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PREGNANCY Society for Maternal-Fetal Medicine

SMFM STATEMENT

Society for Maternal-Fetal Medicine Statement: Acetaminophen use during pregnancy

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Abstract

Acetaminophen has long been considered safe to use during pregnancy. A growing body of cohort studies and meta-analyses has explored the potential association between prenatal acetaminophen use and neurodevelopmental outcomes in children. Although some studies report modestly increased risks of neurodevelopmental disorders in children exposed to acetaminophen prenatally, these findings must be interpreted with caution, given the inherent limitations of observational studies that may significantly affect the validity of the observed associations. While advanced statistical methods can help reduce bias, they cannot fully account for unmeasured or familial confounding. The Society for Maternal-Fetal Medicine continues to advise that acetaminophen is the recommended first-line medication for the treatment of pain and fever during pregnancy.

KEYWORDS

attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), fever, neurodevelopmental disorders, pain, pregnancy

1 | INTRODUCTION

Acetaminophen is an over-the-counter medication commonly used during pregnancy for the management of fever and pain [1]. Untreated fever during pregnancy, especially during the first trimester, poses risks to the fetus [2–4], and untreated pain negatively impacts maternal well-being [5–10]. Large surveys have reported that up to 65% of pregnant individuals use acetaminophen at some time during their pregnancy, with headache and fever being the most common indications [6].

Some observational studies have reported an association between acetaminophen use during pregnancy and increased risk for adverse neurodevelopmental outcomes, such as attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) in offspring. In 2015, the United States Food and Drug Administration (FDA) released a statement advising that "the weight of evidence is inconclusive regarding a possible connection between acetaminophen use in pregnancy and ADHD in children." [7]

The Society for Maternal-Fetal Medicine (SMFM) affirmed in 2017 that acetaminophen is considered safe for use during pregnancy [8]. Since the FDA and SMFM statements were published, more research has been completed, and additional scrutiny has been placed on the potential risks of acetaminophen use during pregnancy. While SMFM's recommendations remain unchanged, the reinvigorated discussion in scientific journals and popular media merits an updated statement. This guidance summarizes what is known about prenatal acetaminophen use and neurodevelopmental outcomes in offspring to aid clinicians and patients in shared decision-making.

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2 | ORIENTATION TO THE EVIDENCE AND EXISTING RESEARCH

Several categories of observational research studies have explored potential associations between prenatal acetaminophen exposure and neurodevelopmental outcomes. (See Supporting Information, Table 1, for study details.) These include large prospective cohort and case-control studies that compare neurodevelopmental outcomes in children who were or were not exposed to acetaminophen during pregnancy; sibling comparison and genetically-informed analyses that assess whether familial, genetic, or environmental factors explain observed associations; and biomarker-based studies that quantify acetaminophen or its metabolites in cord blood, maternal blood, or maternal urine. In addition, multiple systematic reviews and meta-analyses have pooled data from these observational studies to generate summary estimates. (See Supporting Information, Table 2, for meta-analysis details.)

Drawing firm conclusions from the published data requires considering how accurately each study ascertained exposure to acetaminophen; defined and identified adverse neurodevelopmental outcomes; and statistically adjusted for key confounders, including the indication for acetaminophen use (e.g., pain, fever), other maternal characteristics, and shared familial or genetic predispositions. In this Statement, we summarize the findings from major studies and discuss the strengths and limitations of the overall evidence base. (See the Supporting Information, Search Strategy, for details on study inclusion.)

2.1 | Population-based prospective cohort and case-control studies

Dozens of prospective cohort and nested case-control studies, mainly from North America, Europe, and Asia, have evaluated associations between acetaminophen exposure and neurodevelopmental outcomes in offspring (see Table S1). These studies generally report small to modest effect estimates for adverse neurobehavioral outcomes, including ADHD; ASD; and related cognitive, behavioral, or executive function deficits. Across this body of work, several methodological patterns and limitations exist.

2.1.1 | Heterogeneity of outcome ascertainment and measurement tools

In the published literature, neurodevelopmental outcomes have been variably assessed using parent-reported behavioral checklists, standardized psychometric instruments, or clinician-verified registry diagnoses. While registry data provide diagnostic specificity, they may under-ascertain milder and subthreshold phenotypes. Conversely, parental questionnaires are sensitive to reporting bias and social context. The resulting heterogeneity in outcome ascertainment amplifies between-study variability and complicates meta-analytic synthesis.

