

Autism and Neurodiversity:

Rethinking What It Means to be Different

Introduction

Autism is a relatively new idea. Throughout history, there have almost certainly been people whose minds worked in ways that today we would recognize as autistic. But autism itself is remarkably young, only a little more than eighty years old. And in those eighty years, we have changed our minds repeatedly about what autism is.

I want to begin with two adults.

The first is a successful professional. Perhaps she is a physician, an attorney, a scientist, or a professor. She has always found large social gatherings exhausting. She prefers routines and becomes disproportionately uncomfortable when plans change unexpectedly. She has intense interests and can talk about them at considerable length. She learned years ago that people expect eye contact, so she makes herself do it, although she finds it distracting. Before important conversations, she mentally rehearses what she will say. She has a spouse, a successful career, and several graduate degrees. At age 52, she is diagnosed with autism. For her, the diagnosis may finally explain a lifetime of feeling out of step with other people.

The second adult does not speak. He has significant intellectual disabilities and epilepsy. He needs help dressing, bathing, and eating. When distressed, or unable to communicate pain, he sometimes injures himself. He will need substantial support throughout his life. His parents are growing old, and they worry about what will happen when they can no longer care for him. He, too, is autistic.

The distance between these lives is almost difficult to comprehend. One person may experience autism as an inseparable part of identity; yet another family may live with profound disabilities and the daily work of caregiving. Yet our diagnostic language places them together.

So today I want to talk about autism from several perspectives: what it is, how we came to define it, what we know—and still do not know—about its causes, why diagnoses have increased so dramatically, and the way many people think about treatment.

Part I — From Diagnosis to Neurodiversity

In 1943, psychiatrist Leo Kanner at Johns Hopkins published a paper describing eleven children who seemed different from children with other psychiatric or developmental conditions. They shared an unusual constellation of characteristics: difficulties with social interaction, an

intense preference for sameness, repetitive behaviors, unusual language, and highly focused interests. ¹ Kanner called the condition early infantile autism.

His first case was a five-year-old boy named Donald Triplett. ¹ Donald is important because we know considerably more about his life than we do about most children who appeared in early psychiatric literature. He grew up in Mississippi, learned to drive, traveled extensively, played golf, and worked at a local bank. He was known around town not merely as a former patient in a famous study, but as Donald: a neighbor, a golfer, a familiar face at the bank. He lived into his eighties.² But Donald's story also illustrates something important about the history of autism: his life depended enormously on the environment around him. He had parents with the resources and determination to advocate for him. He lived in a community that largely accepted his eccentricities. He was given opportunities to participate rather than being permanently separated from society. ² For many people with developmental disabilities in that era, the outcome was very different. Institutionalization was common. Children might be described as “feble-minded,” mentally retarded, schizophrenic, or simply uneducable. Families were frequently advised to place children in institutions and move on with their lives. So, autism did not suddenly appear in 1943. We simply acquired a new way of seeing some people who had been there all along.

By the 1950s and early 1960s, psychogenic theories of autism gained enormous influence. Autism, according to this thinking, could result from emotionally distant parenting, particularly emotionally distant mothers. ³ The most famous popularizer of this idea was psychologist Bruno Bettelheim. Dr. Bettelheim directed a residential treatment school for children with emotional and developmental difficulties affiliated with the University of Chicago.³ The phrase that became associated with this period was “refrigerator mother.” The theory was devastating. Imagine being the parent of a child who does not speak, who struggles to interact with other people, or who exhibits behavior no one understands. You go to professionals seeking an explanation. And the professionals tell you that the explanation is you. More specifically, very often, the mother. There was no convincing scientific evidence that emotionally cold parenting caused autism.

By the mid-1960s, researchers were beginning to challenge this theory. Among them was Bernard Rimland, a psychologist in San Diego and the father of an autistic son. His landmark 1964 book, *Infantile Autism: The Syndrome and Its Implications for Neural Theory of Behavior*, helped redirect the field away from blaming parents and toward understanding autism as a condition rooted in biology.⁴

Rimland went on to become an important advocate for autistic children and their families. In 1965, he helped establish the National Society for Autistic Children, which later became the Autism Society of America. Two years later, he founded the Institute for Child Behavior Research

in San Diego, later renamed the Autism Research Institute, which he directed for decades.⁴ His influence extended beyond challenging a harmful theory. He helped establish organizations that gave families a voice and made autism a serious subject of scientific investigation.

But the refrigerator-mother era, one that Rimland helped to end, is worth remembering for more than its historical interest. The refrigerator-mother era reminds us that even widely accepted scientific ideas must be challenged. Progress depends on our willingness to question prevailing assumptions—and the humility to recognize that what we believe with confidence today may look very different tomorrow.

