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Prenatal Paracetamol Exposure and Offspring ADHD Risk: A Narrative Review of Evidence, Plausibility, and Implications

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1. Abstract

Background: Paracetamol is the most commonly used analgesic and antipyretic during pregnancy. Concurrently, attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental condition. Recent evidence has raised concerns regarding the potential impact of prenatal paracetamol exposure on offspring ADHD risk.

Objective: To examine the current evidence linking prenatal paracetamol exposure to ADHD, evaluate biological plausibility, and assess clinical and public health implications.

Methods: This narrative review synthesized findings from prospective cohort studies, case–control studies, and meta-analyses published mostly within the past decade and indexed in PubMed, PMC, Lancet.

Results: Several observational cohort studies and meta-analyses report modest associations between prenatal paracetamol exposure and increased risk of ADHD. Meta-analyses and umbrella reviews highlight moderate heterogeneity and substantial vulnerability to residual confounding, particularly confounding by indication and shared familial factors. Importantly, large nationwide cohort studies incorporating sibling-controlled analyses suggest that previously observed relationships may largely reflect genetic, environmental, or maternal health-related confounding rather than causal effects.

Conclusion: Recent reviews emphasize that while observed associations warrant a precautionary approach, the available evidence from observational studies cannot establish a causal link between typical prenatal acetaminophen (paracetamol) use and ADHD diagnosis in children. As such it is still present as first-line treatment, when clinically indicated, used at the lowest effective dose and shortest duration according to regulatory agencies such as EMA.

Keywords: paracetamol, ADHD, pregnancy, neurodevelopment, prenatal

1.2 AI Utilization.

AI was utilized for two specific purposes in this research: 1) Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. 2) Initial organization and synthesis of thematic points from the collected literature. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final selection of literature, critical interpretation of evidence, synthesis of conclusions, and clinical recommendations were determined by the authors. The AI tools served primarily to enhance efficiency in data organization and linguistic refinement.

2. Introduction

Paracetamol Use in Pregnancy: Paracetamol has traditionally been recommended during pregnancy due to its established analgesic and antipyretic effects and perceived favorable safety profile. Estimates suggest that up to >50% of pregnant women use paracetamol at some point, predominantly for pain, fever, or headache. Regulatory and expert bodies typically advise that paracetamol is the first-line pain and fever treatment for pregnant individuals.

Overview of ADHD:

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental disorder with onset typically in childhood. Its core symptoms, which include persistent patterns of inattention, hyperactive behavior, and impulsivity that are inappropriate for the individual's age, often lead to significant difficulties in educational, professional, and interpersonal settings throughout the lifespan [1]. Current estimates suggest that ADHD impacts approximately 7-

10% of children in elementary schools globally, with a substantial number continuing to experience symptoms as adults [1].

The aetiology of ADHD is understood to be multifactorial, involving a complex interplay between genetic susceptibility and environmental influences [1]. Genetic factors play a particularly strong role, with heritability studies indicating that genetics account for about 75% of the risk for developing the condition [2]. Research points to the involvement of numerous genes, many associated with the brain's dopamine and noradrenaline systems, with each gene contributing a small amount of risk—a clear profile characteristic of a polygenic disorder [2]. Non-genetic risks are also significant, especially exposures occurring before and shortly after birth. Factors such as prenatal maternal stress, exposure to environmental toxins, medications, smoking cigarettes, diet factors, perinatal complications, and early immune system activation can interact with an individual's genetic background to shape neurodevelopment and influence ADHD risk [2,3]. Additionally using ADHD medication posed as a hypothetical risk factor.[3]

Despite considerable progress in identifying these risk factors, the diagnosis of ADHD remains clinical, based on observed behavior and reported history, as there are no reliable biological markers [1]. Consequently, treatment strategies are primarily aimed at managing symptoms rather than addressing an underlying cause, underscoring a key gap between scientific discovery and the development of tailored interventions in clinical practice [1].

