Autism and the crisis of science: A conversation with Dr. Alycia Halladay

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In the aftermath of a White House press conference that reignited the most reactionary narratives surrounding autism, the crisis of public health in the United States has reached a new and dangerous phase. Standing beside Donald Trump, Health and Human Services Secretary Robert F. Kennedy Jr. declared that acetaminophen use during pregnancy contributes to autism and that a folate-based therapy might mitigate its symptoms. No new data was presented. Instead, the announcement relied on tenuous associations and long-discredited claims—echoing the decades-old "vaccines cause autism" myth that has served as a political rallying point for the far right. Scientists and watchdog organizations immediately condemned the spectacle for its distortion of evidence and its brazen manipulation of parental fear.

These events are not isolated missteps but part of a systematic dismantling of the scientific and institutional infrastructure that underpins public health. In 2025, Kennedy summarily dismissed all seventeen members of the Advisory Committee on Immunization Practices (ACIP), replacing them with appointees loyal to his anti-vaccine agenda. A month later, CDC Director Susan Monarez was forced out, triggering a wave of resignations among career scientists who warned that the agency was being weaponized to legitimize pseudoscience. In this climate of intimidation and political intrusion, research itself becomes suspect; expertise, long cultivated through collective inquiry, is recast as an elite conspiracy.

It is against this backdrop that the following conversation with Dr. Alycia Halladay, Chief Science Officer of the Autism Science Foundation (ASF), took place. Halladay, who has dedicated her career to advancing rigorous, evidence-based autism research, now finds herself working within an atmosphere of suspicion deliberately cultivated by those in power. Her insistence on scientific integrity—and on communicating that science clearly to the public—stands in sharp contrast to the reactionary forces that have sought to turn autism into a political weapon.

Dr. Halladay brings both technical expertise and moral clarity to this discussion. At ASF, she oversees grants and research programs that examine gene-environment interactions in autism, improve early detection and diagnosis, and support brain donation and open data initiatives. Previously at Autism Speaks and the National Alliance for Autism Research, she has been instrumental in establishing funding streams for studies that integrate genetics, neurodevelopment, and environmental exposure research. She holds a Ph.D. in biopsychology and a postdoctoral fellowship in pharmacology and toxicology from Rutgers University and has authored more than two dozen peer-reviewed papers. Her service on editorial boards such as Neurotoxicology *and* Frontiers in Pediatrics*, and her membership on the U.S. Interagency Autism Coordinating Committee, underscore her central role in the contemporary autism research landscape.*

In this interview, Dr. Halladay discusses how science has been hijacked by political interests, why simplistic "one-cause" theories of autism persist, and how misinformation spreads through a media ecosystem driven by profit and ideology. She speaks candidly about the social roots of distrust in science, the enduring power of collective public health measures like vaccination, and the human realities of autism research—parents seeking answers, scientists working under pressure, and the fragile boundary between knowledge and manipulation.

Benjamin Mateus (BM): Dr. Halladay, thank you for taking the time to speak with me today. It's a pleasure to have you join us.

Alycia Halladay (AH): Thank you, Benjamin. Please, call me Alycia. I'm happy to be here.

BM: I appreciate Professor Dorit Reiss connecting us. We've spoken with her about the legal and policy aspects of vaccine science, and I wanted to follow up with you to explore the scientific dimensions—especially the current controversy over Tylenol, autism, and the broader public health issues emerging under the Trump–Kennedy administration.

To begin, could you please introduce yourself and describe your role as Chief Science Officer at the Autism Science Foundation, and perhaps outline the foundation's commitment to evidence-based science regarding autism's causes?

AH: My name is Alycia Halladay, and I'm the Chief Science Officer at the Autism Science Foundation. I oversee our scientific initiatives and research grants, including our Early Career Investigator awards, targeted funding programs, including those that support collaborations studying early signs and features of autism and genetic causes of autism. We also fund projects that examine specific scientific questions—for example, the Autism Sisters Project, which explores why females are less likely to be diagnosed with autism compared to males.

BM: Following the White House's recent statements suggesting a link between acetaminophen—or Tylenol—use during pregnancy and autism, the Autism Science Foundation cautioned that these claims rest on limited, conflicting, and inconsistent evidence. Could you explain why the administration's claims are scientifically unsupported or potentially misleading?

AH: The idea of a link between acetaminophen use during pregnancy and autism has been circulating for several years. A few small studies have reported an association—not causation—suggesting that as reported Tylenol use increased, so did the likelihood of a child being diagnosed with autism.

The problem is that many of those early studies, such as some published around 2020, had very small sample sizes and couldn't adequately account for confounding variables. For example, they didn't always examine why a pregnant woman was taking acetaminophen, how frequently, or under what medical circumstances. They also rarely controlled for underlying genetic factors or family history of autism. With such small samples, it's impossible to draw reliable conclusions.