2.1.2 | Confounding by indication and maternal illness

The conditions treated with acetaminophen (e.g., fever, migraine, musculoskeletal pain) may independently influence fetal neurodevelopment through inflammatory or metabolic pathways [9, 10]. Although most analyses adjust for maternal fever, infection, and psychiatric comorbidity, these variables are often self-reported, incompletely measured, or temporally misaligned with medication exposure. Statistical adjustment typically attenuates but rarely eliminates the observed associations, suggesting that residual confounding (i.e., confounding that remains even after adjusting for measured variables) likely contributes to the remaining association.

2.1.3 | Misclassification of exposure and its directionality

Most available studies rely on maternal recall of acetaminophen use during pregnancy, often collected postpartum, through structured questionnaires or interviews. This approach introduces potential exposure misclassification, in which participants may inaccurately recall medications taken many months earlier. Moreover, few studies capture dose, frequency, or timing of use with sufficient precision to permit detailed dose-response or critical window analyses. As a result, exposure assessment is often coarse, limiting causal inference and comparability across cohorts. This shortcoming leads to both non-differential misclassification—in which errors in measuring acetaminophen use or diagnoses of ADHD or ASD occur similarly across groups and are unrelated to true disease or exposure, typically biasing estimates toward the null—and differential misclassification—in which recall differs by ADHD or ASD status, potentially inflating associations if those with affected children remember exposures more accurately.

2.1.4 | Inconsistent diagnostic criteria for neurodevelopmental outcomes over time

The diagnostic criteria for ASD [11, 12] and ADHD [13, 14] have substantially evolved over recent decades,

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transitioning from narrow categorical diagnoses under the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), 3rd edition [15], and DSM-IV [16, 17] to broader spectrums under DSM-5 [18] and the *International Classification of Diseases*, 11th revision [19]. Such definitional shifts complicate temporal and cross-study comparisons.

2.1.5 | Small effect sizes and residual uncertainty

Even when statistically significant, the absolute risk increases for ADHD or ASD associated with reported prenatal acetaminophen use are small. In epidemiologic terms, such modest increases can result from unmeasured or poorly measured cofounding, exposure misclassification, or selection bias. Disentangling genuine pharmacologic effects from correlated familial, behavioral, or environmental factors, therefore, remains challenging.

2.2 | Cohort studies employing sibling comparison and genetically informed designs

To address residual confounding by shared familial factors (e.g., genetics, socioeconomic status, parental psychopathology), some cohort studies have employed sibling comparison and genetically informed/negative-control cohort designs. These approaches compare siblings discordant for prenatal acetaminophen exposure or adjust for parental polygenic risk scores. Within-family (sibling-comparison) analyses generally find no association with acetaminophen use and neurodevelopment outcomes, suggesting that shared familial factors, rather than acetaminophen itself, may account for the observed associations. This interpretation is supported by the well-established familial contributions to neurobehavioral disorders such as ADHD and ASD [20, 21].

Similarly, genetically informed cohort studies incorporating parental polygenic risk scores for ADHD and ASD suggest that parental genetic factors may contribute to observed associations between reported prenatal acetaminophen use and neurodevelopmental outcomes. While some studies have examined this question, none have incorporated both maternal and child ADHD or ASD polygenic risk scores within the same analytic model of acetaminophen exposure and neurodevelopmental outcomes to conclusively show that genetic factors explain the observed associations.

2.3 | Observational biomarker studies

Biomarkers of acetaminophen exposure in maternal plasma, maternal urine, cord blood, and meconium have also been explored. Most biomarker studies have relied on a single sample. Because acetaminophen has an elimination half-life of 2–3 h in adults [22, 23], detectable concentrations reflect recent use (within the past 24–48 h). Additionally, acetaminophen is commonly administered during labor and postpartum, and the prevalence of measurable exposure approaches universality in many populations. Temporal misclassification is therefore inevitable, and total fetal exposure over the course of the pregnancy cannot be calculated. These limitations underscore that biomarker-based evidence, though novel in its method of quantifying exposure, cannot by itself establish temporally specific or dose-dependent associations between maternal acetaminophen use and offspring neurodevelopment. Additionally, variation in the reported prevalence of unchanged acetaminophen in cord blood within the same study cohort also raises questions about this emerging methodology [24, 25].

