.07; 95% CI 0.95 to 1.19; $p = 0.26$; $I^2 = 0\%$; Figure 3) trimesters.

Image 1:



Image 2:

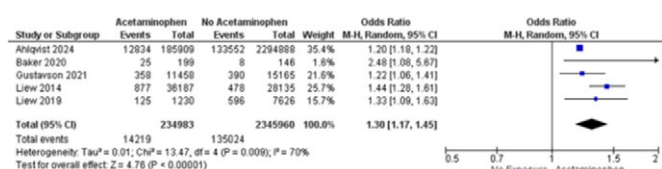
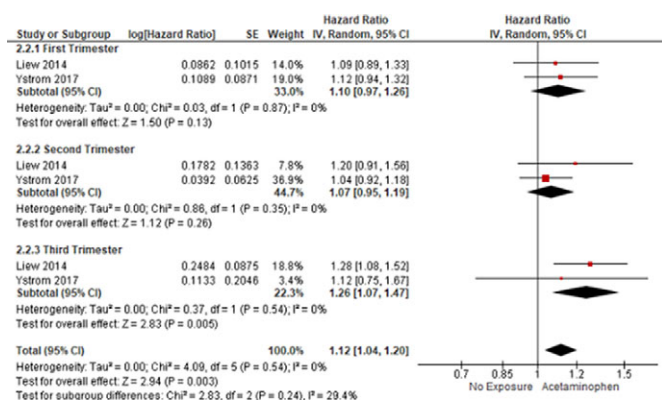


Image 3:



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. Eley², N. M. Davies¹, H. Bould³, C. M. Bulik⁴, G. Krebs⁵, P. Diedrichs⁶, G. Lewis¹, G. Lewis¹, C. Llewellyn⁷, D. Nicholls⁸, J.-B. Pingault⁹ and F. Solmi¹

¹Division of Psychiatry, University College London; ²Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College, London; ³Population Health Sciences, University of Bristol, Bristol, United Kingdom; ⁴Department of Psychiatry, University of North Carolina, Chapel Hill, United States; ⁵Research Department of Clinical, Educational and Health Psychology, University College London, London; ⁶Centre for Appearance Research, University of the West of England, Bristol; ⁷Department of Behavioural Science & Health, University College London; ⁸Division of Psychiatry, Imperial College London and ⁹Department 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