One often-cited study didn't even measure acetaminophen use during pregnancy—it measured acetaminophen in umbilical cord blood at birth. But because the drug's half-life is only a few hours, that result likely

reflects use immediately before or during labor, not throughout pregnancy. Yet those limited findings are what the administration has cited as evidence that Tylenol causes autism.

More recent large-scale studies contradict that claim. For example, a Swedish study analyzed 2.4 million pregnancies—about 35,000 of which involved children later diagnosed with autism—and found no association between acetaminophen use during pregnancy and autism. A separate Japanese study of roughly 220,000 participants also found no link.

These larger studies were able to compare siblings within the same family—one exposed to more acetaminophen, another less—while controlling for genetic background and other shared environmental factors. They also tracked the reason for taking acetaminophen and verified reported use at medical visits. That level of rigor provides a much more accurate picture.

So, despite repeated claims about "40 studies" showing a connection, that simply isn't true. It's more like a game of telephone—where information becomes distorted as it's passed along through media and political commentary. Figures get repeated, misquoted, and amplified until the public hears something entirely different from what the original studies said.

When you examine the research, about half of the small studies show some weak association, and half do not. Those that find one typically have methodological limitations, while the large, well-controlled studies find no evidence of a causal relationship.

Smaller studies still have value because they raise questions worth exploring further. But they shouldn't be treated as conclusive proof. Unfortunately, in this case, those preliminary, underpowered studies were promoted as definitive answers. That's not how public health decisions should be made.

The review by Dr. Andrea Baccarelli

BM: The administration—led by Secretary Kennedy and with support from NIH Director Bhattacharya—heavily promoted a review associated with Dr. Andrea Baccarelli of Harvard. Baccarelli was also a prominent expert witness in the 2023 Tylenol litigation, although a judge excluded his testimony for being methodologically selective.

In their Mt. Sinai study, Baccarelli and colleagues had downplayed the large Swedish sibling-control study, dismissing it as "high bias." Could you walk us through the strengths and weaknesses of that Baccarelli-led review? And to what extent do you see political or agenda-driven motives shaping this particular study and the administration's emphasis on it rather than on the larger, more robust studies?

AH: Yes. The paper you're referring to—the one often cited by the administration—was the systematic review led by Dr. Andrea Baccarelli, who is now the Dean of the Harvard T.H. Chan School of Public Health.

A systematic review means that investigators start with a specific hypothesis—in this case, whether acetaminophen use during pregnancy is associated with autism—and then collect and evaluate all available studies that appear to address that question. The Baccarelli group did exactly that.

They gathered existing studies and summarized what each reported. Some were small and showed a weak association, while others found none. But the review's strength depends entirely on the quality of the studies included, and that's where the main concern lies.

For instance, one of the studies they heavily weighted measured acetaminophen levels in umbilical cord blood, not during pregnancy. Even the study's own authors clearly acknowledged that limitation in their paper, writing that cord blood reflects exposure only in the final hours before birth, not across the full gestation. They also noted that their analysis didn't account for a family history of autism or other genetic and environmental factors. In short, the authors themselves cautioned against overinterpreting their results.

The systematic review did attempt to evaluate "risk of bias" across studies, ranking each according to sample size, data completeness, and potential conflicts of interest. But it appears they applied that rating unevenly—being somewhat lenient with small, limited studies, while applying unusually harsh criteria to the large Swedish population study that found no association. I can't speak to their internal reasoning, but the risk of bias assessment has been questioned by other scientists.

The authors were transparent about their process, and to their credit, they published their bias ratings in full. They also excluded the large Japanese sibling-control study simply because it was published after their review period, which ended in 2024. That's understandable—you either publish or risk waiting indefinitely for new data.

Still, the end result was a review that gave disproportionate weight to weaker studies and discounted the strongest evidence available. Some of the included papers didn't even address autism as a diagnosis, but rather measured isolated behavioral traits. So, it's fair to say that, while the review was conducted systematically, its conclusions reflect the limitations of the underlying data.

As the saying goes, "perspectives may vary." But from a scientific standpoint, those methodological inconsistencies are significant—and they certainly don't justify the administration's claim of a proven causal link.

BM: I think it's also important to emphasize that one of the major strengths of the Swedish study was its sibling-comparison design. It wasn't just a broad population analysis — it compared outcomes within the same families, which allowed researchers to control for genetic and environmental factors that are otherwise very difficult to separate.

And beyond that, the study drew on Sweden's national health registries, which are exceptionally comprehensive and detailed. That level of population data is rare and gives the findings real weight. I don't mean to suggest there was anything devious in how the Harvard group handled their review, but it does seem that by downplaying such a robust dataset, the overall picture can become skewed. Especially since most readers aren't going to examine these studies line by line, which makes it easy for misleading conclusions to take hold.

