



Autism

Autism, also called **autism spectrum disorder**^[a] (ASD) or **autism spectrum condition** (ASC),^[10] is a neurodevelopmental disorder characterized by symptoms of deficient reciprocal social communication and the presence of restricted, repetitive, and inflexible patterns of behavior that are impairing in multiple contexts and excessive or atypical to be developmentally and socioculturally inappropriate.^{[11][12]} Other common signs include difficulty with social interaction, verbal and nonverbal communication, along with perseverative interests, stereotypic body movements, rigid routines, and hyper- or hyporeactivity to sensory input. Autism is clinically regarded as a *spectrum disorder*, meaning that it can manifest very differently in each person. For example, some are nonspeaking, while others have proficient spoken language. Because of this, there is wide variation in the support needs of people across the autism spectrum.

Psychiatry has traditionally classified autism as a mental disorder, but the autism rights movement (and an increasing number of researchers) see autistic people as part of humanity's natural neurodiversity.^{[13][14]} From this point of view, autistic people may also be diagnosed with a disability of some sort, but that disability may be rooted in the systemic structures of a society rather than in the person;^[15] thus, proponents argue that autistic people should be accommodated rather than cured.^[16] The neurodiversity perspective has led to significant controversy among those who are autistic and advocates, practitioners, and charities.^{[17][18]}

There are many theories about the causes of autism; it is highly heritable and mainly genetic, but many genes are involved, and environmental factors may also be relevant.^[19] The syndrome frequently co-occurs with attention deficit hyperactivity disorder, epilepsy, and intellectual disability. Disagreements persist about what should be included as part of the diagnosis, whether there are meaningful subtypes or stages of autism,^[20] and the significance of autism-associated traits in the wider population.^{[21][22]} The combination of broader criteria, increased awareness, and the potential increase of actual prevalence, has led to a trend of steadily increasing *estimates* of autism prevalence,^[23] unintentionally aiding the disproven myth perpetuated by anti-vaccine activists that it is caused by vaccines.^[24]

There is no cure for autism. Although early intervention services based on applied behavior analysis (ABA) can help children gain self-care, social, and language skills,^{[25][26][27][28]} independent living is unlikely in more severe forms of the condition. Speech and occupational therapy, as well as augmentative and alternative modes of communication, are effective adjunctive therapies, but some in the autism rights movement consider ABA therapy unethical and unhelpful.^[29] Pharmacological treatments may also be useful; the atypical antipsychotics

Autism	
Other names	Autism spectrum disorder (ASD), ^[a] autism spectrum condition (ASC) Formerly: <u>Kanner syndrome/autistic disorder/childhood autism</u> , <u>Asperger syndrome</u> (AS), <u>childhood disintegrative disorder</u> (CDD), <u>pervasive developmental disorder not otherwise specified</u> (PDD-NOS)
Specialty	Psychiatry, <u>clinical psychology</u> , <u>pediatrics</u> , <u>occupational medicine</u>
Symptoms	Difficulties in <u>social interaction</u> , <u>verbal and nonverbal communication</u> , and the presence of <u>repetitive behavior</u> or <u>restricted interests</u>
Complications	<u>Social isolation</u> , <u>educational and employment problems</u> , ^[1] <u>anxiety</u> , ^[1] <u>stress</u> , ^[1] <u>bullying</u> , <u>depression</u> , ^{[1][2]} <u>self-harm</u> , <u>suicidality</u> ^{[3][4]}
Usual onset	Early childhood
Duration	Lifelong
Causes	<u>Multifactorial</u> , with many uncertain factors

risperidone and aripiprazole are empirically validated for alleviating comorbid irritability, though these drugs tend to be associated with sedation and weight gain.^[30]

Classification

Spectrum model

Before the DSM-5 (2013) and ICD-11 (2022) diagnostic manuals were adopted, ASD was found under the diagnostic category pervasive developmental disorder. The previous system relied on a set of closely related and overlapping diagnoses such as Asperger syndrome and the syndrome formerly known as Kanner syndrome. This created unclear boundaries between the terms, so for the DSM-5 and ICD-11, a spectrum approach was taken. The new system is also more restrictive, meaning fewer people qualify for diagnosis.^[31]

The DSM-5 and ICD-11 use different categorization tools to define this spectrum. DSM-5 uses a "level" system, which ranks how in need of support the patient is,^[32] while the ICD-11 system has two axes, intellectual impairment and language impairment,^[33] as these are seen as the most crucial factors.

