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Autism Agenda

Linn Benton Lincoln ESD-Cascade Regional Autism Program

What is the latest research on autism?

Doctors have defined autism spectrum disorder (ASD) as a neurobiological developmental condition that can impact communication, sensory processing, and social interactions. Although recent research has advanced the understanding of autism, there is much more to learn about the factors that influence this neurotype.

As of March 26, 2021, the Centers for Disease Control and Prevention (CDC) report that among 8-year-old children, one in 54Trusted Source are autistic. This number has increased from the one in 59 prevalence reported in previous estimates.

With autism rates on the increase, the scientific community has become all the more interested in uncovering the factors linked with autism.

Some scientists speculate that gene variantsTrusted Source cause autism, while others believe environmental factorsTrusted Source, such as exposure to toxinsTrusted Source, contribute to this neurotype. Still others theorize imbalances in the intestinal microbiomeTrusted Source may be at play. The latest autism research includes investigations into factors associated with this neurotype, as well as genetic variants, gut biome imbalances, and neurological factors that may contribute to it.

In this Special Feature, *Medical News Today* examines the latest scientific discoveries and what researchers have learned about autism.

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THE LATEST REASEARCH ON AUTISM CONTINUED….

Exploring contributing factors

A multiyear study funded by the CDC is underway to learn more about factors potentially linked to autism.

The Study to Explore Early DevelopmentTrusted Source is a collaboration between six study sites in the United States. These sites are part of the Autism and Developmental Disabilities Research and Epidemiology network and focus on children aged 2–5 years.

One of the goals of the study is discovering what health conditions occur in autistic and neurotypical children and what factors are associated with the likelihood of developing ASD.

Another objective of the study is to differentiate the physical and behavioral characteristics of autistic children, children with other developmental conditions, and those without these conditions.

This ongoing research has already produced several published studies. The latest found an association between ASD and a mother's exposure to ozone pollution during the third trimester of pregnancy.

Researchers also found that exposure to another type of air pollution called particulate matter during an infant's first year also increased the likelihood of the infant later receiving a diagnosis of ASD.

Current research on genes

This research appears in the journal *Epidemiology*Trusted Source.
Other avenues of research on autism include investigations into gene variants that could play a role in the development of ASD.

A recent study analyzed the DNA of more than 35,584 people worldwide, including 11,986 autistic individuals. The scientists identified variants in 102 genesTrusted Source linked with an increased probability of developing ASD. The researchers also discovered that 53 of the genes identified were mostly associated with autism and not other developmental conditions.

Expanding the research further, the team found that autistic people who carried the ASD-specific gene variants Trusted Source showed increased intellectual function compared with autistic individuals who did not have the variants.

The gene variants the scientists identified mainly reside in the cerebral cortex, which is responsible for complex behaviors.

These variants may play a role in how the brain neurons connect and also help turn other genes on or off — a possible factor that may contribute to autism.

Investigating neurological factors

Biological research has unearthed some interesting findings linking certain types of cell malfunctions to ASD.

Scientists at the Lieber Institute for Brain Development in Baltimore, MD, discovered a decrease in the integrity of myelin, a protective sheath surrounding nerve cells in the brain, in mice with a syndromic form of ASD. …….

Please continue reading the rest of this article at https://www.medicalnewstoday.com/articles/what-is-the-latest-research-on-autism

Gut-brain connection in autism

Research in mice identifies possible mechanism linking autism, intestinal inflammation

Many people with autism spectrum disorders also experience unusual gastrointestinal inflammation, but thus far scientists have not established whether and how those conditions might be linked.

Now, Harvard Medical School and MIT researchers, working with mouse models, may have found the missing link: Infections during pregnancy can lead to high levels of the inflammatory signaling molecule interleukin-17a (IL-17a), which can not only affect brain development in the fetus, but also alter the maternal microbiome in a way that primes the newborn's immune system for future inflammatory attacks.

In four studies beginning in 2016, study co-senior authors Gloria Choi of MIT and Jun Huh of Harvard Medical School have traced how elevated IL-17a during pregnancy acts on neural receptors in a specific region of the fetal brain to alter circuit development, leading to autism-like behavioral symptoms in mouse models.

The new research findings, which were described in the journal Immunity last month, show how IL-17a can also act to alter the trajectory of offspring's immune system development.

"We've shown that IL-17a acting on the fetal brain can induce autism-like behavioral phenotypes such as social deficits," said Choi, the Mark Hyman Jr. Career Development Associate Professor in the Picower Institute for Learning and Memory and the Department of Brain and Cognitive Sciences at MIT. "Now we are showing that the same IL-17a in mothers, through changes in the microbiome community, produces comorbid symptoms in the offspring, specifically a primed immune system."

The researchers caution that while the study findings are yet to be confirmed in humans, they do offer a hint that central nervous and immune system problems in individuals with autism-spectrum disorders share an environmental driver: maternal infection during pregnancy.