Would you like to comment on that aspect—the sibling-control design and the significance of having such rich, reliable registry data in drawing evidence-based conclusions?

AH: That's exactly the case with both the Swedish and the Japanese studies—the latter, unfortunately, wasn't included in the Baccarelli review, though it really should have been part of the discussion. I sometimes feel like only a handful of us have even mentioned that Japanese study, because the public focus has been almost entirely on those smaller, less rigorous papers.

What makes the Swedish study so important is its sibling-comparison design. Instead of comparing one family that used acetaminophen with another that didn't, it compared pregnancies within the same family. So, for instance, one child was later diagnosed with autism while a sibling was not.

That approach allows researchers to control for shared genetic background and many environmental factors that are nearly impossible to separate across unrelated families.

You can only do this kind of analysis when you have an enormous sample, which is why the Swedish registry is so valuable. Sweden tracks pregnancies, maternal medication use, and child health outcomes throughout development, sometimes even into adulthood. That makes it possible to study subtle relationships between exposures and diagnoses on a scale that smaller studies simply can't match.

By examining siblings within the same household, researchers can rule out many confounding factors—especially genetics and, to some extent,

socioeconomic conditions and access to medical care, which are likely consistent within families. That's a major methodological strength.

The Japanese study followed a similar logic, although on a smaller scale, and it also found no association between acetaminophen exposure and autism. Together, those two studies provide the most compelling evidence to date.

Now, one limitation across all these analyses is that acetaminophen is an over-the-counter medication. Unlike a prescription drug, it's difficult to track exact doses and frequency. Researchers often rely on self-report—mothers estimating how often they took it—which introduces some uncertainty. Even so, because acetaminophen use during pregnancy is relatively common—somewhere between 7.5 and 50 percent of pregnant women report taking it—it's still one of the more accessible exposures to study.

The key takeaway is that these registry-based sibling studies, with their massive sample sizes and careful controls, carry far more weight than small, hypothesis-generating studies. They're not perfect, but they're far closer to what we should rely on for public health guidance.

BM: And I think both the Japanese and Swedish studies initially showed a slight uptick in association. But once they applied the full sibling—or co-sibling—comparison methodology, that association disappeared, correct?

AH: Absolutely, yes. When they incorporated the sibling analyses, any initial signal of association vanished. And those studies didn't stop there—they also accounted for a range of additional factors: other medications taken during pregnancy, underlying conditions such as arthritis or chronic headaches, maternal body mass index, smoking history, psychiatric disorders, and prescription drug use. In other words, they were able to analyze not just whether someone took Tylenol, but the broader medical and behavioral context around that pregnancy.

The recommendations of obstetricians and gynecologists

BM: Along with this line of inquiry, I have one last question. Both the American College of Obstetricians and Gynecologists (ACOG) and the Society for Maternal-Fetal Medicine continue to recommend acetaminophen as safe when used appropriately during pregnancy. Given the current climate, what are the public health risks if pregnant individuals begin avoiding necessary treatment out of fear? And how should families approach acetaminophen use responsibly amid this confusion?

AH: Thank you for that question. I think this entire situation has been incredibly confusing for families—and understandably so. On September 22, there was a nationally televised press conference where President Trump said, essentially, "Don't do it. Don't take it." He even added, "If you have to take it, it'll happen," which was never explained but was widely interpreted as a warning that Tylenol causes autism.

Then, standing beside him, the FDA Commissioner stated that women should exercise caution and use the lowest possible dose of acetaminophen during pregnancy. That advice, however, was not new—it's consistent with what ACOG and most obstetricians have said for years: if you're experiencing pain or fever, talk to your doctor, describe what's going on, and use the minimum effective dose when advised.

One critical point that often gets lost in public discussion is that acetaminophen reduces fever—and prolonged fever itself is associated with an increased risk of autism. So, in some ways, this is a "damned if you do, damned if you don't" situation if people misunderstand the context. Avoiding fever treatment entirely could raise the risk that the administration claims to be trying to prevent.

That's why it's so important for pregnant women to consult their doctors rather than react to headlines. Your physician knows your medical history, understands your risks, and can guide you toward the safest course of action.

The official FDA communication to providers was far more measured than the messaging that dominated the news cycle. The letter reiterated existing guidance: use the lowest effective dose, for the shortest necessary time, and only as needed. But the public heard something entirely different from the press conference, where the president claimed that "acetaminophen causes autism." That disconnect between science-based guidance and flashy sound bites is what families are now struggling with.

BM: As a brief aside, I wanted to ask about COVID-19. Given the scale of the pandemic—the number of infections, and the high, sometimes prolonged fevers associated with it—have we seen any evidence of an increase in autism diagnoses among children born during or after the pandemic? In other words, is there any indication that COVID-related maternal fever has translated into higher autism rates?