2.4 | Systematic reviews and meta-analyses

Meta-analyses can enhance statistical power and yield more precise estimates of risk than individual studies, making them indispensable tools for evidence synthesis. Their validity, however, depends heavily on the quality, consistency, and methodological rigor of the included studies. Between 2018 and 2025, several meta-analyses evaluated the relationship between prenatal acetaminophen exposure and adverse neurodevelopmental outcomes, collectively providing a framework for interpreting a heterogeneous body of evidence. For clinicians and policymakers, these analyses are particularly valuable because they aggregate findings across diverse populations and study designs, enabling a more balanced assessment of potential risks versus benefits. In the context of widespread acetaminophen use during pregnancy, relying on this high-level analysis helps ensure that guidelines are grounded in the best available data, mitigating overreliance on isolated studies that may be underpowered, confounded, or inconsistent.

Collectively, meta-analyses demonstrate small and inconsistent associations between prenatal acetaminophen use and neurodevelopmental outcomes. Across pooled analyses, relative risks average 1.2–1.3 for ADHD and 1.1–1.2 for ASD (see Table S2). These modest associations are accompanied by moderate-to-substantial

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heterogeneity and persistent residual confounding, particularly by maternal illness, fever, infection, and genetic predisposition. These findings emphasize the broader limitations of meta-analyses, which remain constrained by the methodological weaknesses of the included studies. Imprecise assessment of acetaminophen exposure, poorly defined neurodevelopmental outcomes, and incomplete control for confounding variables in the primary studies limit the ability to draw firm conclusions from these meta-analyses. Additionally, meta-analysis authors have observed that sibling-comparison studies have yielded null associations, suggesting that familial and genetic confounding likely account for much of the observed associations reported in individual studies. Overall, while meta-analyses provide a quantitative summary, they do not establish a causal relationship between acetaminophen use in pregnancy and neurodevelopmental outcomes. The available data do not support changes to clinical practice.

3 | TRANSLATING EVIDENCE INTO CLINICAL PRACTICE

Taken together, the diverse study designs in the published literature highlight the inherent difficulty in establishing a definitive link between prenatal acetaminophen use and neurodevelopmental outcomes. Current evidence does not support a causal relationship between maternal acetaminophen exposure and ADHD, ASD, or other adverse neurodevelopmental outcomes. Untreated maternal fever carries well-documented risks for the fetus, especially in the first trimester [2, 4, 26–28], and can be a sign of serious maternal health problems at any time during pregnancy [29]. Moreover, large cohort studies have reported associations between maternal fever and an increased risk of neurodevelopmental outcomes [9]. Treating pain and fever during pregnancy is important to promote overall maternal well-being [5, 30, 31]. Common alternative medications for fever and pain during pregnancy (e.g., nonsteroidal anti-inflammatory drugs, opioids) carry additional risks, and their use is often limited depending on the clinical circumstances [32]. Given the established benefits of treating fever and pain in pregnancy, SMFM continues to recommend acetaminophen as the first-line analgesic and antipyretic in pregnancy.

4 | COUNSELING AND SHARED DECISION-MAKING

Clinicians should contextualize the small relative risks reported in observational studies against the well-established safety and efficacy profile of acetaminophen for both the pregnant person and fetus. Key counseling messages include the following:

- The absolute risk of neurodevelopmental disorders associated with acetaminophen use is low across the published studies. The vast majority of exposed children did not develop ASD or ADHD, and most individuals who use acetaminophen in pregnancy will not have children with neurodevelopmental disorders. There are significant limitations of the current evidence base, and reported associations do not establish causality.
- A pregnant patient's unique medical history can independently influence fetal neurodevelopment.
- It is important to treat maternal fever and pain during pregnancy. Untreated maternal fever in the first trimester is a known teratogen linked to congenital anomalies and miscarriage. Maternal fever during labor is also associated with poor neonatal outcomes, including low Apgar scores, sepsis, neonatal intensive care unit admission, and hypoxic-ischemic encephalopathy [33]. Patients should consult their medical provider about concerning fevers at any time during pregnancy.
- Acetaminophen remains the first-line antipyretic and analgesic in pregnancy because alternatives such as nonsteroidal anti-inflammatories may increase the risks of fetal renal impairment, premature closure of the ductus arteriosus, oligohydramnios, and maternal bleeding, and opioids are associated with neonatal abstinence syndrome, addiction, and sedation.
- As with all medications taken during pregnancy, patients should use the lowest effective dose of acetaminophen for the shortest duration necessary.
- Patients should carefully check medication labels and be aware that many combination and multi-ingredient products (e.g., cold and flu medications) contain acetaminophen. Patients who take a combination product containing acetaminophen plus standard acetaminophen may unknowingly exceed the maximum recommended daily dose (generally 4000 mg/day for healthy adults).
- In conjunction with acetaminophen use, nonpharmacologic measures such as rest, hydration, and physical therapy may also be appropriate for managing pain during pregnancy.

Ultimately, providers should give patients information regarding the current evidence available on both the use of acetaminophen in pregnancy and the risks of not treating the condition for which acetaminophen would be used. The available evidence base continues to support the safety of maternal acetaminophen use during pregnancy.

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5 | CONCLUSION

This SMFM Statement is based on a comprehensive review of the available evidence regarding acetaminophen use during pregnancy. Although some studies have reported associations between maternal acetaminophen use and adverse neurodevelopmental outcomes in offspring, methodological limitations preclude conclusions about causality, and the biological mechanism for such an effect remains unestablished. Future research should incorporate parental psychiatric and neurobehavioral diagnoses to better account for heritable risk factors. SMFM continues to advise that acetaminophen is the recommended first-line medication for the treatment of pain and fever during pregnancy.