"There has been no mechanistic understanding of why patients with a neurodevelopmental disorder have dysregulated immune system," said Huh, an associate professor of immunology at the Blavatnik Institute at Harvard Medical School. "With the new findings, we've tied these fragmented links together. It may be that the reason is that they were exposed to this increase in inflammation during pregnancy."

TRACKING TIMING

First, the research team set out to confirm that maternal immune activation (MIA) leads to enhanced susceptibility to intestinal inflammation in their offspring. To do so, they injected pregnant mice with poly (I:C), a substance that mimics viral infection. As expected, their offspring exhibited autism-like behavioral symptoms as well as gut inflammation when exposed to other inflammatory stimuli. These symptoms and inflammation were notably absent in the offspring of pregnant mice in an unaffected control group.

Continued on page 4.

GUT-BRAIN CONNECTION IN AUTISM CONTINUED….

The neurodevelopmental aberrations the team tracked occurred while the fetus was still in the womb, yet it was not clear when the altered immune responses developed. To find out, the researchers switched mouse pups at birth so that those born to mice with immune activation were reared by mice from the inflammation-free control group, while pups born to mice from the control group were reared by mice with MIA. The team found that pups born to mice with MIA but reared by mice from the control group exhibited the autism-like symptoms but not the intestinal inflammation. Pups born to mice from the control group but reared by mice with MIA did not show autism-like symptoms but did experience intestinal inflammation. The results indicate that while neurodevelopmental pathways are altered before birth, the immune response is altered after it.

MICROBIOME-MEDIATED MOLECULAR MECHANISM

Still one question loomed large: How did mice with MIA exercise this immune-altering effect on pups after birth? Other studies have found that the maternal microbiome can influence the immune system development of offspring. To test whether that was the case in the MIA model, the researchers examined stool from MIA and control mice and found that the diversity of the microbial communities was significantly different. Then, to determine whether these differences played a causal role, the researchers raised a new set of female mice in a "germ-free" environment, meaning that the mice do not carry any microbes in or on their body. Then the scientists transplanted stool from MIA or control mice into these germ-free mice that subsequently got pregnant. Unlike with the controls, pups born to mice that received stool from mice with MIA exhibited intestinal inflammation. These results indicate that the altered microbiome of mice with MIA leads to the postnatal immune priming of offspring during rearing.

Among the notable differences the team measured in the intestinal inflammation response was an increase in IL-17a production by T cells, a major class of immune cells. IL-17a is the same cytokine that gets upregulated in mice with MIA. When the scientists compared the T cells of offspring from mice with MIA with those from control offspring, they found that in MIA-offspring, CD4 T cells were more likely to differentiate into Th17 cells, which in turn release IL-17a.

The finding prompted researchers to look at potential differences in how the CD4 T cells of the different groups may alter gene transcription. The CD4 T cells of mice exposed to the microbiomes of animals with MIA had higher expression of genes for T cell activation, suggesting they were more primed for T cell-dependent immune responses in response to infections.

"Thus, increase in IL-17a in moms during pregnancy leads to susceptibility to produce more IL-17a in offspring upon an immune challenge," Choi said.

Having established that the immune system of the offspring can become dysregulated by exposure to the mother's altered microbiome as a result of infection during pregnancy, the remaining question was how that microbiome becomes altered in the first place.

Suspecting IL-17a, the team tested the effects of antibodies that block the cytokine. When they blocked IL-17a in pregnant mice prior to immune activation, their offspring did not exhibit the intestinal inflammation later in life. This also held true when the researchers repeated the experiment of transplanting MIA stool to germ-free mice, this time including stool from pregnant MIA mice with IL-17a blockers. Again, blocking IL-17a amid maternal infection led to a microbiome that did not improperly prime the immune system of offspring.

Continued on page 5.

GUT-BRAIN CONNECTION IN AUTISM CONTINUED….

LONG-TERM QUESTIONS

Huh said the results highlight how environmental exposures during pregnancy, such as infection, could have long-term health consequences for the offspring, a concern that has always been present but that may be exacerbated by the COVID-19 pandemic. Further study is needed, he said, to determine long-term effects on children born to mothers infected with SARS-Cov-2.

Choi added that emerging connections between inflammation and neurodegenerative diseases such as Alzheimer's may also warrant further study given the team's findings of how maternal infection can lead to enhanced inflammation in offspring.

Eunha Kim and Donggi Paik of Huh's lab are the study's co-lead authors. Other investigators included Ricardo Ramirez, Delaney Biggs, Youngjun Park, and Ho-Keun Kwon.

https://news.harvard.edu/gazette/story/2022/01/link-between-inflammation-and-autism-found-within-mouse-models/

'THEORY OF MIND' IN AUTISM

'Theory of Mind' in Autism: A Research Field Reborn

The scientific process is rarely linear. New discoveries can redirect theories or derail them completely — and even disproven ideas are sometimes resurrected in new forms.

One such example is the notion that autistic people have difficulty with 'theory of mind,' the ability to understand other people's thoughts and emotions. Originally proposed in 1985, the theory steadily gained attention from the research community into the 1990s. Some researchers went so far as to assert that it explained everything about autism. But eventually, many came to view it as passé, and it was largely abandoned.

