The manufactured science that claims Tylenol causes autism

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The September 22, 2025, White House press conference on Tylenol and autism marked a turning point in the politicization of science by the Trump administration and Health and Human Services Secretary Robert F. Kennedy Jr. Trump and Kennedy are seeking to impose right-wing ideology as medical fact with far-reaching implications.

Flanked by Kennedy, FDA Commissioner Marty Makary, and other appointees, Trump presided over a stage-managed performance that obliterated the line between governance and propaganda. What should have been a discussion of evidence became a spectacle of political theater, complete with orchestrated outrage and pre-scripted affirmation from his inner circle. During the briefing, Trump, seemingly overdosing on his own scare tactics, repeatedly warned pregnant women, "Don't take Tylenol! There's no downside. Don't take it! You'll be uncomfortable. It won't be as easy, maybe. But don't take it!"

Makary and the Food and Drug Administration (FDA) made good on the president's promise by immediately issuing guidance advising physicians to minimize acetaminophen use for routine low-grade fevers and initiate a process to update product warning labels. This marked an unprecedented political intervention in medical regulation, one that leveraged the machinery of federal public health agencies to advance a claim that lacked broad scientific consensus. Kennedy, long known for promoting baseless anti-vaccine theories, positioned the move as part of a broader crusade against what he called the "hidden environmental causes" of neurodevelopmental disorders.

The White House's rationale for this sweeping policy action rested almost entirely on a single scientific publication, the Mount Sinai-led systematic review published in *BMC Environmental Health* in August 2025. Widely known as the Prada and Baccarelli study, it synthesized findings from 46 observational studies examining prenatal acetaminophen exposure and child neurodevelopmental outcomes. Although the authors explicitly cautioned that their results suggested only the "possibility of a causal relationship" and called for further research, the distinction between *association* and *causation* in scientific research was quickly discarded.

According to the *Harvard Crimson*, in conversations with administration officials, co-author Andrea Baccarelli may have overstated the strength of his findings, employing an assertion of causality that went beyond what his paper could legitimately support. During the White House press conference, FDA Commissioner Marty Makary amplified that exaggeration, quoting Baccarelli as saying there "is a causal relationship between prenatal acetaminophen use and neurodevelopmental disorders of ADHD and autism," a distortion that directly contradicts both the study's published language and the authors' formal statements to the press.

The press briefing marked a decisive shift, transforming what should have remained a cautious discussion of an association between Tylenol and autism, speculative at best and unsupported by better-designed studies, into a state-sanctioned claim of causation. The event amounted to an orchestrated scientific hoax, in which the president and his partners in crime seized and repurposed academic findings of questionable quality to legitimize a predetermined political agenda. The White House further concealed a crucial fact: Baccarelli had served as a paid expert witness in the 2023 federal Tylenol multidistrict litigation, where his testimony—based on the very same studies later used in the August 2025 paper—was excluded by Judge Denise Cote for being "cherry-picked and misrepresented" and for presenting a "result-driven analysis."

On the issue of unreliability of methodology and bias, Judge Cote repeatedly noted, "Cherry-picking is a form of result-driven analysis which undermines principles of the scientific method by applying methodologies (valid or otherwise) in an unreliable fashion… An expert must not cherry-pick from the scientific landscape and present the Court with what he believes the final picture looks like."

A major flaw cited by Judge Cote in excluding Baccarelli's testimony was his failure to properly account for confounding factors, particularly genetic influences, which the court found undermined his claim of a causal link between acetaminophen use and neurodevelopmental disorders. The court observed that "Dr. Baccarelli downplays those studies that undercut his causation thesis and emphasizes those that align with *his* thesis." It further stated that "his failure to confront carefully and fairly the profoundly important issue of confounding by genetics renders his opinion on causation inadmissible."

