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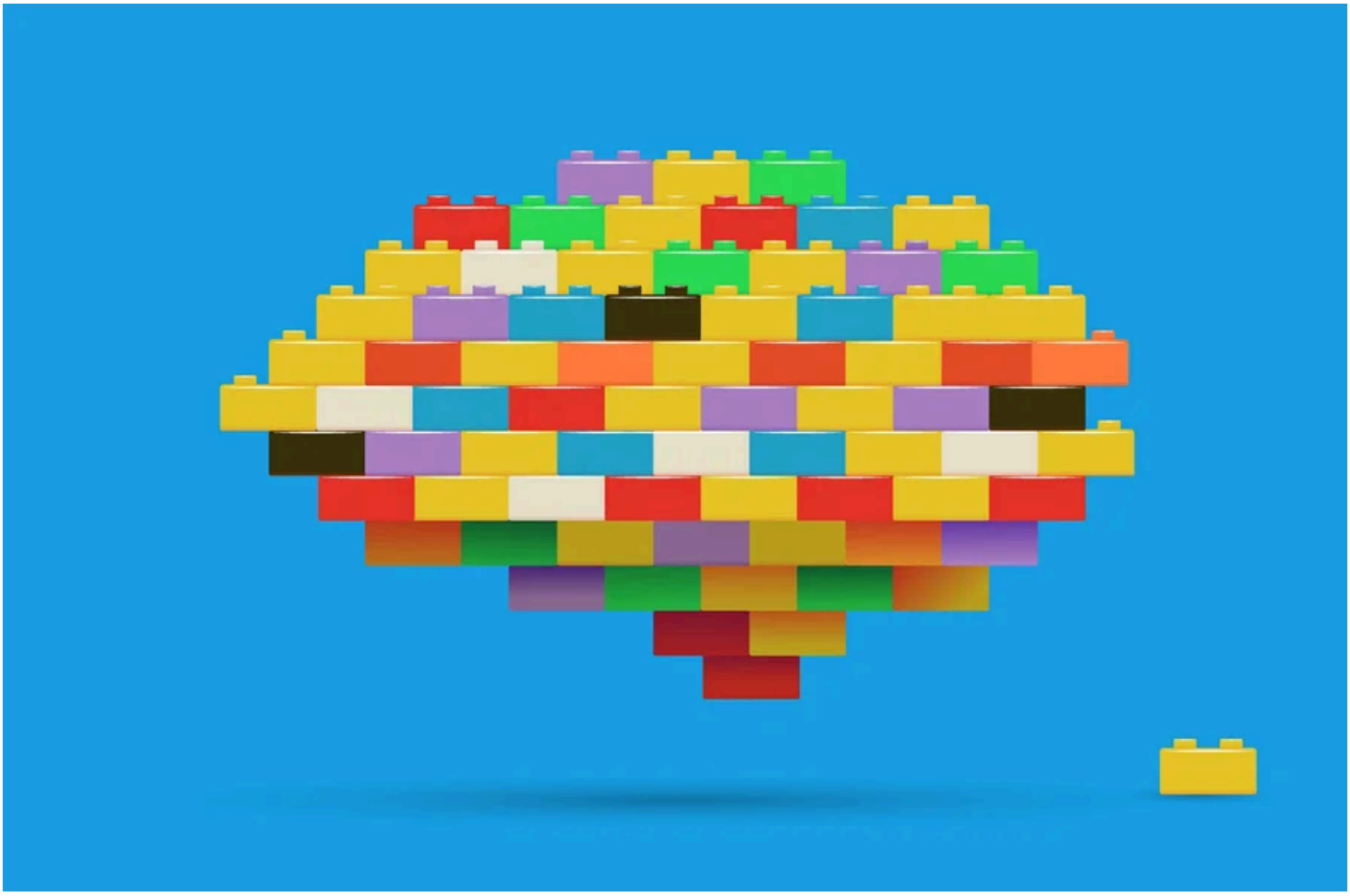
Autism Has No Single Cause. Here's How We Know

Scientists will not find a simple answer to how autism arises, despite Robert F. Kennedy, Jr.'s promise to announce its causes sometime this month. Here's what makes the condition so staggeringly complex

BY ALLISON PARSHALL EDITED BY DEAN VISSER



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Autism

Soon after psychiatrist Leo Kanner first identified autism in the 1940s, he and his colleagues proposed a simple explanation for its cause: mothers' "lack of genuine warmth" toward their children. Being raised by "refrigerator mothers," the researchers explained, was what caused autistic people's difficulties with social communication and sensory processing and their repetitive behaviors and interests.