Over the past decade or so, though, theory of mind has attracted new interest from scientists who say that some aspects of the original concept, if not all of it, may still be relevant to autism.

"People thought it was all done and dusted," says Uta Frith, emeritus professor of cognitive development at University College London in the United Kingdom, and one of the first to show that autistic people perform poorly on standard theory of mind tasks. But, she says, the field might not be finished with the theory after all.

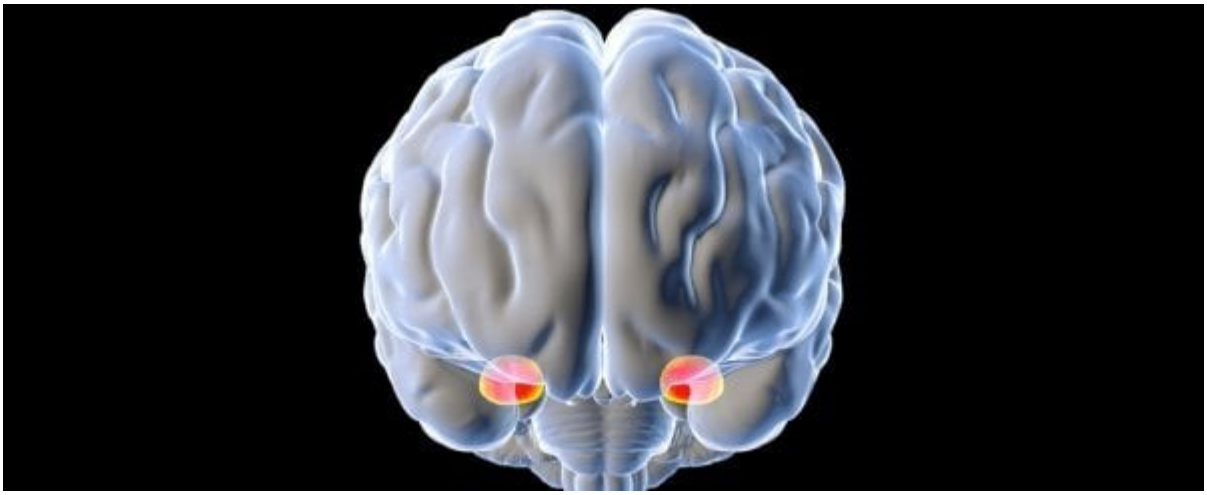
A UNIFYING CONCEPT:

Frith's interest in theory of mind in autism began with a test to identify the age at which children can reason about other people's mindsets. She and her colleagues adapted the test using a story about two dolls, Sally and Anne: Sally puts a marble into a basket and then leaves. Anne takes the marble out of the basket and places it inside a box. When Sally returns, the clinician asks the child where Sally will look for the marble.

https://youtu.be/qsEP7QTIVT0

You can continue to read the rest of this article at https://www.spectrumnews.org/features/deep-dive/theory-of-mind-in-autism-a-research-field-reborn/

RAPID GROWTH OF A KEY BRAIN STRUCTURE...



Rapid Growth of a Key Brain Structure May Be Behind Autism

RACHAEL RETTNER, LIVE SCIENCE 29 MARCH 2022

A brain structure called the amygdala grows too fast in babies who are diagnosed with autism by age 2, a new study suggests.

The study researchers found that this overgrowth occurs between 6 and 12 months of age, before children are typically diagnosed with autism.

The findings, published Friday (March 25) in *The American Journal of Psychiatry*, suggest that therapies for children at high risk of autism may have the best chance of working if they start in infancy.

"Our research suggests an optimal time to start interventions and support children who are at highest likelihood of developing autism may be during the first year of life," study senior author Dr. Joseph Piven, a professor of psychiatry and pediatrics at the University of North Carolina at Chapel Hill, said in a statement.

The amygdala is an almond-shaped structure deep in the brain that's involved with processing emotions, I ncluding feelings of fear, as well as interpreting facial expressions.

Researchers already knew that the amygdala appears larger in school-age children with ASD compared with children without ASD, but exactly when this enlargement starts was not known.

In the new study, researchers scanned the brains of more than 400 infants, including 270 who were at higher risk of developing autism because they had an older sibling with the condition; 109 infants with typical development; and 29 infants with Fragile X syndrome, a genetic disorder that causes developmental and Intellectual disability.

The children underwent MRI scans at ages six months, 12 months, and 24 months. By age 24 months, 58 (or about 21 percent) of the at-risk children had been diagnosed with ASD.

The researchers found that at age six months, all of the children had similar-sized amygdalae. But by 12 months, the children who would later develop autism had enlarged amygdalae compared with children who didn't develop autism and those with Fragile X syndrome.