Addressing the highest-quality studies, the court decision noted that "if that evidence of a modest association is eliminated entirely by a sibling control study, that result should not be ignored." Specifically, Judge Cote wrote that "Dr. Baccarelli failed to sufficiently explain the appropriateness of conducting a single Bradford Hill analysis for NDDs, which included ASD [Autism Spectrum Disorder] and ADHD [Attention-Deficit/Hyperactivity Disorder]; selectively analyzed the consistency of the literature and the issue of genetic confounding; repeatedly pressed conclusions that study authors were not willing to make; and disregarded studies that did not support his opinion due to limitations he did not view as disqualifying in studies that did support his opinion." The court concluded that Baccarelli's opinion regarding consistency "does not adequately address the many conflicting study results."

[*Note: The Bradford Hill analysis is a framework developed by British epidemiologist Sir Austin Bradford Hill in 1965 to help determine whether a statistical association is likely to reflect a causal relationship. It outlines nine guiding considerations that include the strength, consistency, temporality, and biological plausibility of studies that together help researchers judge whether correlation might indicate cause and effect. The method was first applied in establishing the causal link between smoking and lung cancer and remains a foundational, though interpretive, tool in modern epidemiology.*]

Irrespective of the court's decisive critique of Baccarelli's methods and biases as an expert witness, in the weeks leading up to the recent White House press conference, the *Crimson* reported that Baccarelli had held

phone discussions with Kennedy and NIH Director Jay Bhattacharya regarding his new review, meetings that set the stage for its political appropriation. Since the event, he has declined public interviews, now stating only that his research identified an *association* and not a *causal* link, the distinction that lies at the core of the present scientific inquiry on Tylenol and autism, one which the White House has deliberately obfuscated.

The administration's claims were swiftly condemned by experts from leading universities. Samuel S. Wang, a professor of neuroscience at Princeton University, called the assertion that acetaminophen causes autism "a massive overstatement and possibly completely untrue," while Dennis P. Wall, a professor of pediatrics and biomedical data science at Stanford University, emphasized that "there needs to be much more work done … to identify causal mechanisms," and that this "simply hasn't been done." Catherine E. Lord, a professor of psychiatry and education at UCLA, likewise cautioned that "to take it the next step and say this is causal, is really irresponsible."

The American College of Obstetricians and Gynecologists (ACOG), which represents more than 60,000 obstetrician-gynecologists, reaffirmed in their statement that "there is no clear evidence that proves a direct relationship between prudent acetaminophen use during any trimester and fetal developmental issues." The organization explicitly rejected the administration's claims, emphasizing that acetaminophen remains "the safest known option for fever and pain management during pregnancy when used as directed."

Independent epidemiologists and science communicators, including Ellie Murray, ScD, an assistant professor of epidemiology at Boston University's School of Public Health and author of the *E is for Epi* newsletter, and Andrea Love, PhD, an immunologist and science educator, have publicly dismantled the study's claims.

Writing within days of the White House announcement, Murray summarized the issue bluntly in her article titled, "The best evidence that Tylenol causes autism isn't great." She and others pointed out that the review did not generate new data. Instead, it merely reanalyzed previous observational studies (used in the 2023 federal Tylenol multidistrict litigation), many of which suffered from bias and confounding that made causal inference impossible.

They also noted that the Mount Sinai team used the Navigation Guide framework—a tool originally developed for evaluating environmental toxicants, not pharmaceutical safety—to rank and synthesize 46 studies examining acetaminophen use during pregnancy. Murray and Love both noted that this choice was questionable, while pointing out that none of the review's authors were specialists in pharmacoepidemiology or perinatal medicine, and the framework itself was poorly suited to disentangling complex exposure patterns or genetic factors. As Love explained in her analysis, the study "collapsed nuance into a single grade of evidence," relying on subjective judgments about "risk of bias" and "expert opinion" that were neither transparent nor reproducible.

One of the review's most striking flaws was how it treated sibling-controlled studies that found no link between Tylenol and autism. The largest and most rigorous of these, the 2024 Swedish nationwide cohort of 2.48 million children, reported no association between prenatal acetaminophen use and autism, ADHD, or intellectual disability once sibling comparisons were applied. Yet the Mount Sinai team labeled this study as having "high risk of bias" and minimized its weight in the overall analysis. As Ellie Murray noted, this effectively punished the Swedish study for not showing a positive result: "The review treats a large, null study as weak evidence rather than strong evidence of no effect."