Autism is currently defined as a highly variable neurodevelopmental disorder^[34] that is generally thought to cover a broad and deep spectrum, manifesting very differently from one person to another. Some have high support needs, may be non-speaking, and experience developmental delays; this is more likely with other co-existing diagnoses. Others have relatively low support needs; they may have more typical speech-language and intellectual skills but atypical social/conversation skills, narrowly focused interests, and wordy, pedantic communication.^[35] They may still require significant support in some areas of their lives. The spectrum model should not be understood as a continuum running from mild to severe, but instead means that autism can present very differently in each person.^[36] How it presents in a person can depend on context, and may vary over time.^[37]

While the DSM and ICD greatly influence each other, there are also differences. For example, Rett syndrome was included in ASD in the DSM-5, but in the ICD-11 it was excluded and placed in the chapter on Developmental Anomalies. The ICD and the DSM change over time, and there has been collaborative work toward a convergence of the two since 1980 (when DSM-III was published and ICD-9 was current), including more rigorous biological assessment—in place of historical experience—and a simplification of the classification system.^{[38][39][40][41]}

As of 2023, empirical and theoretical research is leading to a growing consensus among researchers that the established ASD criteria are ineffective descriptors of autism as a unitary biological entity, and that alternative research approaches must be encouraged, such as going back to autism prototypes, exploring new causal models of autism, or developing transdiagnostic endophenotypes.^[42] Proposed alternatives to the current disorder-focused spectrum model deconstruct autism into at least two separate phenomena: (1) a non-pathological spectrum of behavioral traits in the population,^{[43][44]} and (2) the neuropathological burden of

Risk factors	Family history, certain genetic conditions, having older parents, certain prescribed drugs, perinatal and neonatal health issues
Diagnostic method	Based on combination of clinical observation of behavior and development and comprehensive diagnostic testing completed by a team of qualified professionals (including psychiatrists, clinical psychologists, neuropsychologists, pediatricians, and speech-language pathologists). For adults, the use of a patient's written and oral history of autistic traits becomes more important
Differential diagnosis	Intellectual disability, anxiety, bipolar disorder, depression, Rett syndrome, attention deficit hyperactivity disorder, schizoid personality disorder, selective mutism, schizophrenia, obsessive–compulsive disorder, social anxiety disorder, Einstein syndrome, PTSD, ^[5] learning disorders (mainly speech disorders)
Management	Positive behavior support, ^{[6][7]} applied

rare genetic mutations and environmental risk factors potentially leading to neurodevelopmental and psychological disorders,^{[43][44]} (3) governed by an individual's cognitive ability to compensate.^[43]

ICD

The World Health Organization's International Classification of Diseases (11th Revision), ICD-11, was released in June 2018 and came into full effect as of January 2022.^{[45][38]} It describes ASD as follows:^[46]

Autism spectrum disorder is characterised by persistent deficits in the ability to initiate and to sustain reciprocal social interaction and social communication, and by a range of restricted, repetitive, and inflexible patterns of behaviour, interests or activities that are clearly atypical or excessive for the individual's age and sociocultural context. The onset of the disorder occurs during the developmental period, typically in early childhood, but symptoms may not become fully manifest until later, when social demands exceed limited capacities. Deficits are sufficiently severe to cause impairment in personal, family, social, educational, occupational or other important areas of functioning and are usually a pervasive feature of the individual's functioning observable in all settings, although they may vary according to social, educational, or other context. Individuals along the spectrum exhibit a full range of intellectual functioning and language abilities.