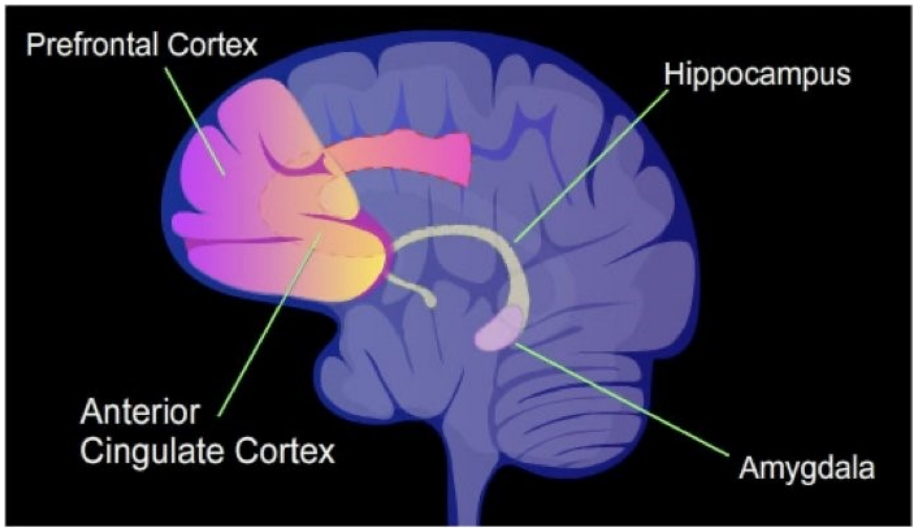
Continued on page 7.

RAPID GROWTH OF A KEY BRAIN STRUCTURE CONTINUED...

What's more, those with the fastest rate of amygdala growth had the most severe symptoms of autism.

"The faster the amygdala grew in infancy, the more social difficulties the child showed when diagnosed with autism a year later," study first author Mark Shen, an assistant professor of psychiatry and neuroscience at UNC Chapel Hill, said in the statement.

The researchers hypothesized that early problems with visual and sensory information processing in infancy may put stress on the amygdala, resulting in its overgrowth. (The amygdala receives signals from the brain's visual system and other sensory systems in order to detect threats.)



Studies have found that children that go on to be diagnosed with autism have problems as babies with how they pay attention to visual stimuli.

Interventions in babies at high risk of autism might want to focus on improving visual and other sensory information processing in babies, Piven said.

Early interventions for autism usually begin around two or three years of age, when a child is diagnosed with autism, according to the National Institutes of Health.

However, some studies have tested interventions in babies that were at risk for autism because they had a sibling with autism or in babies that showed early symptoms, such as visually fixating on certain objects, according to the autism news site Spectrum.

For example, a small 2014 study tested an intervention in children ages 6 to 15 months, which taught parents new ways of interacting with their babies, such as methods to shift the baby's attention away from an object they were fixated on; and found that the therapy reduced autism symptoms by age 3, Spectrum reported.

https://www.sciencealert.com/rapid-growth-of-this-brain-structure-may-be-behind-autism?fbclid=IwAR15HEh49QaVWQ9qu3EjyEUvUt3HBh26fcdPvd3u84Hio1pNtDzwY6PbMp4#l1mvznrsqy1aks1asbm

MINIATURE BRAIN MODELS

Miniature brain models: Understanding autism

Date: April 5, 2022
Source: Institute of Science and Technology Austria
Summary: Scientists use brain organoids to understand how a mutated gene affects brain development.

To better understand the causes of autism spectrum disorders (ASD) it is crucial to look at what is happening in the brain during development. The closest we come to observing human brains this early is by using organoids -- miniature models of organs. With their help, scientists at the Institute of Science and Technology Austria (ISTA) discovered how mutations in a high-risk gene of autism disrupt important developmental processes.
Several hundred genes are associated with autism spectrum disorders. Some patients are only mildly affected, while others have severe disabilities. In addition to characteristic symptoms like difficulties in social interaction and communication with other people, as well as repetitive-stereotypic behaviors, patients with mutations of the gene *CHD8* oftentimes have intellectual disabilities and macrocephaly -- an unusually large brain. How *CHD8* causes these symptoms has long been unclear.

Tiny artificial brains

Since *CHD8* mutations affect the brain at a very early stage of its development, it has proven difficult for scientists to get the full picture. Over the past years, many researchers therefore used mice as model organisms to better understand what is going on. "But mice with a *CHD8* mutation barely showed the symptoms human patients are showing. The effects in mice are not comparable to humans. We needed some kind of human model," Professor Gaia Novarino explains.

Together with collaborators from the Italian Human Technopole institute, the European Institute of Oncology, and the University of Milan, as well as the Allen Institute for Brain Science, USA, Novarino and her team at ISTA turned to organoids. These simplified miniature versions of organs are made from stem cells, which have the ability to become almost every other type of cell. By creating the right circumstances and giving the proper input at just the right time, the scientists were able to mimic developmental processes to create basic versions of brain tissue the size of lentils. "Organoids are the only way you can study human brain development at such an early phase," says Bárbara Oliveira, postdoc in the Novarino group and one of the authors of the study.

***CHD8* mutations disrupt balance of neuron production**

In petri dishes the team created brain organoids with and without mutations of the gene *CHD8*. "After some time, we could see by eye that the mutant organoids were much bigger. That was the first evidence that the model works," her colleague and co-author, PhD student Christoph Dotter, describes. Like patients with a *CHD8* mutation, the organoids were showing signs of brain overgrowth.

