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Have We Been Thinking About A.D.H.D. All Wrong?

With diagnoses at a record high, some experts have begun to question our assumptions about the condition — and how to treat it.

By PAUL TOUGH

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In the early 1990s, James Swanson was working as a research psychologist at the University of California, Irvine, where he specialized in the study of attention disorders. It was a touchy time for the field. The Church of Scientology had organized a nationwide protest campaign against the psychiatric profession, and Ritalin, then the leading medication prescribed to children diagnosed with attention deficit hyperactivity disorder, was one of its main targets. Whenever Swanson and his colleagues gathered for a scientific conference, they were met by chanting protesters waving signs and airplanes overhead pulling banners that read, "Psychs, Stop Drugging Our Kids."

It was true that prescription rates for Ritalin were on the rise. The number of American children diagnosed with A.D.H.D. more than doubled in the early 1990s, from fewer than a million patients in 1990 to more than two million in 1993, almost two-thirds of whom were prescribed Ritalin. To Swanson, at the time, that increase seemed entirely appropriate. Those two million children represented about 3 percent of the nation's child population, and 3 percent was the rate that he and many other scientists believed was an accurate measure of A.D.H.D. among children.

Still, you didn't have to be a Scientologist to acknowledge that there were some legitimate questions about A.D.H.D. Despite Ritalin's rapid growth, no one knew exactly how the medication worked or whether it really was the best way to treat children's attention issues. Anecdotally, doctors and parents would observe that when many children began taking stimulant medications like Ritalin, their behavior would improve almost overnight, but no one had measured in a careful, large-scale scientific study how common that positive response was or, for that matter, what the effects were on a child of taking Ritalin over the long term. And so Swanson and a team of researchers, with funding from the National Institute of Mental Health, began a vast, multisite randomized controlled trial comparing stimulant treatment for A.D.H.D. with nonpharmaceutical approaches like parent training and behavioral coaching.

Swanson was in charge of the site in Orange County, Calif. He recruited and selected about 100 children with A.D.H.D. symptoms, all from 7 to 9 years old. They were divided into treatment groups — some were given regular doses of Ritalin, some were given high-quality behavioral training, some were given a combination and the remainder, a comparison group, were left alone to figure out their own treatment. The same thing happened at five other sites across the continent. Known as the Multimodal Treatment of Attention Deficit Hyperactivity Disorder Study, or M.T.A., it was one of the largest studies ever undertaken of the long-term effects of any psychiatric medication.

The initial results of the M.T.A. study, published in 1999, underscored the case for stimulant medication. After 14 months of treatment, the children who took Ritalin every day had significantly fewer symptoms than the ones who received only behavioral training. Word went out to clinics and pediatricians' offices around the country: Ritalin worked. This was good news not only for families with children who struggled with attention issues but also for the corporations that offered them pharmaceutical solutions. In the years after the study's initial publication, Swanson began consulting for drug companies. He advised Shire, which manufactured Adderall, a similar stimulant medication, on how to formulate an extended-release version of its product, so that children could take just one pill each morning instead of needing to visit the school nurse's office in the middle of the day.

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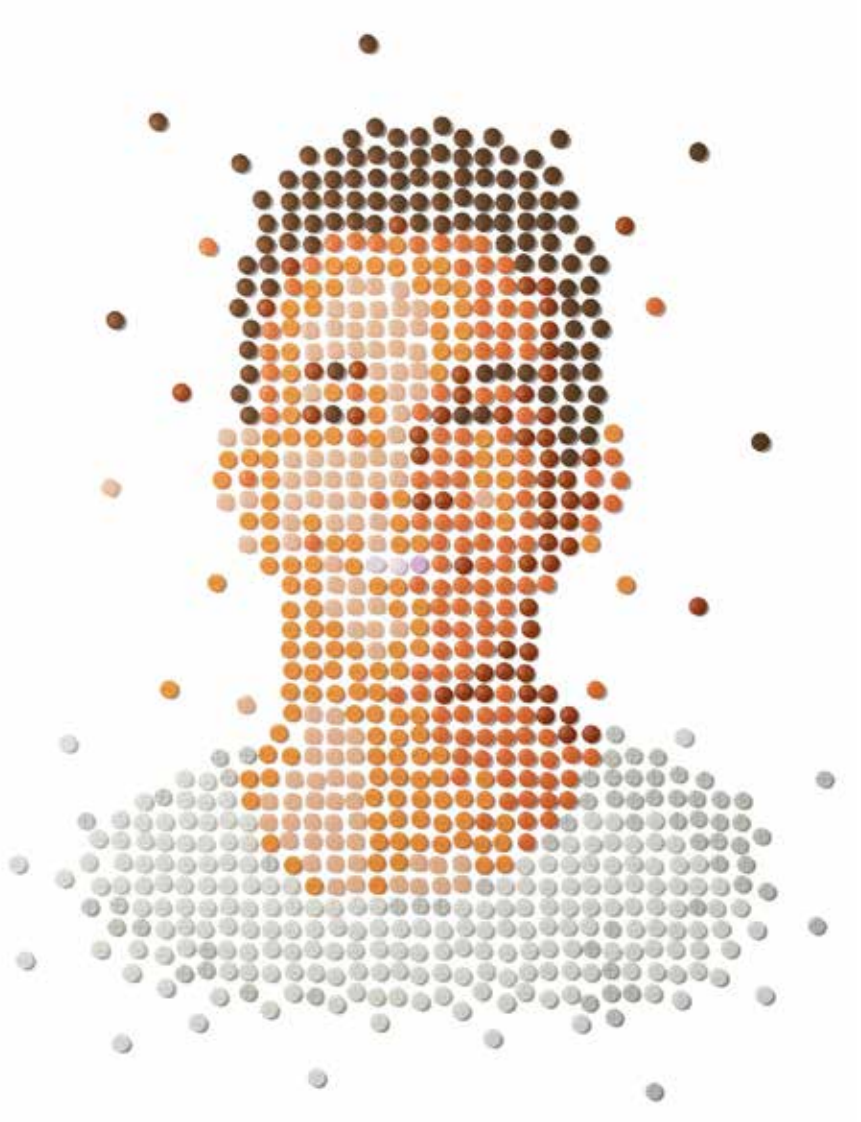
Though Swanson had welcomed that initial increase in the diagnosis rate, he expected it to plateau at 3 percent. Instead, it kept rising, hitting 5.5 percent of American children in 1997, then 6.6 percent in 2000. As time passed, Swanson began to grow uneasy. He and his colleagues were continuing to follow the almost 600 children in the M.T.A. study, and by the mid-2000s, they realized that the new data they were collecting was telling a different — and less hopeful — story than the one they initially report-

ed. It was still true that after 14 months of treatment, the children taking Ritalin behaved better than those in the other groups. But by 36 months, that advantage had faded completely, and children in every group, including the comparison group, displayed exactly the same level of symptoms. Swanson is now 80 and close to the end of his career, and when he talks about his life's work, he sounds troubled — not just about the M.T.A. results but about the state of the A.D.H.D. field in general. "There are things about the way we do this work," he told me, "that just are definitely wrong."