REFERENCES

1. Chambers, C. 2015. "Over-the-Counter Medications: Risk and Safety in Pregnancy." *Seminars in Perinatology* 39(7): 541–4. https://doi.org/10.1053/j.semperi.2015.08.009.
2. Moretti, M. E., B. Bar-Oz, S. Fried, and G. Koren. 2005. "Maternal Hyperthermia and the Risk for Neural Tube Defects in Offspring: Systematic Review and Meta-Analysis." *Epidemiology* 16(2): 216–9. https://doi.org/10.1097/01.ede.0000152903.55579.15.
3. Shahrukh Hashmi, S., M. S. Gallaway, D. K. Waller, P. H. Langlois, and J. T. Hecht. 2010. "Maternal Fever During Early Pregnancy and the Risk of Oral Clefts." *Birth Defects Research Part A: Clinical and Molecular Teratology* 88(3): 186–94. https://doi.org/10.1002/bdra.20646.
4. Shi, Q. Y., J. B. Zhang, Y. Q. Mi, Y. Song, J. Ma, and Y. L. Zhang. 2014. "Congenital Heart Defects and Maternal Fever: Systematic Review and Meta-Analysis." *Journal of Perinatology* 34(9): 677–82. https://doi.org/10.1038/jp.2014.76.
5. Whitten, C. E., M. Donovan, and K. Cristobal. 2005. "Treating Chronic Pain: New Knowledge, More Choices." *Permanente Journal* 9(4): 9–18. https://doi.org/10.7812/tpp/05-067.
6. Hoover, R. M., V. A. Hayes, and J. Erramouspe. 2015. "Association Between Prenatal Acetaminophen Exposure and Future Risk of Attention Deficit/Hyperactivity Disorder in Children." *Annals of Pharmacotherapy* 49(12): 1357–61. https://doi.org/10.1177/1060028015606469.
7. U.S. Food and Drug Association. FDA Drug Safety Communication: FDA Has Reviewed Possible Risks of Pain Medicine Use During Pregnancy. Updated January 9, 2015. Accessed April 29, 2026. http://www.sefap.it/web/upload/FDA_farmaci_per_il_dolore_in_gravidanza.pdf.
8. Society for Maternal-Fetal Medicine. 2017. "Prenatal Acetaminophen Use and Outcomes in Children." *American Journal of Obstetrics and Gynecology* 216(3): B14–B5. https://doi.org/10.1016/j.ajog.2017.01.021.
9. Antoun, S., P. Ellul, H. Peyre, M. Rosenzwajg, P. Gressens, D. Klatzmann, and R. Delorme. 2021. "Fever During Pregnancy as a Risk Factor for Neurodevelopmental Disorders: Results From a Systematic Review and Meta-Analysis." *Molecular Autism* 12(1): 60. https://doi.org/10.1186/s13229-021-00464-4.
10. Kwon, H. K., G. B. Choi, and J. R. Huh. 2022. "Maternal Inflammation and Its Ramifications on Fetal Neurodevelopment." *Trends in Immunology* 43(3): 230–44. https://doi.org/10.1016/j.it.2022.01.007.
11. Tsai, L. Y. and M. Ghaziuddin. 2014. "DSM-5 ASD Moves Forward into the Past." *Journal of Autism and Developmental Disorders* 44(2): 321–30. https://doi.org/10.1007/s10803-013-1870-3.
12. Doernberg, E. and E. Hollander. 2016. "Neurodevelopmental Disorders (ASD and ADHD): DSM-5, ICD-10, and ICD-11." *CNS Spectrums* 21(4): 295–9. https://doi.org/10.1017/s1092852916000262.
13. Austerman, J. 2015. "ADHD and Behavioral Disorders: Assessment, Management, and an Update From DSM-5." *Cleveland Clinic Journal of Medicine* 82(11 suppl 1): S2–S7. https://doi.org/10.3949/ccjm.82.s1.01.
14. Ophir, Y. 2024. "Reevaluating ADHD and Its First-Line Treatment: Insights From DSM-5-TR and Modern Approaches." *Clinical Neuropsychiatry* 21(5): 436–43. https://doi.org/10.36131/cnfioritieditore20240507.
15. Volkmar, F. R., D. J. Cohen, and R. Paul. 1986. "An Evaluation of DSM-III Criteria for Infantile Autism." *Journal of the American Academy of Child Psychiatry* 25(2): 190–7. https://doi.org/10.1016/s0002-7138(09)60226-0.
16. Willcutt, E. G., J. T. Nigg, B. F. Pennington, M. V. Solanto, L. A. Rohde, R. Tannock, S. K. Loo, C. L. Carlson, K. McBurnett, and B. B. Lahey. 2012. "Validity of DSM-IV Attention Deficit/Hyperactivity Disorder Symptom Dimensions and Subtypes." *Journal of Abnormal Psychology* 121(4): 991–1010. https://doi.org/10.1037/a0027347.
17. First, M. B. and H. A. Pincus. 2002. "The DSM-IV Text Revision: Rationale and Potential Impact on Clinical Practice." *Psychiatric Services* 53(3): 288–92. https://doi.org/10.1176/appi.ps.53.3.288.
18. Kulage, K. M., J. Goldberg, J. Usseglio, D. Romero, J. M. Bain, and A. M. Smaldone. 2020. "How Has DSM-5 Affected Autism Diagnosis? A 5-Year Follow-Up Systematic Literature Review and Meta-Analysis." *Journal of Autism and Developmental Disorders* 50(6): 2102–27. https://doi.org/10.1007/s10803-019-03967-5.
19. Stein, D. J., P. Szatmari, W. Gaebel, M. Berk, E. Vieta, M. Maj, Y. A. de Vries, et al. 2020. "Mental, Behavioral and Neurodevelopmental Disorders in the ICD-11: An International Perspective on Key Changes and Controversies." *BMC Medicine [Electronic Resource]* 18(1): 21. https://doi.org/10.1186/s12916-020-1495-2.
20. Faraone, S. V. and H. Larsson. 2019. "Genetics of Attention Deficit Hyperactivity Disorder." *Molecular Psychiatry* 24(4): 562–75. https://doi.org/10.1038/s41380-018-0070-0.
21. Thapar, A., and M. Rutter. 2021. "Genetic Advances in Autism." *Journal of Autism and Developmental Disorders* 51(12): 4321–32. https://doi.org/10.1007/s10803-020-04685-z.
22. Forrest, J. A., J. A. Clements, and L. F. Prescott. 1982. "Clinical Pharmacokinetics of Paracetamol." *Clinical Pharmacokinetics* 7(2): 93–107. https://doi.org/10.2165/00003088-198207020-00001.
23. Cetaruk, E. W., R. C. Dart, K. M. Hurlbut, R. S. Horowitz, and R. Shih. 1997. "Tylenol Extended Relief Overdose." *Annals of Emergency Medicine* 30(1): 104–8. https://doi.org/10.1016/s0196-0644(97)70120-3.