Getting an overview of all the cell types in the organoids, the team notices something very early on: The mutant organoids started to produce a specific type of neurons, inhibitory neurons, much earlier than the control group. So called excitatory neurons, however, were produced later. Furthermore, the mutant organoids produced much more proliferating cells that later on produce a larger amount of this kind of neurons. Over all, the scientists concluded, this leads to them being significantly bigger than the organoids without *CHD8* mutations correlating with patient's macrocephaly.

Continued on page 9.

MINITURE BRAIN MODELS CONTINUED...

Starting to understand our brain

Like previous studies by the Novarino group, their recent study shows just how important time is when studying autism. "Looking at different time points gives us the information that what you see in the end might not be the full picture of how the brain of a patient developed -- much more might have happened before," says Novarino. "We still have a limited understanding of how different trajectories affect functions of the brain." To one day help patients with a *CHD8* mutation, the basics of brain development need to be better understood. By reproducing genetic and clinical features from ASD patients in brain organoids, the Novarino group was able to make an important contribution.

Story Source:

Materials provided by **Institute of Science and Technology Austria**. *Note: Content may be edited for style and length.*

https://www.sciencedaily.com/releases/2022/04/220405123915.htm

AUTISM ACCEPTANCE MONTH

Autism Awareness Month shifting to Autism Acceptance Month

"Autism Awareness Month" is now being rebranded as Autism Awareness, Acceptance and Advocacy Month, or 4A for short.

COLUMBUS, Ohio — Autism Awareness month is getting a bit of a makeover.

While one in 44 children are diagnosed with autism, advocates say it's likely everyone has been directly or indirectly impacted by the disorder in some way.

That's one of the reasons why "Autism Awareness Month" is now being rebranded as Autism Awareness, Acceptance and Advocacy Month, or 4A for short.

Sarah McClary is the mother of a 10-year-old girl with autism, but she's not just a parent. McClary is also a board-certified behavior analyst for Hopebridge Autism Therapy. She agrees it's time to move past awareness and move to acceptance.

"To know of their differences and to teach your children at home and at school and in the community about differences and how we accept that acceptance and recognition of the fact that kids with autism experience the world differently and may be overwhelmed by an environment," McClary said.

While there is no cure for autism, behavior analysts say early intervention is essential.

Signs of autism spectrum disorder include difficulty with communication and social interactions, obsessive interests and repetitive behaviors.

Applied behavioral analysis and services can allow children with ASD to build skills to learn, grow and succeed in a world not created for them to easily adapt.

To learn more about Autism Acceptance Month, click here.

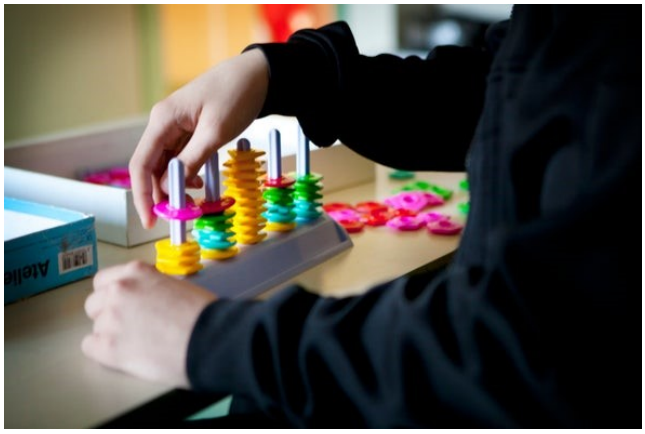
https://www.10tv.com/article/news/local/autism-awareness-month-shifting-to-autism-acceptance-month-4a/530-5cf8fb79-f35b-4fd2-ad2a-eced36546983#:~:text=Local%20News-,Autism%20Awareness%20Month%20shifting%20to%20Autism%20Acceptance%20Month,Month%2C%20or%204A%

THE CONCEPT OF NEURODIVERSITY

The Concept of Neurodiversity Is Dividing the Autism Community

It remains controversial—but it doesn't have to be

By Simon Baron-Cohen on April 30, 2019



At the annual meeting of the International Society for Autism Research (INSAR) in Montreal, Canada in May, one topic widely debated was the concept of neurodiversity.It is dividing the autism community, but it doesn't have to.

The term "neurodiversity" gained popular currency in recent years but was first used by Judy Singer, an Australian social scientist, herself autistic, and first appeared in print in the *Atlantic* in 1998.

Neurodiversity is related to the more familiar concept of biodiversity, and both are respectful ways of thinking about our planet and our communities. The notion of neurodiversity is very compatible with the civil rights plea for minorities to be accorded dignity and acceptance, and not to be pathologized. And while the neurodiversity movement acknowledges that parents or autistic people may choose to try different interventions for specific symptoms that may be causing suffering, it challenges the default assumption that autism itself is a disease or disorder that needs to be eradicated, prevented, treated or cured.