I've spent the last year speaking with some of the leading A.D.H.D. researchers in the United States and abroad, and many of them, like Swanson, express concern over what they see as a disconnect between the emerging scientific understanding of A.D.H.D. and the way the condition is being treated in clinics and doctors' offices. Edmund Sonuga-Barke, a researcher in psychiatry and neuroscience at King's College London, described the situation in personal terms. "I've invested 35 years of my life trying to identify the causes of A.D.H.D., and somehow we seem to be farther away from our goal than we were when we started," he told me. "We have a clinical definition of A.D.H.D. that is increasingly unanchored from what we're finding in our science."

Despite the questions these scientists have begun to raise, the growth of the diagnosis shows no signs of stopping or even slowing down. Last year, the Centers for Disease Control and Prevention reported that 11.4 percent of American children had been diagnosed with A.D.H.D., a record high. That figure includes 15.5 percent of American adolescents, 21 percent of 14-year-old boys and 23 percent of 17-year-old boys. Seven million American children have received an A.D.H.D. diagnosis, up from six million in 2016 and two million in the mid-1990s.

The preferred treatment for A.D.H.D. remains stimulant medications, including Ritalin and Adderall, and the market for those stimulants has expanded rapidly in recent years, in step with the growth of the diagnosis. From 2012 to 2022, the total number of prescriptions for stimulants to treat A.D.H.D. increased in the United States by 58 percent. Although the prescription rate is highest among boys ages 10 to 14, the real growth market today for stimulant medication is adults. In 2012, Americans in their 30s were issued five million prescriptions for stimulants to treat A.D.H.D.; a decade later, that figure had more than tripled, rising to 18 million.



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That ever-expanding mountain of pills rests on certain assumptions: that A.D.H.D. is a medical disorder that demands a medical solution; that it is caused by inherent deficits in children's brains; and that the medications we give them repair those deficits. Scientists who study A.D.H.D. are now challenging each one of those assumptions — and uncovering new evidence for the role of a child's environment in the progression of his symptoms. They don't question the very real problems that lead families to seek treatment for A.D.H.D., but many believe that our current approach isn't doing enough to help — and that we can do better. But first, they say, we need to rethink many of our old ideas about the disorder and begin looking at A.D.H.D. anew.

A.D.H.D. has always been a controversial diagnosis. Skeptics argue that many of the classic symptoms of the disorder — fidgeting, losing things, not following instructions — are simply typical, if annoying, behaviors of childhood. In response, others point to the serious consequences that can result when those symptoms grow more intense,

including school failure, social rejection and serious emotional distress.

So where do you draw the line? How do you tell a normally rambunctious kid from a child with A.D.H.D.? The tool that clinicians use to make that distinction is the Diagnostic and Statistical Manual of Mental Disorders, or D.S.M., which provides a checklist of symptoms to use in diagnosing patients, including nine potential symptoms for inattention and nine for hyperactivity/impulsivity. To qualify for the diagnosis, a child must display six symptoms from either category, of sufficient severity and level of impairment, for at least six months, starting before age 12, and those symptoms must be present in two different settings (like home and school).

That seems pretty scientific — six symptoms, two settings, six months, age 12 — and it reflects a longstanding effort by many in the field to portray A.D.H.D. as a straightforward medical condition with clear diagnostic boundaries. Russell Barkley, one of the most prominent A.D.H.D. researchers, has labeled the disorder "diabetes of the brain," and in a lecture that has been viewed more than four million times on YouTube, he says that like diabetes, A.D.H.D. is "a chronic disorder that must be managed every day to prevent the secondary harms it's going to cause." In a recent article in ADDitude, a popular magazine for families and patients, Wes Crenshaw, a psychologist who is listed as a member of the magazine's medical-review panel, drew the borders of A.D.H.D. even more sharply. "Your child either has A.D.H.D. or he does not," Crenshaw wrote. "If he does have it, he is either impaired, or not. And if he is impaired, talk therapy or supplements or nutrition or exercise or discipline isn't going to resolve that."

Now, however, some scientists have begun to argue that the traditional conception of A.D.H.D. as an unchanging, essential fact about you — something you simply have or don't have, something wired deep in your brain — is both inaccurate and unhelpful. According to Sonuga-Barke, the British researcher, the traditional notion that there is a natural category of "people with A.D.H.D." that clinicians can objectively measure and define "just doesn't seem to be the case."

Accurately diagnosing A.D.H.D. can be challenging, for a number of reasons. Unlike with diabetes, there is no reliable biological test for A.D.H.D. The diagnostic criteria in the D.S.M. often require subjective judgment, and historically those criteria have been quite fluid, shifting with each revision of the manual. The diagnosis encompasses a wide variety of behaviors. There are two main kinds of A.D.H.D., inattentive and hyperactive/impulsive, and children in one category often seem to have little in common with children in the other. There are people with A.D.H.D. whom you can't get to stop talking and others whom you can't get to start. Some are excessively eager and enthusiastic; others are irritable and moody.

A.D.H.D. is defined in the D.S.M. as a neurodevelopmental disorder, but the symptoms of A.D.H.D. can be produced by a variety of environmental causes as well. Difficulty sitting still and sustaining attention can also be symptoms of a serious head injury, fetal alcohol syndrome, childhood lead exposure, early trauma and more. There is also a high rate of overlap between the symptoms of A.D.H.D. and those of other psychiatric disorders, including depression, anxiety, dyslexia and autism. Although the D.S.M. specifies that clinicians shouldn't diagnose children with A.D.H.D. if their symptoms are better explained by another mental disorder, more than three quarters of children diagnosed with A.D.H.D. do have another mental-health condition as well, according to the C.D.C. More than a third have a diagnosis of anxiety, and a similar fraction have a diagnosed learning disorder. Forty-four percent have been diagnosed with a behavioral disorder like oppositional defiant disorder.