AH: That's a really good question. There was a recent study that used medical records collected early in the pandemic—back when people were getting PCR tests through pharmacies or clinics and those results were automatically linked to their health records. Because those records included information about pregnancies and newborn screenings, researchers were later able to track developmental outcomes, including autism, in the children born during that time.

What they found was no difference in autism screening outcomes between children whose mothers tested positive for COVID during pregnancy and those who didn't. That doesn't mean there's absolutely no connection, but so far, the data haven't shown any clear association.

Now, there are some limitations to keep in mind. Many pregnant women didn't get tested at all, or they used at-home tests that weren't recorded in medical databases. Others may have had COVID symptoms or fevers but never sought medical attention—especially early in the pandemic, when people were being told to isolate and avoid healthcare settings unless absolutely necessary.

So, the picture is incomplete. We do know from previous research that maternal fever—not specifically COVID-related—can be associated with a slightly higher likelihood of neurodevelopmental differences, including autism. But for COVID infection itself, the evidence just isn't there yet. The key next step is to study COVID with fever and inflammatory response, not just positive test results. That's where the more meaningful biological questions lie.

The wide range of autism

BM: Secretary Kennedy often describes autism as an "epidemic" or even a "preventable disease." From your perspective, what are the dangers of this kind of rhetoric—for autistic individuals and their families, and for the integrity of autism research?

AH: Characterizing autism as a "disease" is problematic on several levels. Autism is a neurodevelopmental condition—not a communicable, fatal illness. When people call it a disease or an epidemic, it implies there's something to be eradicated, which undermines the lived realities of autistic individuals and, more importantly, is a mischaracterization of autism to begin with. Autism involves complex genetic and environmental contributions and manifests very differently from person to person. Roughly 20 percent of cases are associated with rare genetic syndromes; the rest reflect a combination of factors that are still being studied.

Secretary Kennedy appears to focus on "profound autism"—individuals who are minimally verbal, have significant intellectual disability, and need intensive support. But research and media coverage often treat the entire spectrum as one homogeneous group. That's misleading. On one end of

the spectrum are people who require round?the?clock supervision for safety; on the other are individuals with university degrees, jobs, friendships, and largely independent lives, though they may still have specific challenges.

Because autism encompasses such a wide range of experiences and needs, many advocates now talk about "autisms," to capture the diverse genetic backgrounds, causes, and trajectories. When policymakers frame autism as a preventable epidemic, it not only stigmatizes autistic people but also threatens to divert research and resources away from understanding its complexity and improving quality of life across the spectrum.

BM: Understood. Stepping back, what is the current scientific consensus on the main factors that contribute to autism? How do genetics and environment interact, rather than it being one or the other? And at a high level, which biological pathways are most implicated?

AH: Genetics plays a major role. We now know of well over 200 genes where variants or mutations are linked to neurodevelopmental conditions—and, for a substantial subset, specifically to autism. Autism also runs in families. Siblings have a much higher likelihood of diagnosis than unrelated individuals, and different combinations of rare and common genetic variants can increase probability.

That said, environment also contributes, but it needs to be defined broadly. It includes social and contextual factors (neighborhood, education, stress, access to care), chemical exposures (air quality, pollutants), and medical factors (infections, medications, maternal health). Think of hundreds—if not thousands—of environmental influences interacting with both known and as-yet-unknown genes. That gene-environment interplay is likely central to why autism develops in some individuals.

For some people, the genetic component is dominant; for others, environmental influences and timing may matter more. And importantly, social determinants—like socioeconomic status and healthcare access—shape who gets evaluated and diagnosed in the first place. So, it's outdated to seek a single culprit (a medication or one exposure). The more accurate picture is multiple biological pathways—including synaptic development, chromatin and transcriptional regulation, and immune–inflammatory signaling—operating within diverse life contexts.

BM: These genes you're referencing—the more than 200—these are primarily involved in how neural connections form in the brain, correct?

AH: Absolutely. Many of these genes are active very early in development, even when a fetus is just beginning to form the basic structures of the brain. They guide how neurons migrate to their proper locations and establish connections between different regions.

Think of it this way: the fetal brain starts with thousands of neurons scattered throughout, and these genes—through the proteins and signaling molecules they encode—help determine where those neurons should go and how they should connect. Some genes influence which cells become excitatory neurons, which activate other cells, while others help form inhibitory neurons, which act more like stoplights, regulating and balancing brain activity.

If everything were excitatory all the time, the brain would be overactive and chaotic. Inhibitory neurons ensure efficiency and proper signaling, keeping neural communication precise. So, these genes essentially converge on key biological processes: how brain cells form, differentiate, connect, and communicate. They shape both the structure and the functional wiring of the developing brain.