Many autistic people—especially those who have intact language and no learning difficulties such that they can self-advocate—have adopted the neurodiversity framework, coining the term "neurotypical" to describe the majority brain and seeing autism as an example of diversity in the set of all possible diverse brains, none of which is "normal" and all of which are simply different.

They argue that in highly social and unpredictable environments some of their differences may manifest as disabilities, while in more autism-friendly environments the disabilities can be minimized, allowing other differences to blossom as talents. The neurodiversity perspective reminds us that disability and even disorder may be about the person-environment fit. To quote an autistic person: "We are freshwater fish in salt water. Put us in fresh water and we function just fine. Put us in salt water and we struggle to survive."

There are also those who, while embracing some aspects of the concept of neurodiversity as applied to autism, argue that the severe challenges faced by many autistic people fit better within a more classical medical model. Many of these are parents of autistic children or autistic individuals who struggle substantially in any environment, who may have almost no language, exhibit severe learning difficulties, suffer gastrointestinal pain or epilepsy, appear to be in anguish for no apparent reason or lash out against themselves or others.

Many of those who adopt the medical model of autism call for prevention and cure of the serious impairments that can be associated with autism. In contrast, those who support neurodiversity see such language as a threat to autistic people's existence, no different than eugenics.

No wonder this concept is causing such divisions. Yet, I argue that these viewpoints are not mutually exclusive, and that we can integrate both by acknowledging that autism contains huge heterogeneity.

Continued on page 11.

THE CONCEPT OF NEURODIVERSITY CONTINUED...

Before we address heterogeneity, a technical aside about terminology: The term "disorder" is used when an individual shows symptoms that are causing dysfunction and where the cause is unknown, while the term "disease" is used when a disorder can be ascribed to a specific causal mechanism. The term "disability" is used when an individual is below average on a standardized measure of functioning and when this causes suffering in a particular environment. In contrast, the term "difference" simply refers to variation in a trait, like having blue or brown eyes .

So what is the huge heterogeneity in the autism spectrum? One source of this is in language and intelligence: As I hinted at, some autistic people have no functional language and severe developmental delay (both of which I would view as disorders), others have milder learning difficulties, while yet others have average or excellent language skills and average or even high IQ.

What all individuals on the autism spectrum share in common are social communication difficulties (both are disabilities), difficulties adjusting to unexpected change (another disability), a love of repetition or "need for sameness," unusually narrow interests, and sensory hyper- and hypo-sensitivities (all examples of difference). Autism can also be associated with cognitive strengths and even talents, notably in attention to and memory for detail, and a strong drive to detect patterns (all of these are differences). How these are manifested is likely to be strongly influenced by language and IQ.

The other source of the huge heterogeneity is that autism is frequently accompanied by co-occurring conditions. I mentioned gastrointestinal pain or epilepsy (both examples of disorders and sometimes diseases), dyspraxia, ADHD and dyslexia (all examples of disabilities), and anxiety and depression (both examples of mental health conditions). This is just a partial list. A recent study shows that 50 percent of autistic people have at least four such co-occurring conditions (including language disorder or learning difficulties), and more than 95 percent of autistic children have at least one condition in addition to autism.

The relevance of this for the neurodiversity debate is that if we dip into the wide range of features that are seen in autism, we will find differences and disabilities (both compatible with the neurodiversity framework), and we will find examples of disorders and even diseases, which are more compatible with a medical than a neurodiversity model.

Regarding scientific evidence, there is evidence for both neurodiversity and disorder. For example, at the genetic level, about 5 to15 percent of the variance in autism can be attributed to rare genetic variants/mutations, many of which cause not just autism but also severe developmental delays (disorder), while about 10 to 50 percent of the variance in autism can be attributed to common genetic variants such as single nucleotide polymorphisms (SNPs), which simply reflect individual differences or natural variation.

At the neural level, some regions of the autistic brain (such as the amygdala, in childhood) are larger, and others (such as the posterior section of the corpus callosum) are smaller. These are evidence of difference but not necessarily disorder. Early brain overgrowth is another sign of difference but not necessarily disorder.

Post-mortem studies of the autistic brain reveal a greater number of neurons in the frontal lobe, suggesting that there may be reduced apoptosis (or pruning of of neural connections) in autism, but again this may just be evidence for difference rather than disorder. Against this, structural differences in the language areas of the brain in autistic individuals who are minimally verbal are likely to be a sign of disorder.

Functional MRI (fMRI) studies at times show less or more brain activity during different tasks, and again this can be interpreted in terms of difference and disability, but not clearly evidence of disorder. On the other hand, where autistic individuals have demonstrable epilepsy with a clear electrophysiological signature, this is a sign of disorder or even disease.

Continued on page 12.