This all complicates the effort to portray A.D.H.D. as a distinct, unique biological disorder. Is a patient with six symptoms really that different from one with five? If a child who experienced early trauma now can't sit still or stay organized, should she be treated for A.D.H.D.? What about a child with an anxiety disorder who is constantly distracted by her worries? Does she have A.D.H.D., or just A.D.H.D.-like symptoms caused by her anxiety?

To try to clarify and better define the boundaries of A.D.H.D., researchers have long sought to identify a biological signature, or "biomarker," for the disorder — a clear test, like the blood-glucose test for diabetes, that would allow clinicians to say for sure which children have A.D.H.D. and which do not. And in the early years of the 21st century, it seemed as though they were on the verge of success.

In 2002, Russell Barkley, then a professor of psychiatry and neurology at the University of Massachusetts Medical School as well as the author of several popular books on A.D.H.D., drafted an "international consensus statement," signed by 85 prominent researchers, that defended the validity of the A.D.H.D. diagnosis. It leaned heavily on early studies that suggested that there were indeed solid biomarkers for the disorder, asserting, for instance, that people with A.D.H.D. had "less brain electrical activity" in certain regions than those without the diagnosis; that a single gene had been found to be associated with the disorder; and that people diagnosed with A.D.H.D. had "relatively smaller areas of brain matter."

In the years since the consensus statement was published, however, the evidence for each of these A.D.H.D. biomarkers has faltered. Attempts to replicate the studies that showed differences in brain electrical activity came up empty. And though scientists have identified complex collections of genes that together may be signs of greater risk for A.D.H.D., they have failed to find a specific gene that predicts the disorder. "There is no single-gene story," John Gabrieli, an M.I.T. neuroscientist, told me recently. "Fifteen years ago, there was incredible optimism, and now we realize how far away we are."

'There literally is no natural cutting point where you could say, "This person has got A.D.H.D., and this person hasn't got it."'

The most ambitious effort to find a biomarker for A.D.H.D. was run by the Enigma Consortium, a global network of scientists that shares brain-scan data from more than 4,000 subjects. Earlier studies had found indications of physi-

cal differences in the brains of patients diagnosed with A.D.H.D. — the "relatively smaller areas of brain matter" in Barkley's statement. But when a team led by Martine Hoogman, a Dutch neuroscientist, spent years comparing the "cortical volumes" of Enigma subjects diagnosed with A.D.H.D. with those of a control group, the results were once again disappointing. Among adults and adolescents, there was no difference at all between the two groups; among children, the differences were so minor as to be almost imperceptible. As Edmund Sonuga-Barke told me, "What Enigma showed is that what we thought was there isn't really there."

To the surprise of many, when Hoogman and her team published their results in 2017, they claimed that the data, in fact, showed the opposite, conclusively demonstrating the biological nature of A.D.H.D.: "We confirm, with high-powered analysis, that patients with A.D.H.D. have altered brains; therefore A.D.H.D. is a disorder of the brain," the researchers wrote. "This message is clear for clinicians to convey to parents and patients, which can help to reduce the stigma of A.D.H.D. and improve understanding of the disorder."

When I interviewed Hoogman by email recently, I was surprised to learn that she now wishes she could have revised that statement. "Back then, we emphasized the differences that we found (although small), but you can also conclude that the subcortical and cortical volumes of people with A.D.H.D. and those without A.D.H.D. are almost identical," she wrote. In retrospect, she added, it wasn't fitting to conclude from her findings that A.D.H.D. is a brain disorder. "The A.D.H.D. neurobiology is so much more complex than that."

Sonuga-Barke goes further, arguing that the entire decades-long quest for a biomarker has been "a red herring" for the field. He understands his colleagues' desire to find airtight evidence for the biological nature of A.D.H.D. that could help them defend the diagnosis against those who would dismiss it altogether. "In the field, we're so frightened that people will say it doesn't exist," he says. "That this is just bad parenting, from the right, or this is just a product of our postindustrial society, from the left. We have to double down because we're terrified of what will happen to the kids who can't get the meds. We've seen the impact they can have on people's lives."

But the reality, he says, is that "there literally is no natural cutting point where you could say, 'This person has got A.D.H.D., and this person hasn't got it.' Those decisions are to some extent arbitrary. That doesn't mean that the suffering associated with A.D.H.D. is imaginary, it just means it's on a continuum. And that is the conundrum — the empirical crisis — for A.D.H.D."

The failure to find a clear biomarker doesn't mean that there is no biological basis for A.D.H.D.; most scientists I spoke to agreed that the condition is produced by some combination of biological and environmental forces, though there is little consensus about the relative importance of each. But it does have certain implications for the field, including for the question of medication. If we're no longer confident that A.D.H.D. has a purely biological basis, does it make sense that our go-to treatment is still rooted in biology?

The roots of the current treatment model go back to 1937, when a Harvard-trained psychiatrist named Charles Bradley published (in the dramatically named American Journal of Insanity) the results of an experiment in the clinic he ran for children with behavioral problems in East Providence, R.I. For one week, Bradley gave 30 of his young patients a daily dose of benzedrine, an amphetamine then popular among jazz musicians and college students. Fourteen of the children responded in what Bradley described as "a spectacular fashion." From the day of their first dose, their teachers reported "remarkably improved school performance." Overnight, the students seemed, for the first time, interested in their schoolwork. They became more "placid and easygoing," and they spontaneously made comments to their teachers like "I feel fine and can't seem to do things fast enough today" and "I start to make my bed, and before I know it, it is done."

Nearly ninety years later, the treatment of A.D.H.D. hasn't moved very far beyond Bradley's discovery. Adderall, now the leading treatment for the disorder, is a type of amphetamine, just like the benzedrine pills that Bradley administered to his patients; the other leading prescription stimulants, including Ritalin, are all variations on the same chemical compound.

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F. Xavier Castellanos, a neuroscience researcher at New York University, occasionally goes back and reads Bradley's original paper, and he told me that today, when his A.D.H.D. patients take stimulant medication for the first time, he often sees the same effect that Bradley observed back in the 1930s. "The first dose is almost like a mystical experience," Castellanos said. "You see this transformation. The behavioral benefits are really sort of stunning, especially in younger kids."

But like James Swanson, the researcher who helped lead the M.T.A. study, Castellanos has some real concerns about stimulant treatment for A.D.H.D. He says he is frustrated by a persistent finding in the research: While the medications can have a powerful effect on how children behave in the classroom, they do little to improve how they learn. "It's a puzzle," Castellanos says. "There's a real disconnect between the almost awesome effects on behavior and the minimal effects on academic achievement or attainment. What bothers me is that the kids do more seatwork — you can see that they've done more problems — but then when you test them a week or two later, their scores barely budge. Or they don't budge at all. That's the thing that really frustrates me."