Over subsequent decades, autism moved away from psychological theories and toward an understanding rooted in development and biology. Its diagnostic boundaries also expanded. People who would once have received other diagnoses, or none at all, increasingly came under the autism umbrella. Today, the American Psychiatric Association's diagnostic framework places what were previously several separate diagnoses—including autistic disorder and Asperger's disorder—under the umbrella of autism spectrum disorder, or ASD.⁵ The defining features involve persistent differences or difficulties in social communication and social interaction, together with restricted or repetitive patterns of behavior, interests, or activities. Symptoms begin during development and must cause clinically significant impairment, although they may not become fully apparent until social demands exceed a person's capacities.⁵

The term *neurodiversity* emerged in the late 1990's, particularly through the work of sociologist Judy Singer at the University of Technology Sydney, and the growing ability of autistic people to connect with one another through the early internet.⁶ The idea behind neurodiversity is fairly simple: human brains do not all work the same way. Just as people differ in their physical characteristics, they also differ in how they think, learn, communicate, and experience the world. Autism is one example of this neurological variation, but it is far from the only one. ADHD, dyslexia, and other neurological or developmental differences also fall under the broader umbrella of neurodiversity. We often describe individuals with these differences as *neurodivergent*, meaning their brains function differently from what society considers typical.

But there is an important distinction. Autism is a clinical diagnosis. Neurodiversity is not. Neurodiversity describes the variation that exists among human brains, while the neurodiversity movement asks us to think differently about that variation.⁷ It challenges us to reconsider which differences truly represent disorders that require treatment—and which might simply be different ways of experiencing the world. For example, we learned long ago to stop forcing left-handed children to use their right hands. Sometimes the sensible intervention is to change the environment rather than the person. Some autistic advocates have asked whether at least some features of autism should be understood similarly.⁷

If an autistic person avoids eye contact because eye contact is distracting or uncomfortable, must we train that person to maintain eye contact? If someone regulates sensory or emotional overload by rocking or moving their hands—a behavior often called *stimming*, and the behavior harms no one, why should eliminating it be considered a therapeutic success? If someone prefers direct, explicit communication to hints, implications, and social ritual, is that inherently a communication deficit?

Take Temple Grandin, an autistic animal scientist, professor, and advocate. She did not begin speaking until she was three and a half years old yet went on to earn a Ph.D. and revolutionize the handling of livestock through her remarkable ability to think in pictures. She has written about seeing detailed images rather than first assembling ideas into words.⁸ That way of perceiving the world helped her notice features of livestock facilities that other designers overlooked: shadows, reflections, abrupt contrasts, small things that might frighten an animal moving through a chute. She used that attention to reshape the way livestock-handling systems were designed.⁸ Grandin's story does not mean that autism confers an exceptional talent on everyone who has it. Her achievements are her own. But her work challenges the assumption that there is only one proper way to perceive and understand the world.

Consider again the two adults with whom we began.

For the professional diagnosed at 52, the neurodiversity framework may be liberating. A diagnosis may finally explain a lifetime of feeling slightly out of step with everyone else. She may not want anyone to cure her intense concentration, attention to detail, directness, or unusual way of thinking. Those characteristics may be intertwined with the very abilities that made her successful. She may reasonably say: If you could remove my autism, the person who remained would no longer entirely be me.

But what about our second adult? What does the analogy to left-handedness mean to parents whose adult child cannot communicate that he is in pain? What does it mean to a family managing severe self-injury, intellectual disability, or uncontrolled epilepsy? What does “difference, not disability” mean when parents in their seventies are still providing round-the-clock care—and lying awake at night wondering what will happen when they die?

Autism can be an identity, and it can be a disability.

Autism can be associated with remarkable abilities, and it can coexist with profound impairment.

Part II — The Biology of Autism: What We Know and What We Don't

These very different experiences raise a scientific question: what do they have in common biologically? The answer is more complicated than we once imagined. We know a great deal

about factors that contribute to autism, particularly genetics. But we have not identified a single cause, or even one developmental pathway that explains everyone who receives the diagnosis.

Autism is strongly influenced by genetics. But there is no single autism gene. Researchers have identified many genetic variants associated with autism. Some are common and have small effects; others are rare but more consequential.⁹ Many of the genes associated with autism are involved in brain development: the formation and function of synapses, regulation of gene expression, neuronal communication, and the development and refinement of neural circuits.⁹ One particularly interesting process is called *synaptic pruning*. Early in childhood, the brain produces an extraordinary number of connections between neurons. As we grow, some connections are strengthened while others are eliminated. This is a normal and essential part of brain development. Think of pruning a tree. A gardener selectively removes branches, allowing the remaining branches to grow and function more effectively. The developing brain does something similar, refining its network of connections over time. But researchers have found evidence that this pruning process may occur differently in autism.