BM: Something you said earlier made me think about the social context you come from, and how certain narratives take hold. During that White House press conference, Trump asked Kennedy whether the Amish have autism, and Kennedy replied that they don't. That struck me because there's well-documented research showing the Amish do have autism—and that they also vaccinate. It felt like a deliberate distortion of the facts.

AH: That's right. The claim that the Amish don't have autism simply isn't true. There are published studies documenting autism diagnoses within Amish communities, and research also shows that many Amish families choose to vaccinate. What hasn't been done is a specific, population-wide study linking vaccination rates to autism among the Amish. But to suggest they're untouched by either vaccines or autism is just factually wrong.

The rising prevalence of autism

BM: Can you speak about the rising prevalence of autism—from one in 150 in 2000 to roughly one in 36 today? How do experts explain this increase, and why do most researchers reject the idea that it represents an epidemic caused by environmental toxins?

AH: The rise in autism prevalence is undeniable, but the crucial question is why it's happening. The evidence tells us that most of the increase reflects changes in how autism is identified, diagnosed, and reported—not a sudden surge in cases caused by an external agent. Over the past twenty years, several important developments have taken place. First, clinicians and educators have become much more aware of what autism looks like, including milder and less obvious forms that were once overlooked. Second, diagnostic criteria have broadened multiple times, so people who might not have met the definition decades ago are now being accurately recognized. Third, improved insurance coverage and state mandates have made autism evaluations and treatments far more accessible, meaning more families can seek and receive a diagnosis.

We've also seen that the biggest increases have occurred among those with what we call non-profound or less severe forms of autism, while prevalence among people with profound autism has also grown but at a slower pace. These shifts strongly suggest that greater awareness, changing criteria, and improved access to care are major drivers of the rising numbers.

This isn't an either-or explanation. An unknown portion of the increase may reflect real changes in incidence, influenced by environmental or biological factors—though "environmental" in this context doesn't necessarily mean chemical toxins. It can include many aspects of the world we live in, from prenatal and perinatal factors to social and medical influences. But to date, no credible evidence supports the claim that autism is an epidemic triggered by an environmental toxin. The far stronger evidence points to multiple factors.

BM: One of the things I came across when looking into this was what you just mentioned — that shifts in diagnosis mean some conditions have gone down as autism has gone up. In other words, it's not that the total number of cases suddenly increased, but that people previously diagnosed with one disorder are now being identified more accurately as autistic.

AH: Yes, that's right. We saw that most clearly with intellectual disability, previously referred to as mental retardation. For a period, as autism diagnoses rose, the number of intellectual disability diagnoses declined. That substitution explained part of the increase early on.

But more recently, the rise in autism prevalence can't be explained by that alone. The diagnostic criteria have changed multiple times; insurance mandates have expanded coverage; and families now have stronger incentives to seek an evaluation, since services are often tied to an official diagnosis. Increased public awareness and a deeper understanding of autism have also played big roles.

So, while diagnostic substitution helped explain the early trends a couple of decades ago, the ongoing increase today is driven more by these broader systemic and social changes. That said, there's still room to

consider how environmental factors—broadly defined—might also influence prevalence over time.

BM: And one last follow-up before we move on. Do we know why autism is more common in males than females?

AH: Autism is diagnosed about four times more often in males than in females—or, more precisely, in people assigned male at birth. There are several possible reasons for this. Some studies suggest a biological or genetic protective factor that makes girls less likely to cross the diagnostic threshold. Others point to differences in how autism manifests.

Girls and women may have more subtle social communication differences or different kinds of restricted and repetitive interests, which can make their symptoms less visible or less likely to meet current diagnostic criteria. Thankfully, there's been a surge of research and clinical interest in better identifying and supporting autistic females, who have historically been underserved and underdiagnosed.

The "vaccines cause autism" lie

BM: I'm also curious from a historical perspective about how this false link between vaccines and autism took hold. How did that claim begin, and why has it persisted for so long despite overwhelming scientific evidence to the contrary? Anti-vaccine movements have existed since vaccines themselves, as Professor Reiss pointed out, but what explains this particular connection between autism and vaccines?

AH: Anti-vaccine sentiment has existed for as long as vaccines have, especially whenever mandates were introduced. It intensified during COVID, but the specific link between autism and vaccines really began in the late 1990s.

In 1998, a British physician named Andrew Wakefield, and several colleagues published a paper in *The Lancet* claiming that the MMR vaccine—measles, mumps, and rubella—was associated with autism and gastrointestinal symptoms in twelve children. The study was small, poorly designed, and later found to involve serious ethical violations—Wakefield had taken blood from children at a birthday party without proper consent and failed to disclose financial conflicts of interest. The paper was eventually retracted in 2010, and he later lost his medical license.

But by that time, the damage was done. The idea that a common childhood vaccine could cause autism triggered understandable fear among parents, especially because symptoms of autism often become noticeable around the same age the MMR is given. That timing created the false impression that one caused the other.