THE CONCEPT OF NEURODIVERSITY CONTINUED…

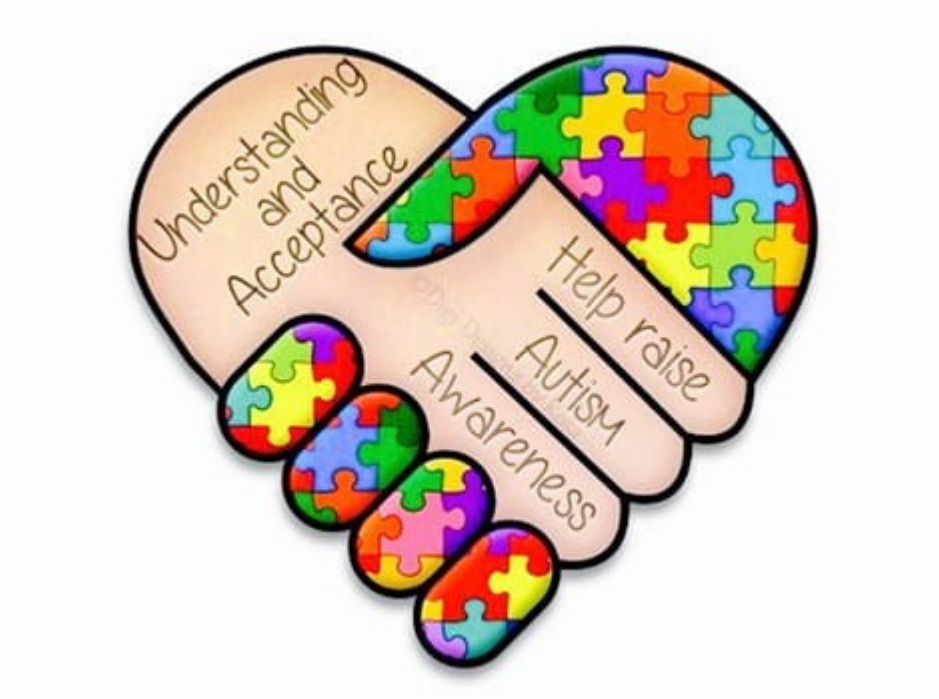
At the behavioral and cognitive levels autistic people show both differences, signs of disability and disorder. For example, young autistic toddlers may look for longer at nonsocial stimuli than at social stimuli, and autistic people may show their best performance on IQ tests on the Block Design subtest, perhaps reflecting their strong aptitude for attention to detail and disassembling complex information into its component parts.

Both of these are simply differences, compatible with the neurodiversity model. Aspects of social cognition reflect areas of disability in autism, and are often the reason for why they seek and receive a diagnosis. But if an autistic person has severe learning difficulties or is minimally verbal (defined as having fewer than 30 words), this is arguably beyond neurodiversity and more compatible with the medical model.

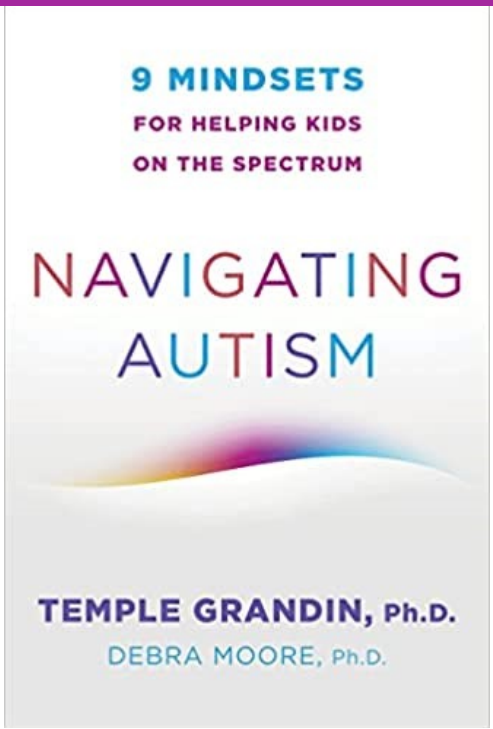
In sum, there is a case for all of the terms “disorder,” “disability,” “difference” and “disease” being applicable to different forms of autism or to the co-occurring conditions. Neurodiversity is a fact of nature; our brains are all different. So there is no point in being a neurodiversity denier, any more than being a biodiversity denier. But by taking a fine-grained look at the heterogeneity within autism we can see how sometimes the neurodiversity model fits autism very well, and that sometimes the disorder/medical model is a better explanation.

What is attractive about the neurodiversity model is that it doesn’t pathologize and focus disproportionately on what the person struggles with, and instead takes a more balanced view, to give equal attention to what the person can do. In addition it recognizes that genetic or other kinds of biological variation are intrinsic to people’s identity, their sense of self and personhood, which should be given equal respect alongside any other form of diversity, such as gender. But to encompass the breadth of the autism spectrum, we need to make space for the medical model too.

https://blogs.scientificamerican.com/observations/the-concept-of-neurodiversity-is-dividing-the-autism-community/