This effect has turned up in a number of studies over the years, but there are two relatively recent ones that illustrate it well. One was published in 2023 by Elizabeth Bowman, an Australian neuroscientist, and David Coghill, a British psychiatrist. They recruited 40 young adults in Australia, gave some of them stimulant A.D.H.D. medications and others a placebo and then asked them to solve a series of complex tests called

knapsack-optimization problems. Knapsack problems are well-known puzzles in economics and computer science. You're given a virtual backpack and a series of items of different weights and prices, and you need to figure out the assortment of items that will maximize the dollar value of your load.

The subjects who were given stimulants worked more quickly and intensely than the ones who took the placebo. They dutifully packed and repacked their virtual backpacks, pulling items in and out, trying various combinations. In the end, though, their scores on the knapsack test were no better than the placebo group. The reason? Their strategies for choosing items became significantly worse under the medication. Their choices didn't make much sense — they just kept pulling random items in and out of the backpack. To an observer, they appeared to be focused, well behaved, on task. But in fact, they weren't accomplishing anything of much value.

A Florida researcher named William Pelham Jr. found something similar in a study published in 2022. Unlike the Australian study, this one involved not adults but children ages 7 to 12, all attending an eight-week summer camp for kids with A.D.H.D. Their days were split between classroom learning and regular camp activities. Pelham and his colleagues randomly divided the children into a treatment group and a control group. The treatment group got a regular daily dose of the active ingredient in Ritalin, and the control group was given a placebo.

As with the Australian study, the children taking Ritalin worked faster and behaved better in the classroom than those in the placebo group. But again, they didn't learn any more than the control group. "Although it has been believed for decades that medication effects on academic seatwork productivity and classroom behavior would translate into improved learning of new academic material," the scientists wrote, "we found no such translation."

So what's going on? If these studies are accurate, stimulant medications don't do much to improve cognitive ability or academic performance. And yet millions of young Americans (and their parents) feel that the pills are essential to their success in school. Why?

One possible explanation can be found in the work of Martha Farah, a cognitive neuroscientist at the University of Pennsylvania. In one study, she and a colleague, Irena Ilieva, recruited 46 young adults, gave half of them a dose of Adderall and half a placebo and then had them perform 13 different cognitive tests. The ones who took the medication didn't do better on any of the tests than the ones who took the placebo, but when the researchers asked the subjects to evaluate their performance on the assessments, the ones who took Adderall believed they had done better. They felt more confident, even if their actual abilities didn't improve.

Farah directed me to the work of Scott Vrecko, a sociologist who conducted a series of interviews with students at an American university who used stimulant medication without a prescription. He wrote that the students he interviewed would often "frame the functional benefits of stimulants in cognitive-sounding terms." But when he dug a little deeper, he found that the students tended to talk about their attention struggles, and the benefits they experienced with medication, in emotional terms rather than intellectual ones. Without the pills, they said, they just didn't feel interested in the assignments they were supposed to be doing. They didn't feel motivated. It all seemed pointless.

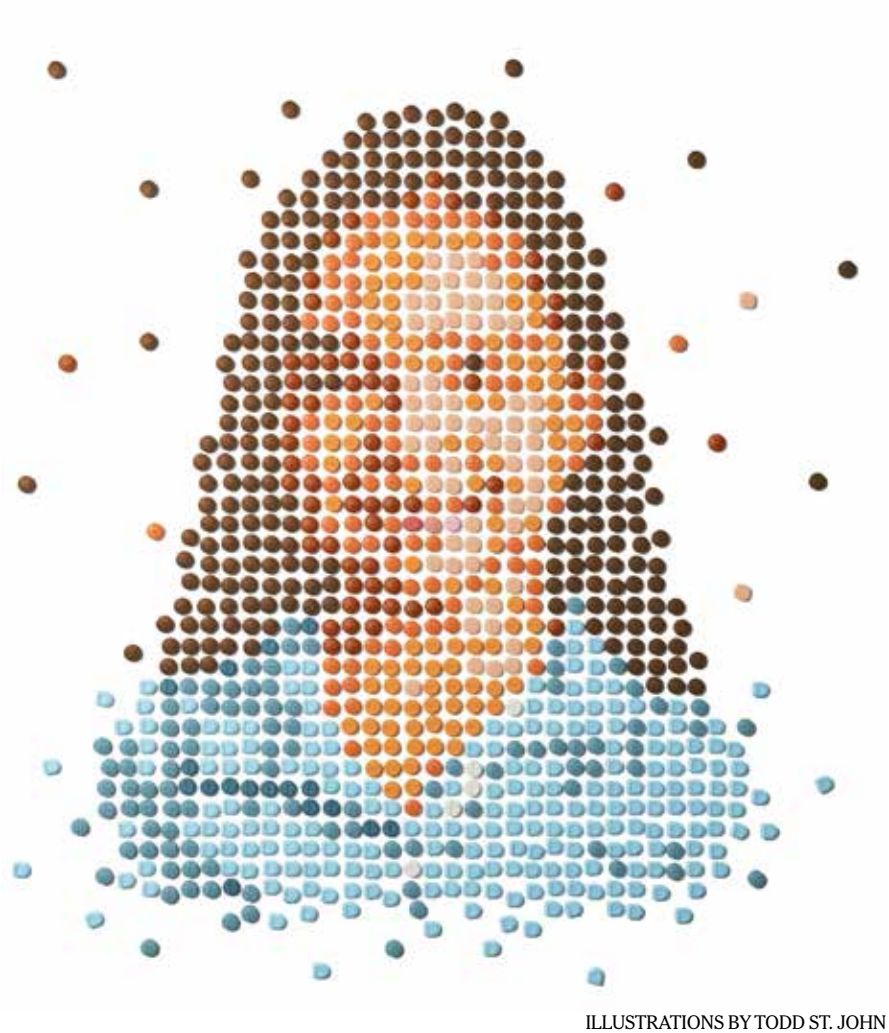
On stimulant medication, those emotions flipped. "You start to feel such a connection to what you're working on," one undergraduate told Vrecko. "It's almost like you fall in love with it." As another student put it: On Adderall, "you're interested in what you're doing, even if it's boring."