Rationale for Investigating the Link: Concerns regarding prenatal paracetamol exposure have arisen from cohort and mechanistic studies indicating potential disruption of fetal neurodevelopment. Conflicting evidence and residual confounding necessitate a comprehensive review to inform clinical guidance and research priorities.

3. Pharmacology of Paracetamol in Pregnancy

Mechanism of Action: Paracetamol (acetaminophen) exerts analgesic and antipyretic effects through mechanisms involving central inhibition of cyclooxygenase (COX) enzyme activity in the central nervous system and modulation of descending serotonergic pathways, although its precise molecular mechanisms remain incompletely defined. Central inhibition of prostaglandin synthesis via COX enzymes likely contributes to its pain-relieving and fever-reducing actions, and paracetamol has been shown to increase serotonergic neurotransmission, which may enhance its antinociceptive effects in the spinal cord and brain.

Additional central pathways, including interactions with the endocannabinoid system and other neurotransmitter systems, have been implicated in its pharmacological profile [4,5,6].

Placental Transfer and Fetal Exposure: Paracetamol (acetaminophen) is known to cross the human placenta with high efficiency. Studies using an ex vivo perfusion model of the human placenta have demonstrated that paracetamol moves readily in both directions, from mother to fetus and from fetus to mother, with transfer reaching a state of equilibrium between the two compartments during the experimental period. This finding confirms that the drug undergoes significant bidirectional transfer. In the same model, the measured transfer efficiency was approximately 45% for both directions (relative transfer rate under ex vivo conditions).[7] However, the major phase II metabolites of paracetamol—paracetamol sulfate and paracetamol glucuronide—exhibited a markedly lower rate of placental transfer. This reduced transfer is consistent with the physicochemical properties of these metabolites, as they are larger molecules and more hydrophilic than the parent drug [7].

Metabolism in Pregnant Women and Fetuses: The metabolic pathways of glucuronidation and sulfation, essential for detoxification in the mother's liver. Whereas sulfation seemingly is the best developed, glucuronidation is developmentally immature in the fetus. This functional deficit may potentially heighten fetal vulnerability to harmful reactive metabolites [8].

4. Epidemiological Evidence

The epidemiological literature examining the association between prenatal paracetamol exposure and ADHD in offspring is extensive but characterized by mixed findings, methodological heterogeneity, and ongoing debate regarding the role of confounding.

4.1 Issues in epidemiological findings

A major limitation of prenatal paracetamol (acetaminophen) studies is confounding by indication, where maternal conditions like fever, infections, or pain that prompt its use may independently affect fetal neurodevelopment via inflammation or other pathways [9,10,11]

4.2 Sibling-Controlled and Advanced Design Studies

The sibling-controlled analyses conducted in 2013 showed that exposure for more than 28 days during pregnancy was linked to poorer psychomotor and behavioral scores at 3 years of age, including effects on gross motor skills, communication, and activity level. [12]

Recent sibling-controlled analyses have challenged the long-held assumption that prenatal acetaminophen exposure directly causes neurodevelopmental disorders. Emerging evidence indicates that shared familial factors—such as genetic predispositions and environmental influences common to siblings—may largely account for the associations observed in earlier research.

Large-scale studies employing within-family designs provide the strongest support for this interpretation. For instance, Gustavson et al.[13] conducted a sibling comparison analysis using more than 12,000 sibling pairs from the Norwegian Mother, Father and Child Cohort Study (MoBa)[14]. While conventional models suggested a small association between prenatal acetaminophen exposure and later ADHD diagnosis, this association was greatly attenuated and no longer statistically significant when comparing siblings with differing exposure histories, demonstrating the influence of familial confounding [13].

Similarly, a population-based Swedish study conducted by Ahlqvist et al. examined 2.4 million children to assess the relationship between acetaminophen use during pregnancy and ADHD, autism spectrum disorder, and intellectual disability. In conventional cohort analyses, modest associations were observed; however, sibling-comparison models, which control for shared family background, substantially attenuated these associations [9].