The media amplified the claim, framing it as a legitimate scientific debate when in fact there was no supporting data. In response, researchers and governments around the world conducted dozens of large-scale studies to test the hypothesis. The first major one, from Denmark in 2002, followed more than half a million children and found no association between vaccination and autism. Many more studies since—using registry data from the U.S., Japan, Sweden, Israel, and elsewhere—have reached the same conclusion.

Even so, the myth persists. Vaccination can be an emotional experience for parents: it happens when children are very young and vulnerable, and the diseases vaccines prevent are no longer visible in daily life. That combination makes vaccines, as many public health experts say, a victim of their own success.

Scientifically, the verdict is clear—vaccines do not cause autism. What keeps this idea alive isn't evidence but emotion, misinformation, and the erosion of trust in institutions.

BM: Would it be correct to say that autism spectrum disorder begins in utero?

AH: It begins in utero, and some researchers would even say the biological groundwork starts at conception.

One of the known factors that increases the likelihood of an autism diagnosis is advanced parental age, especially in fathers. Scientists have investigated why that is, and one explanation involves de novo mutations—genetic changes that occur spontaneously in sperm or egg cells and aren't inherited from either parent.

Because collecting eggs for research is invasive, most of the data comes from sperm studies. These have shown that sperm from older men are more likely to carry new mutations, and some of those mutations have been found in children with autism. It's important to emphasize, though, that this doesn't explain all cases. Many young parents have autistic children, so this is just one biological pathway among many.

What's interesting about this finding is that it illustrates how genetic and environmental factors interact. Age itself isn't a cause; it's a condition that can increase the likelihood of certain mutations that might influence brain development.

There's also growing evidence that some autism-related traits and vulnerabilities can be passed down across generations, even through non-genetic mechanisms—things like epigenetic changes or factors in the prenatal environment. So, whether it's through inherited genes, de novo mutations, or early developmental processes, the consensus is clear: autism's origins lie very early in life, most likely before birth.

BM: That brings up an important point: if autism's origins lie in utero—or even at conception—then the vaccines children receive at 18 or 24 months occur long after those developmental changes have taken place. So, it couldn't be the vaccines. Is that a fair conclusion?

AH: Absolutely. We know that the biological processes underlying autism begin far earlier than when vaccines are given.

When scientists have been able to follow infants who are later diagnosed with autism, they find measurable brain differences as early as six months of age—long before the 18- to 24-month period when the MMR vaccine is administered. Those differences don't allow us to make a diagnosis yet, but they show that autism begins as a process of early brain development, not as something caused later in childhood.

Researchers have also seen this in laboratory models. Using stem-cell technology, they can take a person's skin cell, reprogram it into a neuron, and observe how it behaves as it connects with others. Even at that earliest stage, we see distinct patterns in cells from people with autism compared to those without it. These findings make it clear that autism's biological roots form before birth.

For parents, though, the developmental milestones can be confusing—especially for first-time parents who may not know what to expect. Pediatricians track things like babbling, pointing, eye contact, or social play, but those are broad ranges, not hard rules. Every child develops at a different pace. That's why public health groups have created tools like Autism Navigator, which help families recognize early signs and bring questions to their doctors.

So, my message to parents is, "Please don't blame yourselves. Autism isn't caused by something you did or didn't do, including vaccination. Timing alone makes that impossible. Autism is nobody's fault."

Environmental factors in autism

BM: Along this line of inquiry, what environmental factors are researchers at the Autism Science Foundation studying? Have any been identified as potential contributors? And secondly, are there tests—such as serum markers or imaging studies—that can detect early indicators of autism?

AH: There are several environmental factors that can contribute to the likelihood of an autism diagnosis, but none that cause autism on their own. It's important to understand that these are risk modifiers, not direct triggers.

One example is valproic acid, an anti-seizure medication that's known to significantly increase the risk of autism and other developmental differences when taken during pregnancy. Women who are pregnant or planning to become pregnant should not stop medication on their own but should talk to their physician about switching to a safer alternative. Valproic acid carries a specific warning for this reason.

Other contributing factors include premature birth and very low birth weight. Advances in neonatal care mean that babies born before 27 weeks or weighing less than a pound now often survive, but they can face a range of neurodevelopmental challenges, including a higher risk for autism.

Parental age is another consistent association, particularly advanced paternal age. As we discussed earlier, this seems related not to age itself but to genetic mechanisms that become more common in sperm as men get older.

There are also links between maternal illness during pregnancy, especially infections accompanied by high fever, and a modest increase in risk. Air pollution exposure, particularly during the first or second trimester, has also been associated with higher rates of autism, but usually at very high levels—for example, in families living near major highways or agricultural equipment emitting heavy particulate matter.