Historically, this is one of the main reasons people have taken amphetamines: They make tedious tasks seem more interesting. During World War II, the American military distributed tens of millions of amphetamine tablets to enlisted men for use during the many boring stretches of war. The pills were given to Air Force pilots flying long missions and to Navy sailors who had to keep watch all night. In the 1950s, suburban housewives took amphetamines to get through the boredom of endless days of housework and child care. Long-distance truckers have for decades used them to tolerate the tedium of the road. For the college students Scott Vrecko interviewed, term papers were just as boring as laundry or a long-haul truck route — but they became more bearable with the help of stimulants.

'There is no long-term effect. The only long-term effect that I know of has been the suppression of growth.''

The original M.T.A. study, like the later knapsack-problem study and summer-camp study, showed a strong effect of stimulant medication on behavior and next to no impact on academic achievement. Back in the early 2000s, Swanson was troubled by those results, but the bigger issue, for him, was the fact that even the behavioral benefits under stimulants faded out so completely. He and his colleagues spent much of that decade analyzing and reanalyzing the M.T.A. data, and they kept coming back with the same result: After the first year of treatment, the relative positive effects of Ritalin on behavior started to shrink, and by the end of the third year, they had disappeared altogether.

There was another distressing result they noticed in their data — a physiological one. The children who took Ritalin for an extended period grew less quickly than the nonmedicated children did. By the end of those 36 months, subjects who had consistently taken stimulant medication were, on average, more than an inch shorter than the ones who had never received medication. Many of the scientists in the M.T.A. group assumed that this height suppression in childhood would be temporary — that the shorter children would catch up during adolescence — but when data was collected again nine years after the initial experiment, the height gap remained. In 2017, Swanson and the M.T.A. group published yet another follow-up, this time tracking the subjects until age 25. The ones who had consistently taken stimulant medication remained about an inch shorter than their peers. Their A.D.H.D. symptoms, meanwhile, were no better than those who had stopped taking the medication or who had never started.



ILLUSTRATIONS BY TODD ST. JOHN

Researchers acknowledge that there are other risks inherent in taking prescription stimulants. Amphetamines can be powerfully addictive, and last year, a study in The American Journal of Psychiatry found that even a medium-strength daily dose of Adderall more than tripled a patient's likelihood of developing psychosis or mania. A high dose increased the risk by a factor of five. Still, for most scientists, including Castellanos, Sonuga-Barke and Gabrieli, the positives of medication outweigh the negatives. As Gabrieli put it, "I feel the bigger risk is people not getting help who are struggling in everyday life."

Compared with other psychiatric medications, Gabrieli explained, Ritalin and Adderall (and the many similar formulations on the market today) are relatively safe and effective. They don't help everyone, but in the short term, at least, they provide significant symptom control in most of the children who take them. Clinicians generally consider them easy to prescribe, in part because they're usually easy for patients to quit. Unlike antidepressants or many anti-anxiety medications, they don't linger in the bloodstream for more than a day, which means that even with the extended-release versions, they don't require a weaning process. You can just stop taking them. "At some level," Gabrieli told me, "these stimulants are not that far from Red Bull."

After three decades of studying stimulants, Swanson differs with many of his colleagues on their value. "I don't agree with people who say that stimulant treatment is good," he told me. "It's not good." He acknowledges that medication can often produce short-term improvements in children's behavior. But, he says, "there is no long-term effect. The only long-term effect that I know of has been the suppression of growth. If you're honest, you should tell kids that, look, if you're interested in next week or next month or even the next year, this is the right treatment for you. But in the long run, you're going to be shorter. How many kids would agree to take medication? Probably none."

When I spoke to students across the country about their experiences with A.D.H.D. medication, their relationship to stimulants often turned out to be quite complex. Cap, a suburban teenager on the East Coast, told me that he began taking Ritalin the summer after his sophomore year of high school. In the affluent neighborhood where he grew up, SAT prep was an important rite of passage, and that summer, his parents enrolled him in a prep course at a local tutoring center. Cap (a nickname) told me that he found studying for the SAT to be "very boring" and that every time he went in for tutoring, he felt unable to concentrate.

Then he was prescribed Ritalin, and his experience of test prep changed. "I used to hate doing the SAT reading," Cap said. "But if I took the medication, I could read through it all and, like, comprehend it really well. I would actually enjoy reading it."

Cap was on the varsity baseball team, and one day that fall, he went to batting practice right after one of his SAT tutoring sessions. To his surprise, he found that the medication helped with his hitting too. "I was so focused," he said. "My contact rate was higher. I could see the ball better coming out of the machine." Cap had always been a very social guy, and baseball practice was usually a time to chat and joke with his teammates. But not when he took Ritalin. "When I'm on the medication, I don't get as distracted socially," he told me. "It feels like you're a horse with blinders on. You're just focused on your goal. There's nothing else going on in your head."

Though he found Ritalin effective — both for baseball and test prep — he didn't like it. "Honestly, I pretty much hated taking it," he told me. "But I knew I needed to for my SAT." He described for me the daily ups and downs of the medication, which affected not only his mood but also his appetite. "When I take it for studying, it does feel like I'm getting an adrenaline rush," he said. "I

feel happy. When it peaks, you feel good about yourself. You're studying, you're locked in. But then once it wears off, you just feel awful."

John, a teammate of Cap's, was first prescribed Adderall to treat A.D.H.D. in eighth grade. He told me it helped him get through his classes, especially English, which he never liked much. "It would make it so that if I tried to pay attention, I would be able," he told me. "It would still be very boring, but I was able to finish books and pay attention to what was happening."

Socially, though, there was a price. "Around my friends, I'm usually the most social, but when I'm on it, it feels like my spark is kind of gone," John said. "I laugh a lot less. I can't think of anything to say. Life is just less fun. It's not like I'm sad; I'm just not as happy. It flattens things out."

It was late summer when John and I spoke, and he was preparing to head off to his freshman year of college. I asked him if he was planning to take A.D.H.D. medication once he got there. He said yes, probably. He wasn't crazy about the idea, but for him it felt like a trade-off that would be worth it in the end. "I kind of feel like it's just a sacrifice I'm going to have to make," he told me.

'If I don't have to do any work, then I'm just a completely regular person.'

For other teenagers, the negatives of stimulants outweigh the positives, and they lobby their parents to stop taking the medication — or they just quit on their own. Statistically, most adolescents don't stay on stimulants for more than a year. To Swanson, the high quit rate is further evidence that over the long term, the medications just aren't that good. "If it's so effective, why do people stop?" he asked. "The physicians say, 'They stop because they don't know what's good for them.' So the parents hear the message: 'If you don't fill this prescription, you just don't know what's good for your kid.' But if you ask the kids themselves, they say, 'It makes me feel bad.' Or, 'It didn't help me.' Or, 'It stopped working.' Who do you believe?"