Another major flaw was the review's "transdiagnostic" approach—lumping all neurodevelopmental disorders into a single outcome category. This broad grouping blurred key distinctions and concealed the fact that only a few studies examined autism diagnoses directly. As Ellie Murray noted, combining such varied conditions "is like grouping migraines, seizures, and insomnia under one heading and declaring you've found a new disease." The result was an analysis that appeared to confirm the authors' preexisting belief that acetaminophen was harmful, shifting the burden of proof onto anyone who questioned that conclusion.

In the meantime, the strongest, most methodologically sound research points away from any causal connection. Revisiting the Swedish sibling-control study, the researchers analyzed data from nearly 2.5 million children, comparing outcomes between siblings—one exposed to acetaminophen during pregnancy and another not. By holding genetics and shared family environment constant, the study effectively eliminated many of the biases that had produced weak associations in simpler observational research. The results clearly revealed that the small increases in autism and ADHD risk seen in conventional models—about 5 to 7 percent—disappeared entirely once sibling comparisons were applied. The authors concluded that the earlier associations were likely due to familial confounding, not acetaminophen exposure itself.

A year later, the Japanese nationwide birth cohort study led by Yusuke Okubo reached the same conclusion. Using data from more than 217,000 mother-child pairs, the researchers applied propensity score matching, sibling comparisons, and probabilistic bias analysis to identify hidden confounders. As in the Swedish study, small risk increases for ADHD and autism seen in basic models disappeared once genetic and environmental factors were controlled for. The authors cautioned that "unmeasured confounding, misclassification, and other biases may partially explain these associations," effectively nullifying any causal link.

Taken together, these two large-scale studies, conducted independently on different continents, using distinct healthcare systems and genetic populations, dismantle the narrative that the Trump-Kennedy administration elevated to national policy. What appeared, in weaker analyses, as a potential signal has now been shown to be statistical noise.

Trump and Kennedy's declaration that Tylenol causes autism stands in direct opposition to the evidence base. No scientifically validated study has established a causal relationship between prenatal acetaminophen exposure and neurodevelopmental disorders. Their claim relies on a handful of observational studies showing modest statistical associations, none of which withstand more rigorous analyses.

This distinction between *association* and *causation* lies at the core of scientific reasoning. An association simply means two factors occur together—for example, women taking more pain relievers during pregnancy may also experience higher rates of fever, infection, or inflammation, each of which can influence fetal development. Causation, by contrast, implies a direct, mechanistic link where changing one factor changes the outcome. Establishing causation requires evidence that rules out alternative explanations, confirms time sequence, and demonstrates a plausible biological mechanism. Absent these criteria, asserting causation is speculative at best and deceptive at worst. In the Tylenol debate, no experimental or longitudinal evidence demonstrates a causal pathway between acetaminophen and autism; only weak correlations that vanish when examined under more rigorous designs.

The history of smoking and lung cancer offers a vivid example of how science progresses from correlation to causation. When early studies in the 1950s observed higher lung cancer rates among smokers, tobacco companies dismissed the findings as mere association. Over the next decade, researchers documented dose-response relationships, consistent results across populations, and biological mechanisms linking tobacco smoke to carcinogenic mutations. By the 1964 Surgeon General's Report, the evidence had crossed the threshold from association to causation—prompting sweeping public health reforms. That transformation was not political; it was empirical, grounded in converging data and the deliberate pace of scientific validation.

Kennedy and Trump's declaration that Tylenol causes autism has

essentially reversed the logic of the scientific process. Instead of letting evidence build toward causation, they begin with a predetermined causal claim and retrofitted selected data to support their preconceived illusions. Such inversions represent the process by which science becomes politicized, where consensus and objective scientific truth are disregarded and treated as conspiracy and heresy. Such distortion not only erodes public confidence in science but also endangers lives by replacing evidence-based caution with ideological certainty. Science, by its nature, draws a firm line between what can be observed and what can be known. Crossing that line for political gain transforms the scientific method into spectacle and leaves public health and all of society as collateral damage.