Finally, pregnancy complications such as maternal diabetes, preeclampsia, or prolonged labor may play a role, though it's not clear whether the risk comes from the underlying condition or the physiological stress of the pregnancy itself.

So, these are all contributing factors that may add to genetic vulnerability. None of them, individually or collectively, can be said to cause autism, but they help researchers understand the complex interplay between biology and the environment.

To your other question, although early signs of autism can appear before a year of age, a diagnosis is typically not made until at least 16 months of age. Unfortunately, the average age of diagnosis in the US is still around 4 years of age for boys. We have a long way to go to ensure early detection and intervention for all with a diagnosis.

BM: Returning to Andrew Wakefield and the *Lancet* controversy, it's striking that his claims emerged during a period when vaccines were among the great public-health success stories. By the 1970s and 1980s, vaccination campaigns had drastically reduced childhood disease and mortality. At the same time, clinicians were beginning to better recognize and diagnose autism spectrum disorders.

Do you see a connection between these overlapping developments—and perhaps the rise of more affluent, educated groups of parents—who began attributing their children's neurodevelopmental challenges to vaccines?

AH: I think it was a mixture of several things happening at once. In the 1980s and early 1990s, there was relatively little scientific research on autism, but that began to change with the rise of parent-led advocacy groups. Organizations like the National Alliance for Autism Research (NAAR) and Cure Autism Now (CAN)—both founded in the early 1990s—really helped push autism research and public awareness forward. The Autism Society of America, which was already established, also became more active in promoting education and advocacy.

These groups successfully lobbied for federal funding and helped build momentum for scientific inquiry into autism's causes and early detection. In 2004, the CDC launched its "Learn the Signs. Act Early." campaign to promote awareness of developmental milestones, not just for autism but for child development more broadly. So, the early 1990s became a kind of watershed moment when advocacy, science, and public health began working together to expand recognition and understanding of autism.

Then came the Wakefield paper, published in *The Lancet* in 1998 and later fully retracted. All of Wakefield's co-authors withdrew their support once they realized the findings couldn't be replicated. But by then, the damage was done. The study had been published in one of the world's most respected medical journals, which gave it enormous visibility and legitimacy.

Without that paper, I think the trajectory of autism research might have unfolded differently—perhaps more squarely focused on genetics and neurodevelopment rather than being sidetracked by disproven vaccine theories. But vaccines are an easy thing to blame. They're visible, they involve putting something into the body, and they go against what some people perceive as "natural," especially when it comes to infants.

We've seen this pattern repeat itself with COVID-19: people blaming the vaccine for causing long COVID, or claiming the vaccine gives you COVID, when in reality it's the virus itself that causes those outcomes. When something works as well as vaccines do—quietly preventing disease and saving lives—it paradoxically becomes a target. Because when you no longer see the diseases vaccines prevent, it's easy to forget what's at stake.

Anti-vaccine ideology

BM: If we step back for a moment, it seems that the resistance to vaccines isn't only about science or safety—it's also about ideology. Vaccines represent something deeply social: they don't just protect the individual who gets them, they protect the community. Their success depends on a shared sense of responsibility, a recognition that health is collective, not private.

But that idea—of public health as a social good—runs directly against the grain of a certain worldview that sees medical decisions purely through the lens of personal freedom. The notion that society has any claim on the individual, even in the name of protecting others, provokes a kind of visceral backlash.

Do you see that tension—between individual liberty and collective responsibility—as part of what has historically fueled the anti-vaccine movement?

AH: I think that's absolutely true—there's a philosophical resistance to the idea that health is collective. But layered on top of that, we now have an information environment that amplifies misinformation at an unprecedented scale.

I'm not blaming everything on the internet, but the rise of social media and influencer culture has completely changed how people encounter information about health. Anyone can post something dramatic or conspiratorial, and if it goes viral, they might even be rewarded for it—financially or socially. That's a powerful incentive to spread content that may not be accurate.

There's also evidence that coordinated disinformation campaigns have targeted vaccines specifically. Studies done around the 2016 and 2020 U.S. elections traced a significant number of anti-vaccine posts to automated accounts and foreign bot networks, designed to sow confusion and mistrust. So, it's not just individuals misunderstanding science; it's also a system that profits—politically or economically—from undermining public trust.

Put together, those forces—ideological individualism, misinformation, and deliberate manipulation—create the perfect storm. People are exposed to falsehoods constantly, and when those messages come from someone they already follow or trust online, they feel credible. Meanwhile, the voices of science and public health often struggle to compete in that same emotional, fast-paced space.

So yes, it's about liberty and distrust, but it's also about the way our

modern information ecosystem rewards outrage and uncertainty. And that combination has made vaccine misinformation extraordinarily resilient.