In a 2014 study at Columbia University, scientists examined brain tissue from autistic children and adolescents and compared it with tissue from individuals without autism. They found that the density of synaptic connections declined by approximately 45 percent from childhood to adolescence in the comparison group, but by only about 16 percent in the autistic group.¹⁰ In other words, the autistic brains in this study appeared to retain more of the connections that would ordinarily be eliminated during development. This was a relatively small study, and altered synaptic pruning is not a universal explanation for autism. But it offers a fascinating example of how differences in the timing and refinement of neural connections might contribute to the development of an autistic brain.¹⁰

Beyond genetics, researchers have attempted to study the physical brain structure, but there is no single damaged region, or characteristic lesion, or pattern of connectivity that is identified in every autistic person. Some of the most fascinating brain studies in autism have examined what happens when an autistic child looks at something familiar or personally meaningful. In a 2002 study, researchers showed children photographs of their own mothers, unfamiliar women, favorite toys, and unfamiliar toys while measuring their brain activity.¹¹ Typically developing children showed different brain responses to their mother's face compared with a stranger's. Autistic children did not show that same distinction, although their brains did respond differently to familiar and unfamiliar toys.¹¹ A few years later, researchers studied an autistic boy who was fascinated by the Japanese cartoon characters Digimon.¹² When he looked at human faces, brain regions involved in facial recognition showed relatively little activity. But when he looked at Digimon characters, those same regions became active.¹² Interestingly, later

research found that autistic children could show more typical brain activity when looking at their mothers or other familiar children, compared with unfamiliar adults.¹³ These findings suggest that differences in brain activity may reflect more than an inability to recognize or process faces. Familiarity, attention, and personal interest may influence how the brain responds. In other words, when a brain scan looks different, it doesn't necessarily mean something isn't working. Sometimes the difference may lie in what the individual finds meaningful.

A 2025 study published in *Nature* complicated the picture further. Researchers found that people diagnosed earlier and those diagnosed later showed somewhat different developmental patterns and polygenic profiles. This does not establish two kinds of autism but suggests that one diagnosis may encompass several developmental pathways.¹⁴

Remember our two adults? Perhaps their differences do not simply represent two points on a line from “mild” to “severe.” Perhaps the spectrum is not really a line at all. That possibility has implications for diagnosis, treatment, and research and for the neurodiversity debate itself.

Part III — Why Are Autism Diagnoses Increasing?

If the autism spectrum encompasses several developmental pathways, then the boundaries of the diagnosis become especially important. And those boundaries have changed considerably over the past several decades. One consequence has been an extraordinary increase in the number of people identified as autistic.

In 2000, the CDC identified autism in approximately one in 150 eight-year-old children in the communities it monitored.¹⁵ By 2012, that figure was approximately one in 69.¹⁵ In 2022, approximately one in 31.¹⁵ That is an increase of nearly fivefold in 22 years. One in thirty-one is a striking number, but it is not a direct count of how many children have suddenly developed a new biological condition. It reflects the broader diagnostic criteria, enhanced screening practices, and increased awareness and recognition of autism that we now have today.¹⁵ Still, those changes may not explain the entire increase. Researchers continue to investigate whether genetic, biological, and environmental factors also contribute.

But when the number of children diagnosed with a condition rises so dramatically, people understandably want an explanation. And sometimes, what they really want is someone—or something—to blame. We've already seen this tendency in the refrigerator-mother theory, when parents themselves were blamed for their children's autism. More recently, the blame has shifted from mothers to something else entirely: childhood vaccines. And that brings us to one of the most damaging claims in autism's history. Despite extensive research involving more than a million children, no causal relationship has been found. Yet the claim has persisted, in part because parents understandably want to know why their child's development has taken an

unexpected course.¹⁶ There is something particularly cruel about offering families a simple, confident answer that the evidence cannot support. We did that once when we blamed “refrigerator mothers.” When will we learn to be suspicious of any single, simple explanation for something so complex?

But the expansion of diagnostic criteria has another consequence: it helps explain why so many adults spent much of their lives without a diagnosis. Return to our fifty-two-year-old professional. As a child, she may have studied social rules almost as one studies a foreign language. She learned when to smile, how long to look someone in the eye, and which conversations required a conventional response rather than a literal answer. From the outside, she looked socially competent. From the inside, she may have been working very hard. This is often called masking or camouflaging. It allows a person to succeed professionally but leaves them exhausted by ordinary social demands. It also complicates the familiar phrase “high functioning.” That phrase describes what an observer sees, not necessarily the effort required to produce it.¹⁷ Her experience illustrates another limitation of studying autism from the outside. Whether we are observing behavior or measuring brain activity, what we can see does not necessarily tell us what the individual experiences are. And that distinction becomes especially important when we decide what aspects of autism need to be treated.