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24. Li, Y., X. Hong, A. Chandran, C. A. Keet, C. B. Clish, L. Liang, L. P. Jacobson, X. Wang, and C. Ladd-Acosta. 2024. "Associations Between Cord Blood Acetaminophen Biomarkers and Childhood Asthma With and Without Allergic Comorbidities." *Annals of Allergy, Asthma & Immunology* 132(6): 705–12.e5. https://doi.org/10.1016/j.anai.2024.03.001.
25. Ji, Y., R. E. Azuine, Y. Zhang, W. Hou, X. Hong, G. Wang, A. Riley, C. Pearson, B. Zuckerman, and X. Wang. 2020. "Association of Cord Plasma Biomarkers of In Utero Acetaminophen Exposure With Risk of Attention-Deficit/Hyperactivity Disorder and Autism Spectrum Disorder in Childhood." *JAMA Psychiatry* 77(2): 180–9. https://doi.org/10.1001/jamapsychiatry.2019.3259.
26. Yang, G., X. Deng, J. Xiao, P. Huang, K. Zhang, and Y. Li. 2021. "Maternal Fever During Preconception and Conception Is Associated With Congenital Heart Diseases in Offspring: An Updated Meta-Analysis of Observational Studies." *Medicine* 100(9): e24899. https://doi.org/10.1097/md.0000000000024899.
27. Mohan Dass, N. L., L. D. Botto, S. C. Tinker, M. A. Canfield, R. H. Finnell, M. S. Gallaway, S. S. Hashmi, et al. 2022. "Associations between Maternal Reports of Periconceptional Fever from Miscellaneous Causes and Structural Birth Defects." *Birth Defects Research* 114(15): 885–94. https://doi.org/10.1002/bdr2.2068.
28. Dreier, J. W., A. M. Andersen, and G. Berg-Beckhoff. 2014. "Systematic Review and Meta-Analyses: Fever in Pregnancy and Health Impacts in the Offspring." *Pediatrics* 133(3): e674–e88. https://doi.org/10.1542/peds.2013-3205.
29. Centers for Disease Control and Prevention. HEAR HER Campaign: Urgent Maternal Warning Signs and Symptoms. Updated May 15, 2024. Accessed April 28, 2026. https://www.cdc.gov/hearher/maternal-warning-signs/index.html.
30. Bruehl, S., O. Y. Chung, J. N. Jirjis, and S. Biridepalli. 2005. "Prevalence of Clinical Hypertension in Patients with Chronic Pain Compared to Nonpain General Medical Patients." *Clinical Journal of Pain* 21(2): 147–53. https://doi.org/10.1097/00002508-200503000-00006.
31. Dørheim, S. K., B. Bjorvatn, and M. Eberhard-Gran. 2012. "Insomnia and Depressive Symptoms in Late Pregnancy: A Population-Based Study." *Behavioral Sleep Medicine* 10(3): 152–66. https://doi.org/10.1080/15402002.2012.660588.
32. Nunge, R. A., A. M. Kendle, and S. Alwan. 2025. "Pain Medication Use During Pregnancy." *Seminars in Perinatology* 49(4): 152074. https://doi.org/10.1016/j.semperi.2025.152074.
33. Cornet, M.-C., M. W. Kuzniewicz, A. W. Scheffler, C. Garabedian, S. L. Gaw, and Y. W. Wu. 2025. "Maternal Fever During Labor and the Risk of Neonatal Encephalopathy: Duration and Magnitude of Hyperthermia." *American Journal of Obstetrics and Gynecology* 233(6): 651.e1–12. https://doi.org/10.1016/j.ajog.2025.07.046.

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