Taken together, these studies indicate that the previously reported risks of prenatal acetaminophen exposure are more accurately attributed to underlying familial factors rather than a direct causal effect of the medication. While these findings significantly reduce concern regarding typical acetaminophen use during pregnancy, they do not entirely exclude the possibility of rare, prolonged, context-specific effects. The authors concluded that the associations likely reflected confounding by genetic or environmental factors rather than a causal pharmacological effect. This finding has been supported by other comprehensive reviews [15].

4.3 Meta-Analyses and Systematic Reviews

Several systematic reviews and meta-analyses have sought to synthesize the observational evidence on prenatal paracetamol (acetaminophen) exposure and child neurodevelopment. A large systematic review and meta-analysis published in 2025 pooled data from 16 observational studies and found a modest but statistically significant association between in utero acetaminophen exposure and attention-deficit/hyperactivity disorder (ADHD) in offspring

(pooled odds ratio [OR] approximately 1.17, 95% CI 1.08–1.27) [16]. The authors reported substantial between-study heterogeneity and emphasized the difficulty of fully accounting for residual confounding, particularly confounding by indication, as paracetamol is commonly used during pregnancy for conditions such as fever, pain, and infection. Earlier reviews of the literature, including a comprehensive narrative and systematic review published in 2018, concluded that most prospective cohort studies observed associations between prenatal paracetamol exposure and adverse neurodevelopmental outcomes, while highlighting important methodological limitations and the challenges of causal inference in this body of observational research, however conclusions of the article were to limit indiscriminate use of paracetamol during pregnancy [17].

A 2025 umbrella review using the Navigation Guide framework synthesized existing research and found that studies generally report a link between paracetamol use during pregnancy and the later development of conditions like ADHD and autism in children [18]. The review authors cautioned that this evidence, largely from observational data, is not conclusive for establishing cause and effect, due to persistent issues like unmeasured confounding and inaccuracies in recording exposure. The authors also stated a need for further research and recommended using paracetamol only when needed at a lowest dose, only for as long as it is medically necessary. [18].

4.4 Long term usage and epidemiological findings

Long-term prenatal paracetamol exposure has been associated with adverse neurodevelopmental outcomes in children. Registry-based analyses reported higher hazard ratios for ADHD diagnoses in children with prolonged prenatal acetaminophen exposure [19]. Analysis of data from the Danish National Birth Cohort indicated that acetaminophen use during pregnancy was associated with an increased risk of a hospital diagnosis of hyperkinetic disorder in offspring, with longer duration of use corresponding to higher hazard ratios (“hazard ratio, 1.84; 95% CI, 1.39-2.45” for use exceeding or equal to 20 weeks) [19].

An umbrella review of systematic reviews similarly noted that while cohort studies report associations between maternal acetaminophen use and ADHD or autism spectrum disorder in children, the overall quality of evidence is low and causality remains uncertain [11]. These findings suggested a potential dose-response relationship, wherein prolonged or frequent use

was linked to higher risk estimates, although this pattern was not consistently observed in sibling-controlled analyses.

Multi-cohort meta-analyses further suggest modestly increased odds of borderline or clinical ADHD symptoms following prenatal acetaminophen exposure, with variability across studies depending on confounder adjustment [20].

4.5 Case–Control Studies

Observational studies, particularly retrospective cohort and registry studies, generally align with cohort findings but are subject to recall bias—especially when maternal self-report is used to ascertain ADHD medication use during pregnancy. This type of bias occurs because mothers of children with ADHD might recall or report exposure differently than mothers of neurotypical children.[21] Their contribution can support an association but does not resolve causality.

4.6 Exposure Assessment and Biomarker Studies

A key advancement is the move toward biomarker-based exposure. For example, studies measuring paracetamol metabolites in cord blood or meconium provide a more objective measure of fetal exposure than maternal self-report [22].