But in the 1970s studies of twins revealed that autism is highly heritable, not something that develops after birth. Thus began the search for the genes responsible. "We had rather simple views about what it might be" that caused autism, says Helen Tager-Flusberg, a professor emerita at Boston University. The idea in the 1990s, she recalls, was that "we're talking about six to 10 genes." Instead researchers found hundreds.

No simple theory of autism has ever panned out, and the scientific community has moved on from the search for a simple answer. Researchers now know that autism develops from a staggeringly complex interplay between genes and factors that can influence development in utero. But attempts to pin the condition on one root cause abound, most famously in the disproven idea that vaccines cause autism. And earlier this year U.S. Secretary of Health and Human Services Robert F. Kennedy, Jr., announced that he will reveal the "interventions" that are "almost certainly causing autism" in September.

While scientists rarely proclaim absolutes, autism researchers say that they are as sure as they can be that there's no one cause of the condition. "It's never going to be true," says Tager-Flusberg; decades of data reveal that a complex (and highly variable) origin is the only logical conclusion.

THE GENETIC PICTURE

In the past 50 years, researchers have identified hundreds of genes linked to autism spectrum disorder. But which genes are involved varies greatly from person to person and can be much harder to pin down. About 10 to 15 percent of cases (some estimates are as high as 39 percent) involve genetic mutations that are new to a child—not inherited from their parents—explains Shafali Jeste, an autism researcher and pediatrician, who leads the neurology division at Children's Hospital Los Angeles. In another approximately 50 percent of cases, autism is linked to the combination of many common variants of genes that were inherited from parents. In the remaining cases, the causes are murky.

But even for new genetic mutations that are known to cause autism, the story is far from simple. The same mutations can be present in nonautistic people, too. "If you have this mutation, it doesn't guarantee that you will have autism per se, but it increases the risk substantially," says Jed Elison, who studies brain development and autism at the University of Minnesota. There are still other factors involved that researchers haven't fully characterized—some that are likely genetic and some that are likely not.

Untangling genetic factors from nongenetic ones (which scientists call "environmental factors") can be tricky. For example, studies have consistently shown that parental age at conception can play a role, with older parents being more likely to have autistic children. But that could be because of the effect of age on genes: people accumulate mutations with age and can pass these on to their kids. Other factors that have been linked to autism include people being born prematurely or through cesarean section, as well as pregnant people having obesity, using certain medications (such as the antiseizure drug valproate and the pain reliever acetaminophen, the active ingredient in Tylenol) and being exposed to air pollution. The strength of the evidence for

these links varies, though, and the increases in risk tend to be small. The evidence is also only correlational, meaning it can't establish what caused what. For acetaminophen, for example, researchers can't fully separate the effects of the medication from the effects of the underlying conditions that may lead people to take pain relievers during pregnancy, explains David Mandell, a professor of psychiatry studying autism at the University of Pennsylvania. Overall, "the evidence [from these studies] was really mixed, and the effects were really small," he says. Acetaminophen is also usually used as a fever reducer, which pregnant people might take if they are fighting an infection. Both infections and uncontrolled fevers during pregnancy have been linked to higher rates of autism. "We know that the neurodevelopmental outcomes of having an uncontrolled fever are worse than what we're observing for acetaminophen," Mandell says.

Other proposed environmental factors have not been supported by any credible scientific evidence or studies. This category includes vaccines and many vaccine additives, such as aluminum and thimerosal. Studies of hundreds of thousands of children, born across multiple decades in the U.S. and Denmark, have shown that those who are vaccinated are not at any higher risk of being diagnosed with autism. In contrast, the 1998 study that first purported to show that a vaccine caused autism was carried out in just 12 children and turned out to be a case of egregious fraud; the doctor had been paid to fabricate results to support lawsuits being brought against vaccine manufacturers.