A significant part of the A.D.H.D. establishment does, in fact, promote the message that children and adolescents who resist medication don't know what's good for them. You encounter this point of view often when you read ADDitude magazine, which is owned by the online publisher WebMD. The headline on one recent story read: "Half of College Kids Stop Taking Their A.D.H.D. Medication. Make Sure Your Teen Isn't One of Them." Another article, by Wes Crenshaw, advised parents to "problematize" their children's A.D.H.D. in order to encourage them to take their medication. "To accept treatment, teens need to feel A.D.H.D. as problematic, as a pain in their life that limits and controls them," Crenshaw wrote. "Too many parents normalize their children's struggles to make them feel better."

A third article, by Roberto Olivardia, a clinical psychologist who lectures at Harvard Medical School, gave advice to clinicians on how to respond if parents say they are worried that stimulant medication is muting their child's sense of humor. The suggested response: Maybe your child was the wrong kind of funny. "Parents should know that not all personality changes sparked by medication are negative," Olivardia advised. "If a child known for his sense of humor seems 'less funny' on medication, it could be that the medication is properly inhibiting them. In other words, it's not that the child is less funny; it's that they're more appropriately funny at the right times."

Cap's parents did encourage him to take his Ritalin every day during high school, but in reality, he told me, he used it much more situationally. By the end of senior year, he was taking medication for baseball games more often than he was taking it to study, and on weekends and in the summer, he rarely took it.

John also generally doesn't take his Adderall during the summer. When he's not in school, he told me, he doesn't have any A.D.H.D. symptoms at all. "If I don't have to do any work, then I'm just a completely regular person," he said. "But once I have to focus on things, then I have to take it, or else I just won't get any of my stuff done."

John's sense that his A.D.H.D. is situational — that he has it in some circumstances but not in others — is a challenge to some of psychiatry's longstanding assumptions about the condition. After all, diabetes doesn't go away over summer vacation. But John's intuition is supported by scientific evidence. Increasingly, research suggests that for many people A.D.H.D. might be thought of as a condition they experience, sometimes temporarily, rather than a disorder that they have in some unchanging way.

Last October, the M.T.A. group published a new study that explored how A.D.H.D. symptoms in M.T.A. participants changed over the course of their childhood and young adulthood. In contrast to the categorical model of A.D.H.D. — you either have it or you don't — the researchers showed that for most subjects, their symptoms and level of impairment in fact fluctuated over the years, often quite substantially. Only about 11 percent of the children who entered the study with an A.D.H.D. diagnosis experienced the symptoms consistently year after year. More often, their symptoms would come and go; for a few years, they might stay above the D.S.M.'s symptom threshold, and then for a few years, their symptom count might dip below the cutoff, sometimes disappearing altogether,

When I spoke to Margaret Sibley, the lead author on the fluctuation study and a clinical psychologist and professor at the University of Washington School of Medicine, she pointed out for me a curious finding from an earlier M.T.A. paper: Not only did most of the A.D.H.D. subjects improve, at least temporarily, but 40 percent of the children in the comparison group — who were originally selected for the study specifically because they didn't have A.D.H.D. — at some point in adolescence had enough symptoms to qualify for an A.D.H.D. diagnosis.

"Rather than trying to treat and resolve the biology, we should be focusing on building environments that improve outcomes and mental health."

Sibley told me she didn't believe those children somehow suddenly contract-

ed A.D.H.D. as adolescents. Instead, she said, their circumstances — their environment — might have changed, and that shift in environment might have increased their symptoms. Sibley said it was important to remember that many of the symptoms of A.D.H.D. are actually pretty commonplace; at any given moment, she explained, the average American adult has two or three of them — halfway to an official diagnosis. "This isn't something that you either have nothing or you have it all," she said. "That's part of why this is a gray disorder, when it's not on its extremes."

Those extremes, however, are very important. Conceiving of A.D.H.D. as a yes-or-no, black-or-white diagnosis, the way the profession has often done, has obscured the fact that certain children with A.D.H.D. symptoms are at much greater risk than others. Joel Nigg, a clinical psychologist at Oregon Health & Science University, has identified a number of different subtypes within the A.D.H.D. population. One group of children, those whose A.D.H.D. symptoms are accompanied by intense anger, are at much higher risk of negative outcomes than those with A.D.H.D. symptoms alone. Their early symptoms, Nigg found, are often the beginning of a diagnostic cascade that leads to real problems in adolescence and adulthood, including school dropout, criminal behavior and elevated risk of serious injury or early death. Those patients, representing about a third of children diagnosed with A.D.H.D., need early attention and comprehensive treatment — most likely including medication but often going well beyond it.

Nigg suspects that those high-risk children do have significant biological differences from typical children, and he thinks that those differences may eventually turn up on genetic tests or brain scans as those technologies continue to improve. But for a significant percentage of people diagnosed with A.D.H.D., Nigg says, "there's nothing neurobiologically notable about them. Instead, their symptoms are situational or conditional. They may have had a hard life, or they have a lack of social support, or they're in the wrong niche in life."

If their problems are rooted in their environment as much as in their brain chemistry, Nigg believes, then perhaps their treatment can be based in their environment as well. Sonuga-Barke agrees. For most of his career, he embraced what he now calls the "medical model" of A.D.H.D — the belief that the brains of people with A.D.H.D. are biologically deficient, categorically different from those of typical, healthy individuals. Now, however, Sonuga-Barke is proposing an alternative model, one that largely sidesteps questions of biology. What matters instead, he says, is the distress children feel as they try to make their way in the world.

Sonuga-Barke's proposed model locates A.D.H.D. symptoms on a continuum, rather than presenting the condition as a distinct, natural category. And it departs from the medical model in another crucial way: It considers those symptoms not as indications of neurological deficits but as signals of a misalignment between a child's biological makeup and the environment in which they are trying to function. "I'm not saying it's not biological," he says. "I'm just saying I don't think that's the right target. Rather than trying to treat and resolve the biology, we should be focusing on building environments that improve outcomes and mental health."

Can changing a person's environment really alter their symptoms? In 2016, Arielle Lasky and members of the M.T.A. research group published a paper that suggested that for many young people, the answer is yes. At that point in the history of the study, the subjects were adults in their mid-20s, able to speak for themselves. So rather than simply collecting data on their symptoms or their height, the scientists asked them questions. They conducted long interviews with 125 of these young adults, all of whom were diagnosed with A.D.H.D. as children.