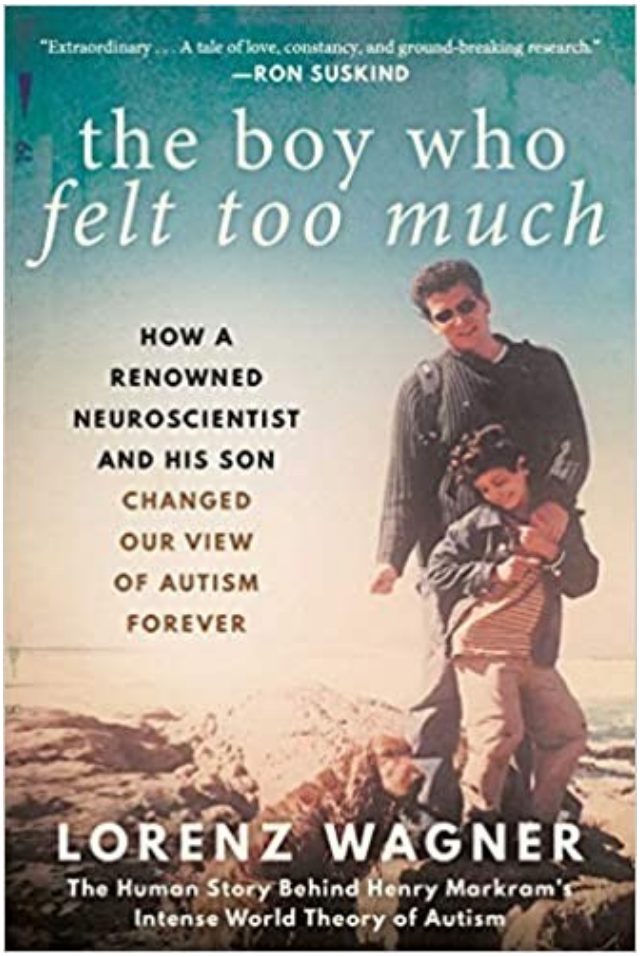
BOOK SUGGESTIONS



Navigating Autism: 9 Mindsets for Helping Kids on the Spectrum

by Temple Grandin and Debra Moore

This is an in-depth, essential resource for those helping people on the autism spectrum who want to move beyond label-locking thinking and provide a whole-person approach that honors the varied experiences of autism. Best-selling autistic writer Temple Grandin joins psychologist Debra Moore in presenting nine strengths-based mindsets necessary to successfully work with young people on the autism spectrum. Examples and stories bring the approaches to life, and detailed suggestions and checklists help readers put them to practical use. Temple Grandin shares her own personal experiences and anecdotes from parents and professionals who have sought her advice, while Debra Moore draws on more than three decades of work as a psychologist with kids on the spectrum and those who love and care for them. This resource will enlighten anyone who interacts with autistic children or teens.



The Boy Who Felt Too Much: How a Renowned Neuroscientist and his Son Changed Our View of Autism Forever

by Lorenz Wagner

Henry Markram is the Elon Musk of neuroscience, the man behind the billion-dollar Blue Brain Project to build a supercomputer model of the brain. He has set the goal of decoding all disturbances of the mind within a generation. The driving force behind this grand ambition has been his son Kai, who has autism. Raising Kai made Markram question all that he thought he knew about neuroscience, and then inspired groundbreaking research that would upend the conventional wisdom about autism, expressed in his now-famous theory of Intense World Syndrome: people like Kai don't feel too little; they feel too much. Their senses are too delicate for this world. This moving father-son story combined with elegant science writing is a must-read for anyone working with people on the spectrum.

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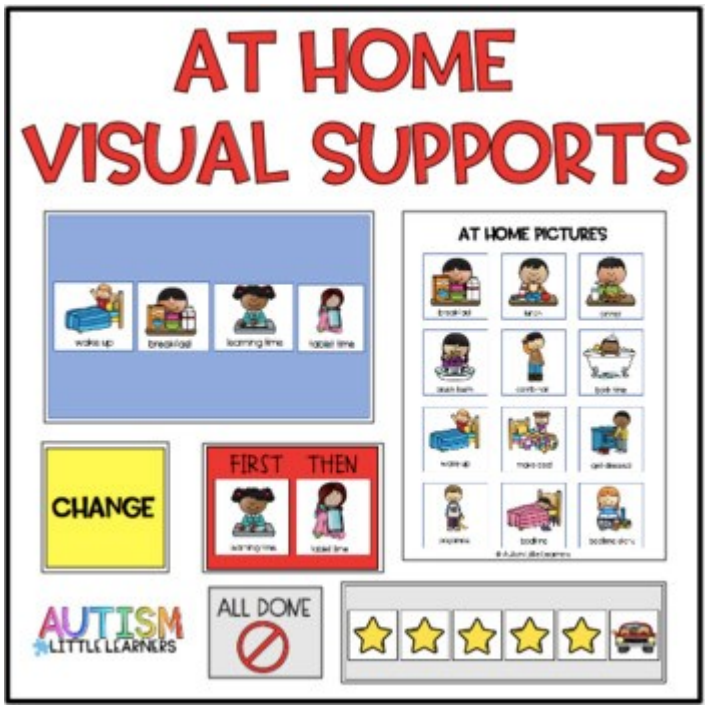

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