—ICD-11, chapter 6, section A02

ICD-11 was produced by professionals from 55 countries out of the 90 involved and is the most widely used reference worldwide.

DSM

The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), released in 2022, is the current version of the DSM. It is the predominant mental health diagnostic system used in the United States and Canada, and is often used in Anglophone countries.

Its fifth edition, DSM-5, released in May 2013, was the first to define ASD as a single diagnosis,^[47] which is still the case in the DSM-5-TR.^[48] ASD encompasses previous diagnoses, including the four traditional diagnoses of autism—classic autism, Asperger syndrome, childhood disintegrative disorder, and Pervasive developmental disorder not otherwise specified (PDD-NOS)—and the range of diagnoses that included the word "autism".^[49] Rather than distinguishing among these diagnoses, the DSM-5 and DSM-5-TR adopt a dimensional approach with one diagnostic category for disorders that fall under the autism spectrum umbrella. Within that category, the DSM-5 and the DSM include a framework that differentiates each person by dimensions of symptom severity, as well as by associated features (i.e., the presence of other disorders or factors that likely contribute to the symptoms, other neurodevelopmental or mental disorders, intellectual disability, or language impairment).^[48] The symptom domains are (a) social communication and (b) restricted, repetitive behaviors, and there is the option of specifying a separate severity—the negative effect of the symptoms on the person—for each domain, rather than just overall severity.^[50] Before the DSM-5, the DSM separated social deficits and communication

behavior analysis,
cognitive behavioral
therapy,
occupational
therapy,
psychotropic
medication,^[8]
speech–language
pathology

Frequency
▪ One in 100
people (1%)
worldwide^[9]

deficits into two domains.^[51] Further, the DSM-5 changed to an onset age in the early developmental period, with a note that symptoms may manifest later when social demands exceed capabilities, rather than the previous, more restricted three years of age.^[52] These changes remain in the DSM-5-TR.^[48]

Common characteristics

Pre-diagnosis

For many autistic people, characteristics first appear during infancy or childhood and follow a steady course without remission (different developmental timelines are described in more detail below).^[53] Autistic people may be severely impaired in some respects but average, or even superior, in others.^{[54][55][56]}

Clinicians consider assessment for ASD when a patient shows:

- regular difficulties in social interaction or communication
- restricted or repetitive behaviors (often called "stimming")
- resistance to changes or restricted interests

These features are typically assessed with the following, when appropriate:

- problems in obtaining or sustaining employment or education
- difficulties in initiating or sustaining social relationships
- connections with mental health or learning disability services
- a history of neurodevelopmental conditions (including learning disabilities and ADHD) or mental health conditions^{[57][58]}

There are many signs associated with autism; the presentation varies widely:^{[59][60]}

Common signs for autism spectrum disorder
<ul style="list-style-type: none">▪ abnormalities in eye contact▪ little or no babbling as an infant▪ not showing interest in indicated objects▪ <u>delayed language skills</u> (e.g., having a smaller vocabulary than peers or difficulty expressing themselves in words)▪ reduced interest in other children or caretakers, possibly with more interest in objects▪ difficulty playing reciprocal games (e.g., peek-a-boo)▪ hyper- or hypo-sensitivity to or unusual response to the smell, texture, sound, taste, or appearance of things▪ resistance to changes in routine▪ repetitive, limited, or otherwise unusual usage of toys (e.g., lining up toys)▪ repetition of words or phrases (<u>echolalia</u>)▪ repetitive motions or movements, including <u>stimming</u>▪ <u>self-harming</u>

Broader autism phenotype

The broader autism phenotype describes people who may not have ASD but do have autistic traits, such as abnormalities in eye contact and stimming.^[61]