One such study found that the detection of paracetamol metabolites in cord plasma was associated with a significantly increased risk of childhood attention-deficit/hyperactivity disorder (ADHD). Specifically, in comparison with children in the lowest tertile of cord paracetamol burden, those in the highest tertile had approximately 2.9-times greater odds of an ADHD diagnosis (“odds ratio [OR] for second tertile, 2.26; 95% CI, 1.40-3.69; OR for third tertile, 2.86; 95% CI, 1.77-4.67”) [23]. Cord paracetamol burden was also associated with increased odds of autism spectrum disorder (ASD) in a dose-response manner [23]. While the use of biomarker-based exposure assessment reduces misclassification compared with self-reported measures, it does not entirely eliminate confounding by indication, or shared familial risk factors. These findings align with earlier epidemiological research linking prenatal paracetamol exposure with behavioral problems and hyperkinetic disorders in children [19]. While this method reduces exposure misclassification, it does not eliminate confounding by indication, a limitation underscored by the null findings from sibling-controlled designs.

5. Confounding Factors and Bias

The interpretation of epidemiological evidence is inseparable from a thorough consideration of confounding and bias:

5.1 Confounding by Indication: The primary methodological challenge in linking prenatal paracetamol use to neurodevelopmental outcomes is confounding by indication. As argued by Bauer et al. [24], the underlying maternal conditions that lead to analgesic use—such as infections, inflammatory states, or high fever—are themselves associated with an increased risk of adverse outcomes in offspring. This risk may be mediated through pathways like maternal immune activation. Therefore, associations observed in epidemiological studies may reflect this confounding rather than a direct pharmacological effect. This concern is substantiated by separate meta-analytic evidence, such as that from Antoun et al. [25], which confirms that maternal fever during pregnancy is a risk factor for neurodevelopmental disorders.

5.2 Familial Confounding: Observed associations between prenatal use of common pain relievers and neurodevelopmental outcomes in children may be partly influenced by familial and genetic factors. Parents with traits associated with conditions like ADHD may be more likely to use analgesics during pregnancy and also pass on genetic susceptibility to their children, creating potential confounding. Population-level studies have shown links between paracetamol use and autism, ADHD, and intellectual disability, but sibling-controlled analyses indicate these associations largely reflect shared familial factors rather than causal effects [9]. Earlier sibling-controlled research found that long-term prenatal exposure may still be associated with subtle motor or behavioral differences, highlighting that some specific developmental effects cannot be fully ruled out [12].

5.3 Socioeconomic and Lifestyle Factors: Variables such as maternal stress, nutrition, smoking, and co-use of other medications are unevenly distributed and difficult to fully measure and adjust for statistically.

Exposure Misclassification: Reliance on maternal recall, often years after pregnancy, leads to non-differential misclassification or differential recall bias.

Diagnostic Bias: Variability in ADHD diagnostic and management practices has been observed across both the United States and Europe, contributing to heterogeneity in outcomes. In the

U.S., reliance on parent-reported provider diagnoses versus standardized clinical assessments can affect the validity and reliability of surveillance data, influencing the consistency and comparability of measured outcomes [26]. Similarly, European research indicates that the clinical approach to ADHD varies significantly between clinics and countries. Key differences include the types of treatment initiated after diagnosis, the outcome of chosen treatment, switch and or discontinuation of medication, overall approach to the diagnosis, pointing to a lack of standardization in how the condition is identified and managed [27]

6. Conclusion of Epidemiological Evidence

In summary, the body of epidemiological evidence presents a complex picture. Conventional observational studies consistently report modest associations between prenatal paracetamol exposure and increased risk of ADHD, with suggestive dose-response patterns. This consistency initially raised legitimate concerns. However, studies employing stronger designs to control for unmeasured familial confounding—particularly sibling-controlled analyses—consistently show that these associations attenuate to the null [9,11,28]. This pattern strongly indicates that the observed links in earlier research are likely not causal but are instead driven by shared genetic, environmental, or maternal health factors that predispose mothers to use paracetamol and predispose children to develop ADHD. Therefore, while the association is a reproducible finding in epidemiology, the most methodologically robust available evidence does not support the conclusion that prenatal paracetamol exposure is a direct cause of ADHD in offspring.

Disclosure

Author's Contribution

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Software: not applicable

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