It is extremely unlikely that any single environmental factor—or any unique combination of factors—could explain what causes autism, according to many experts. "Have we tested every other possible environmental factor? Not directly," Jeste says. "But I think what we do know, based on large epidemiological studies and studies that have examined health records where

you can look at lots of different environmental factors, is that there has not been shown to be one clear association. And I think that's going to hold up."

Doctors and researchers know this is an unsatisfying answer. "I've dedicated my life to doing research and understanding autism better and developing therapeutics that can help kids," Jeste says. "If I had a magic test that could tell a parent, 'This is why your child has autism; this is the treatment that is going to help improve their quality of life,' of course I would be the first person to be providing that to my families. [But] it's not that straightforward."

WHY IS AUTISM SO COMPLEX?

Some neurodevelopmental conditions do have straightforward causes. For example, Rett syndrome, which has a great deal of overlap with autism, arises from a mutation of the *MECP2* gene. But for "other neurodevelopmental or neuropsychiatric conditions—schizophrenia, depression, ADHD [attention deficit hyperactivity disorder]—the story is not that dissimilar" to autism, with genetics and the environment interacting in variable and often unpredictable ways, Jeste says.

Without a perfect understanding of how the brain develops and works—something science is unlikely to achieve for decades, if not a century—mental health professionals generally define conditions by how they look from the outside, not by their causes. They give names to common clusters of traits and only consider something a "disorder" when it impedes someone's life. This means that, unlike Rett syndrome, there's no clear biological line between someone who is depressed and someone who is not, between someone who has schizophrenia and someone who doesn't or between someone who is autistic and someone who isn't.

Diagnoses with firm cut-off lines are necessary for scientific research and insurance reimbursement, making them an unavoidable part of life. “But that’s not how biology works,” says Catherine Burrows, a neuroscientist studying autism at the University of Minnesota. In reality, the clusters of traits involved in conditions like autism are fuzzy around the edges. Many people exhibit autistic traits that may not meet the criteria for a diagnosis. Some researchers hypothesize that there is a “broader autism phenotype” that runs in families, supported by research showing that family members of autistic people often have “milder” versions of key autistic traits, such as difficulties with social communication, repetitive behaviors and sensory processing issues.

Whether these subclinical traits should “count” as autism is a matter of ongoing debate within the scientific field, as well as the autistic community. Still, it is clear that autism is not just one thing but rather a constellation of traits that often occur together and can be used to identify a group of people who may benefit from similar types of support. In some ways, Elison says, “we think of it like an artificial category”—a category that is useful and the best we have at the moment but one that is unlikely to be emerging from any one root cause.

“When we say autism, we’re probably talking about *autisms,*” Mandell says. “We're probably talking about a lot of different biological pathways to get to a [condition that outwardly] looks similar.”

Over time, the category of autism has been expanding to be useful for more people. The criteria were originally designed around how the condition presented in white, affluent boys, but diagnoses among girls, people of color and adults have been increasing. Also, people who, in previous decades, may have received more generic diagnoses such as “intellectual disability” (which

replaced the now offensive term "mental retardation") are now being diagnosed with more specific labels such as autism. "We're playing catch-up right now," Burrows says—and that's leading to the appearance of a rise in autism's prevalence in more recent years.

THE REAL NEED

A large portion of autism research funding has gone toward searching for causes of the condition. And while this research is crucial, it seems unlikely to improve the lives of autistic people and their loved ones in the short term.

"The average autistic person, or their average family member, doesn't wake up in the morning thinking, 'Oh, have they discovered a better mouse model [for simulating autism in laboratory research]?'" explains Ari Ne'eman, co-founder of the Autistic Self Advocacy Network and a health policy researcher at Harvard University. What autistic people and their loved ones most need is research into kinds of support that effectively address their day-to-day needs. A rebalancing of our research priorities, he says, is "long overdue."