What the researchers noticed was that their subjects weren't particularly interested in talking about the specifics of their disorder. Instead, they wanted to talk about the context in which they were now living and how that context had affected their symptoms. Subject after subject spontaneously brought up the importance of finding their "niche," or the right "fit," in school or in the workplace. As adults, they had more freedom than they did as children to control the parameters of their lives — whether to go to college, what to study, what kind of career to pursue. Many of them had sensibly chosen contexts that were a better match for their personalities than what they experienced in school, and as a result, they reported that their A.D.H.D. symptoms had essentially disappeared. In fact, some of them were questioning whether they had ever had a disorder at all — or if they had just been in the wrong environment as children.

'Characterizing A.D.H.D. as a personality trait rather than a disorder, they saw themselves as different rather than defective.'

The work environments where the subjects were thriving varied. For some, the appeal of their new jobs was that they were busy and cognitively demanding, requiring constant multitasking. For others, the right context was physical, hands-on labor. For all of them, what made a difference was having work that to them felt "intrinsically interesting."

One subject, who was studying film in college, said that his ability to thrive in his chosen field made him question the years he spent being treated for A.D.H.D. "Originally, when I was first diagnosed with it, it was explained to me as attention deficit, just a lack of attention," he said. "An ability not to have an attention span for very long. But I can have an attention span for extremely long for the things that I care about." The film student reflected on his earlier struggles. "Public education, you're forced into it," he said. "Maybe that's why I didn't pay attention that much. But now I'm in college, in a subject that I want to be a part of, so me having a lack of attention, it hardly ever happens anymore, because I'm not usually where I don't want to be."

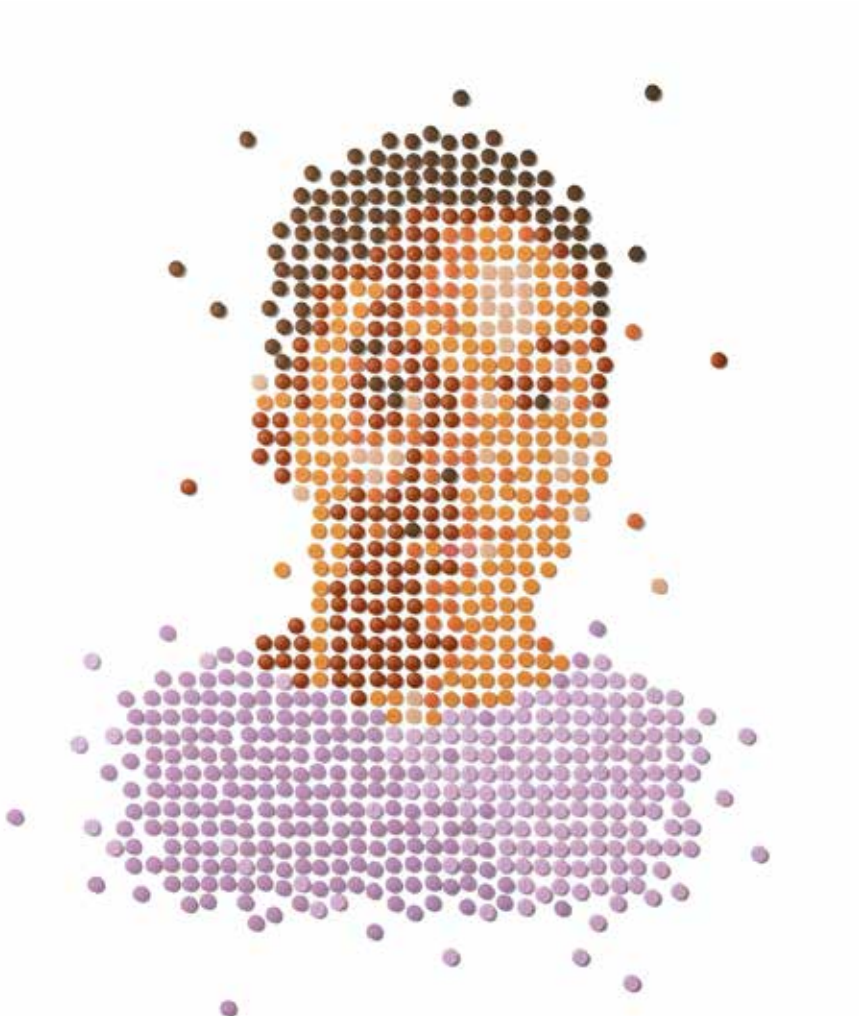
A hairstylist told the researchers that her inability to concentrate in school vanished when she began studying hair. "If you sit up there and give me a lecture on a haircut, I will remember everything you said, word for word," she said. "Stuff that I'm into, I am so immersed in it. But in school, it was awful."

A young man who was training to be an auto technician said that in his new career, his A.D.H.D. was no longer an issue. “It’s just that I had to figure out what I wanted to do,” he explained. “I want to work with cars. I don’t get bored doing that.” If people with A.D.H.D. are directed into areas where their strengths and interests lie, he went on, “I’m pretty sure that they can naturally just go about dealing with it, instead of having to give people medications.”

Margaret Sibley’s recent fluctuations paper provided some additional clues into what might help adolescents and young adults feel better and function better. To Sibley’s surprise, patients’ symptoms tended to improve, rather than worsen, during times of higher “environmental demands” — periods of more responsibility and busier schedules. For many of the young men and women in the “niche” study, the same phenomenon held: Jobs or college courses that were demanding and interesting helped alleviate their symptoms. And as their symptoms lifted, they changed the way they thought about themselves.

“Rather than a static ‘attention deficit’ that appeared under all circumstances,” the M.T.A. researchers wrote, “our subjects described their propensity toward distraction as contextual. … Believing the problem lay in their environments rather than solely in themselves helped individuals allay feelings of inadequacy: Characterizing A.D.H.D. as a personality trait rather than a disorder, they saw themselves as different rather than defective.”

Seen through this lens, the problem for John and Cap and many other adolescents becomes a much more mundane one than a brain disorder. Their problem is the simple fact that high school can be really boring, and without medication, they have a low tolerance for boring stuff. For some children, a different school, or a different kind of school, might produce the same profound shift that the M.T.A. subjects experienced when they enrolled in film school or began studying hair styling. For others, a prescription for Ritalin or Adderall might help make school feel like a better fit. But for them and their parents, the experience of taking medication might feel quite different if it was presented to them not as a medicine to fix their defective brain but as a tool to make an inhospitable environment more tolerable.