BM: As we begin to wrap up, I want to return to something you just touched on—the way science itself can become a political battleground. The recent Tylenol controversy is a good example: evidence was selectively framed and amplified to serve a narrative, not the data.

What lessons do you think this moment holds about the relationship between evidence and power? And when science is misused or systematically undermined, how should the scientific community respond?

AH: I wish there were a single, elegant answer to how we should respond when science becomes politicized—but what I can offer instead are some personal "do's and don'ts" I've learned from watching misinformation spread.

First, listen to your doctor. Trust the professionals who know you, who have reviewed your medical history, and who are trained to interpret evidence—your pediatrician, your OB-GYN, or your family physician. These are the people equipped to give individualized medical advice, not the voices on social media who may be speaking without accountability or expertise.

Second, question motives. Many social media influencers are paid to create content, and there's currently no universal requirement that they disclose sponsorships or financial ties. If someone posts a video claiming that Tylenol or vaccines cause autism, ask yourself: who benefits from this message? Are they being compensated by a rival company or promoting an alternative product? Even if not, they might still be chasing views, followers, or controversy—because online outrage is profitable.

Third, beware of simple explanations. Autism is a profoundly complex neurodevelopmental condition. If anyone claims that a single factor—whether acetaminophen, vaccines, air pollution, or parental age—explains all cases of autism, that should raise every red flag. The same goes for anyone promising a universal "cure." No credible science supports such claims.

Finally, consider the source. Before accepting any scientific claim, look for where the evidence comes from. Is it based on peer-reviewed studies, or just someone's opinion? Has it been replicated, or is it anecdotal? True experts—the physicians and scientists who dedicate their careers to studying neurodevelopment, pregnancy, and child health—are constantly reading and re-evaluating the science. Those are voices worth listening to.

In the end, combating misinformation isn't just about debunking falsehoods—it's about rebuilding trust. Trust in expertise, trust in the scientific process, and trust that most people working in this field genuinely want to help children and families live healthier lives.

BM: Thank you for these excellent insights. As we close, are there any final thoughts you'd like readers to take away from this discussion?

AH: I think it's important to keep an eye on how this conversation unfolds, especially around the political attention being given to acetaminophen. There are investigative journalists already examining possible financial and political ties behind these claims, and it's worth asking why certain figures are suddenly focusing on a single medication when the study they cite was completed long before these recent announcements.

The broader body of research still points to genetic and gene-environment interactions as the most credible explanations for autism. That's where the science continues to move—toward understanding how genetics intersect with the environments in which people live, grow, and develop.

On a positive note, the National Institutes of Health recently announced $50 million in new funding across 13 research sites in the United States to study those very interactions. The goal is to look broadly—not just at chemical exposures or medications, but also at social and environmental contexts such as neighborhood conditions, access to healthcare, maternal health during pregnancy, and other contributing factors.

That kind of comprehensive, collaborative research is exactly what's needed right now. It's encouraging to see support for rigorous science that considers autism in all its complexity. And if readers would like to learn more, we just released a new episode on the ASF Weekly Science Podcast that discusses these studies in greater detail.

BM: Dr. Halladay, thank you for all the time.

AH: Thank you—and thank you for helping share accurate information with your readers. It's so important that people understand the science, the evidence, and how to interpret it, rather than being swayed by every new "flash-in-the-pan" claim about what causes autism. Most of those theories aren't even biologically plausible.

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The day after the interview, Dr. Halladay sent a link to a critical research published on October 5, 2025, by international researchers in the journal Child & Adolescent Psychiatry *which is a systematic review and meta-analysis that studied acetaminophen use during pregnancy and the risk of neurodevelopmental disorders in childhood. She also explained that "this more rigorous meta-analysis published recently failed to show an association between neurodevelopmental disorder—autism—and Tylenol use in pregnancy. This calls into question how much we should be relying on small studies with methodological problems."*

Indeed, the authors of the study found no convincing evidence of a link to autism. When studies relied on confirmed medical diagnoses, the results showed no increased risk. One smaller study using a screening questionnaire, not a diagnostic test, which suggested a possible connection. However, that type of data isn't considered reliable. A large sibling study, which helps account for shared genetics and family factors, also found no difference in autism rates between children exposed and unexposed to acetaminophen in the womb.

For ADHD, the analysis found a modest increase in risk, about 17 percent higher on average, among children whose mothers reported acetaminophen use during pregnancy. Yet, this finding weakened or disappeared in studies that used more rigorous designs, such as sibling comparisons, suggesting that family traits or other confounding factors might explain the difference. Researchers also noted that most studies couldn't precisely measure how much acetaminophen was used, when during pregnancy it was taken, or why, which makes drawing firm conclusions difficult. Basically, this comprehensive study shows that acetaminophen use in pregnancy has not been proven to cause autism or ADHD. The small association seen for ADHD remains uncertain and may not reflect a true effect.