But, Ne'eman adds, "there is no pathway to meeting those needs absent a well-financed Medicaid system." About 5 percent of U.S. children on Medicaid are diagnosed as autistic, whereas about 2 percent who are privately insured or uninsured receive that diagnosis. The $1-trillion cuts to Medicaid that have been carried out under the Trump administration and effectively defended by Kennedy, however, could imperil many autistic people's access to services. "This is something that is going to be catastrophic for autistic people, for all people with disabilities, regardless of severity of impairment—but especially for people with the most severe impairment," Ne'eman says.

Jeste worries about this, too. “I work in a safety-net hospital, where we are taking care of kids who are mostly federally insured [with] Medicaid, and many are not English-speaking,” she says. Because children disproportionately rely on Medicaid for health care, pediatric institutions also rely heavily on Medicaid coverage to keep the lights on. “So every child, no matter what their insurance provider is or how they’re paying for their health care, will be affected by cuts to Medicaid.”

ALLISON PARSHALL is an associate editor at *Scientific American* covering mind and brain and she writes the weekly online Science Quizzes. As a multimedia journalist, she contributes to *Scientific American*'s podcast *Science Quickly*. Parshall's work has also appeared in *Quanta Magazine* and Inverse. She graduated from New York University's Arthur L. Carter Journalism Institute with a master's degree in science, health and environmental reporting. She has a bachelor's degree in psychology from Georgetown University.

More by Allison Parshall

Thank you,
David M. Ewalt, Editor in Chief, *Scientific American*

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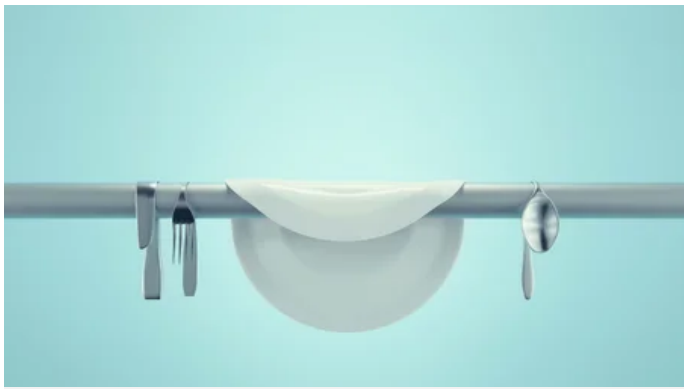

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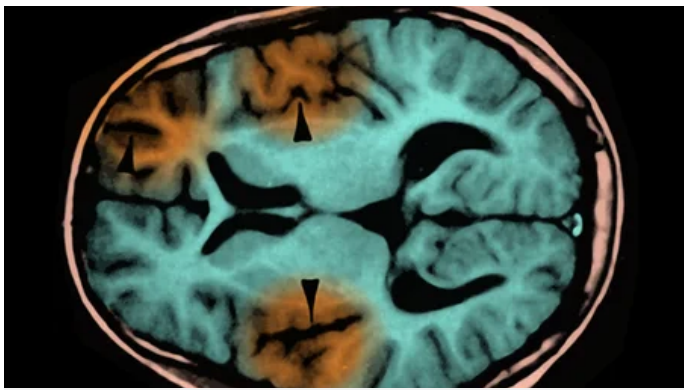

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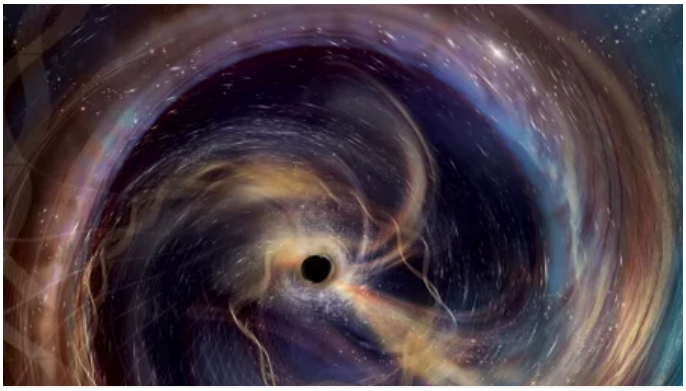

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