Part IV — Treatment, Support, and Life Beyond Childhood

Return to the adult who cannot speak. He may be able to understand far more than he can express; he may also have significant difficulties with language and cognition. We cannot determine either from his silence alone. Speech is not a direct measure of thought. And a person who cannot reliably tell us what hurts or what he wants is especially vulnerable to being misunderstood.

Consider the story of Carly Fleischmann, a nonspeaking autistic girl who, for much of her childhood, was assumed to have very limited intellectual abilities. Then, as a young teenager, she began communicating through typing. The words attributed to her revealed thoughts, observations, and an understanding of the world that those around her had not anticipated.¹⁸ Her story raises an important question: How often do we mistake an inability to communicate for an inability to think? This brings us to an important distinction: what should treatment aim to change in autistic individuals?

There is no medication that cures the core features of autism. But there are many comorbidities for which treatment can make an enormous difference. These conditions are remarkably common. In one CDC study of autistic adolescents, more than half experienced frequent sleep problems, more than a third had anxiety, and approximately one in five

experienced gastrointestinal difficulties. Epilepsy, depression, and ADHD are also important concerns.¹⁹ In fact, nearly two-thirds of these adolescents had at least one additional diagnosed condition.¹⁹ And for someone who cannot easily communicate pain, fear, or discomfort, recognizing those problems becomes especially important. Sometimes a behavior that looks inexplicable is a message. A child who hits his head may be in pain, overwhelmed by noise, frightened, or unable to make a need understood. Stopping the movement without investigating its cause may leave the real problem untouched. Teaching someone a reliable way to communicate pain or distress can be far more consequential than teaching that person to appear typical. Teaching a child to communicate, cross a street safely, or tolerate necessary medical care can increase autonomy. But some earlier behavioral programs emphasized eye contact, suppression of harmless repetitive movements, or appearing indistinguishable from other children. Autistic adults have challenged those goals.²⁰

Changing behavior is not the same as improving life. Applied Behavior Analysis, or ABA, is widely used to teach skills and change behavior of autistic individuals. Its methods and goals have changed over time. Now, the essential question is whether the goals of treatment genuinely serve the person receiving it.²⁰

Our two adults make the distinction plain. The professional may need a workplace that communicates expectations clearly and tolerates differences in eye contact. The non-speaking adult may need intensive support with seizures, communication, self-injury, and daily living. Neither person's needs can be inferred from the other's.

An emerging concern is thinking beyond childhood. Autism is lifelong, yet public attention and research have concentrated heavily on children. A 2020 analysis of federally and privately funded autism research found that roughly 45 percent supported biological research into autism, while only about four percent focused on issues across the lifespan.²¹ Autistic children become adults. They develop chronic illnesses. Some work, marry, and raise children; some require support with almost every aspect of daily life. All of them age. For the parents of our second adult, that fact has an urgent meaning. They may have spent decades learning the small signs that he is uncomfortable, the routines that calm him, the expressions that mean he is in pain. Who will know those things when they are gone? The practical question is not solved by either celebrating neurodiversity or discovering another gene.

That is where the first and second adults meet again—not because their needs are identical, but because both will have to navigate institutions that are more comfortable with a diagnostic label than with the particulars of an individual life. For families providing lifelong care, the relationship between disability and identity is not merely philosophical. Decades of

dependence, responsibility, frustration, and affection shape both the person receiving care and the people providing it.

The Japanese novelist and Nobel laureate Kenzaburō Ōe wrote movingly about his son Hikari, who was born with a brain malformation, had significant intellectual and developmental disabilities, and became a composer. Hikari was not autistic, but their relationship profoundly shaped Ōe's life.²² His writing reminds us that disability can bring substantial challenges without diminishing the value of a person's life or the meaning of their relationships. We can acknowledge both the difficulties and the richness of those relationships without romanticizing either.

Conclusion

That brings us to the question that has been present beneath this entire history: if we could cure autism, what, exactly, would we be trying to cure? For our professional diagnosed at fifty-two, autism may be woven into her attention, her interests, her perceptions, and the way she has learned to understand the world. She may not wish to have those things removed. For our second adult, treating epilepsy, relieving pain, expanding communication, and securing dependable lifelong care could transform his quality of life. It would be strange to call those goals an attack on his identity.

In *Far from the Tree*, Andrew Solomon explores how conditions we think of as disabilities can also become important parts of a person's identity.²³ Autism makes this tension especially clear, because people with the same diagnosis may have profoundly different experiences and needs.

We began with two adults and one diagnosis. The professional may want to understand rather than to correct. The non-speaking adult may need medical treatment, communication support, and a secure future after his parents are gone. Both experiences deserve to be recognized, and neither should define our understanding of everyone with autism.

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