ILLUSTRATIONS BY TODD ST. JOHN

When Edmund Sonuga-Barke thinks about the way attention issues can evolve and change over the course of a person’s lifetime, he often thinks about his own story. Growing up in the 1960s in Derby, a depressed industrial city in the English Midlands, he was unable to sit still in class. At 8, he was assessed with hyperkinesis and minimal brain dysfunction, the terms used at the time for what we now call A.D.H.D. He received no treatment or medication; instead, he was relegated to the remedial class. The only relief he found from the shame and boredom of school was with his friends, a pack of rough and ready young rebels who as teenagers bonded over their love of punk rock.

Then, through a series of miraculous interventions, Sonuga-Barke was given the chance to go to college — Bangor University in Wales — on a scholarship. When he moved to the Welsh countryside, he suddenly found himself in an environment where things were very different from everything he had known growing up.

For the young adults in the “niche” study who were interviewed about their work lives, the transition that helped them overcome their A.D.H.D. symptoms often was leaving academic work for something more kinetic. For Sonuga-Barke, it was the opposite. At university, he would show up at the library at 9 every morning and sit in his carrel working until 5. The next day, he would do it again. Growing up, he says, he had a natural tendency to “hyperfocus,” and back at school in Derby, that tendency looked to his teachers like daydreaming. At university, it became his secret weapon.

“I think my brain had probably matured to the point where I had a capacity

I didn't have earlier on," Sonuga-Barke told me. "At the same time, suddenly I was in a context where my natural way of thinking was valuable. The combination of those two was revelatory to me." He graduated from Bangor, collected a master's degree and a Ph.D. and then went on to positions of academic prestige, including being elected to the Academy of Medical Sciences and named editor in chief of The Journal of Child Psychology and Psychiatry.

I asked Sonuga-Barke what he might have gained if he grew up in a different time and place — if he was prescribed Ritalin or Adderall at age 8 instead of just being packed off to the remedial class.

"I don't think I would have gained anything," he said. "I think without medication, you learn alternative ways of dealing with stuff. In my particular case, there are a lot of characteristics that have helped me. My mind is constantly churning away, thinking of things. I never relax. The way I motivate myself is to turn everything into a problem and to try and solve the problem."

Sonuga-Barke says he has known plenty of young people, including some in his own family, who have benefited from taking stimulant medication. He just doesn't think it's accurate — or helpful — to think of Adderall or Ritalin as a medical solution to a medical disorder.

"The simple model has always been, basically, 'A.D.H.D. plus medication equals no A.D.H.D.,'" he says. "But that's not true. Medication is not a silver bullet. It never will be." What medication can sometimes do, he believes, is allow families more room to communicate. "At its best," he says, "medication can provide a window for parents to engage with their kids," by moderating children's behavior, at least temporarily, so that family life can become more than just endless fights about overdue homework and lost lunchboxes. "If you have a more positive relationship with your child, they're going to have a better outcome. Not for their A.D.H.D. — it's probably going to be just the same. But in terms of dealing with the self-hatred and low self-esteem that often goes along with A.D.H.D."

That might sound a little mushy — that the point of A.D.H.D. treatment is to help you build relationships and improve your self-esteem, rather than the more scientific-sounding goal of repairing your malfunctioning brain. But think back to that controversial statement in Martine Hoogman's 2017 paper. She wrote that it was important to interpret the Enigma data as confirmation that A.D.H.D. patients "have altered brains," because that biological explanation would "help to reduce the stigma of A.D.H.D." But does portraying A.D.H.D. as a "disorder of the brain" actually reduce its stigma? Might it not, in fact, increase a young person's sense of shame and isolation to be told they have a brain disorder?

'The diagnosis can create an identity that enhances prejudice and judgment which are associated with even greater feelings of isolation, exclusion and shame.'

An Australian psychologist named Luise Kazda has studied this very question. In a 2021 review paper, she and her colleagues found 14 studies in which receiving an A.D.H.D. diagnosis created a sense of "empowerment" by "supporting a sense of legitimacy accompanied by understanding and sympathy as well as decreased guilt, blame and anger." But in 22 other studies, Kazda wrote, "a biomedical view of difficulties was shown to be associated with disempowerment. By providing an excuse for problems, a decrease in responsibility by all involved can occur, often followed by inaction and stagnation." An additional 14 studies found that the diagnosis increased feelings of stigmatization. "The diagnosis can create an identity that enhances prejudice and judgment," Kazda reported, "which are associated with even greater feelings of isolation, exclusion and shame."

It's still not entirely clear why the simple act of providing a diagnosis of A.D.H.D. seems to have such profound effects on some children and their families. But it is certainly true that under the medical model, a diagnosis sends a very different message than it does under a model like Sonuga-Barke's, which sees a person's A.D.H.D. symptoms as, at least in part, the product of a mismatch with a particular environment.

For some parents, it may indeed be less stigmatizing, and more comfortable, to be able to say, "My child has A.D.H.D., a medical condition, so he needs to take this medicine every day," rather than, "I want my kid to succeed in environments for which he's not well suited, so therefore I want him to take these pills." For many children, however, a diagnosis of A.D.H.D. that is communicated via the dominant medical model can feel like more than a stigma; it can feel like a life sentence. The message to children is often that A.D.H.D. is a binary, biological category, and if your symptoms place you in that category, your brain has a deficit, and you have a disorder.

The alternative model, by contrast, tells a child a very different story: that his A.D.H.D. symptoms exist on a continuum, one on which we all find ourselves; that he may be experiencing those symptoms as much because of where he is as because of who he is; and that next year, if things change in his surroundings, those symptoms might change as well. Armed with that understanding, he and his family can decide whether medication makes sense — whether for him, the benefits are likely to outweigh the drawbacks. At the same time, they can consider whether there are changes in his situation, at school or at home, that might help alleviate his symptoms. If he is also experiencing other psychological conditions — anxiety or depression or post-traumatic stress — they can take steps to address those deeper issues, independent of his inability to focus in math class.

Admittedly, that version of A.D.H.D. has certain drawbacks. It denies parents the clear, definitive explanation for their children's problems that can come as such a relief, especially after months or years of frustration and uncertainty. It often requires a lot of flexibility and experimentation on the part of patients, families and doctors. But it has two important advantages as well: First, the new model more accurately reflects the latest scientific understanding of A.D.H.D. And second, it gives children a vision of their future in which things might actually improve — not because their brains are chemically refashioned in a way that makes them better able to fit into the world, but because they find a way to make the world fit better around their complicated and distinctive brains.