Social and communication skills

According to the medical model, autistic people experience social communications impairments. Until 2013, deficits in social function and communication were considered two separate symptom domains.^[62] The current social communication domain criteria for autism diagnosis require people to have deficits across three social skills: social-emotional reciprocity, nonverbal communication, and developing and sustaining relationships.^[48]

A deficit-based view predicts that autistic–autistic interaction would be less effective than autistic–non-autistic interactions or even non-functional.^[63] But recent research has found that autistic–autistic interactions are as effective in information transfer as interactions between non-autistics are, and that communication breaks down only between autistics and non-autistics.^{[63][64]} Also contrary to social cognitive deficit interpretations, recent (2019) research recorded similar social cognitive performances in autistic and non-autistic adults, with both of them rating autistic individuals less favorably than non-autistic individuals; however, autistic individuals showed more interest in engaging with autistic people than non-autistic people did, and learning of a person's ASD diagnosis did not influence their interest level.^[65]



In 1996, American academic Temple Grandin published *Emergence: Labeled Autistic*, describing her life experiences as an autistic person.

Thus, there has been a recent shift to acknowledge that autistic people may simply respond and behave differently than people without ASD.^[66] So far, research has identified two unconventional features by which autistic people create shared understanding (intersubjectivity): "a generous assumption of common ground that, when understood, led to rapid rapport, and, when not understood, resulted in potentially disruptive utterances; and a low demand for coordination that ameliorated many challenges associated with disruptive turns."^[64] Autistic interests, and thus conversational topics, seem to be largely driven by an intense interest in specific topics (monotropism).^{[67][68]}

Historically, autistic children were said to be delayed in developing a theory of mind, and the empathizing–systemizing theory has argued that while autistic people have compassion (affective empathy) for others with similar presentation of symptoms, they have limited, though not necessarily absent, cognitive empathy.^[69] This may present as social naïvety,^[70] lower than average intuitive perception of the utility or meaning of body language, social reciprocity,^[71] and/or social expectations, including the habitus, social cues, and/or some aspects of sarcasm,^[72] which to some degree may also be due to comorbid alexithymia.^[73] But recent research has increasingly questioned these findings, as the "double empathy problem" theory (2012) argues that there is a lack of mutual understanding and empathy between both non-autistic persons and autistic individuals.^{[74][75][76][77][78]}

As communication is bidirectional,^[79] research on communication difficulties has since also begun to study non-autistic behavior, with researcher Catherine Crompton writing in 2020 that non-autistic people "struggle to identify autistic mental states, identify autistic facial expressions, overestimate autistic egocentricity, and are less willing to socially interact with autistic people. Thus, although non-autistic people are generally characterised as socially skilled, these skills may not be functional, or effectively applied, when interacting with autistic people."^[63] Any previously observed communication deficits of autistic people may thus have been constructed through a neurotypical bias in autism research, which has come to be scrutinized for "dehumanization, objectification, and stigmatization".^[80] Recent research has proposed that autistics' lack of readability and a neurotypical lack of effort to interpret atypical signals may cause a negative interaction loop, increasingly driving both groups apart into two distinct groups with different social interaction styles.^[79]

Differences in verbal communication begin to be noticeable in childhood, as many autistic children develop language skills at an uneven pace. Verbal communication may be delayed or never develop (nonverbal autism), while reading ability may be present before school age (hyperlexia).^{[81][82]} Reduced joint attention seem to distinguish autistic from non-autistic infants.^[83] Infants may show delayed onset of babbling, unusual gestures, diminished responsiveness, and vocal patterns that are not synchronized with the caregiver. In the second and third years, autistic children may have less frequent and less diverse babbling, consonants, words, and word combinations; their gestures are less often integrated with words. Autistic children are less likely to make requests or share experiences and more likely to simply repeat others' words (echolalia).^[84] The CDC estimated in 2015 that around 40% of autistic children do not speak at all.^[85] Autistic adults' verbal communication skills largely depend on when and how well speech is acquired during childhood.^[81]

Autistic people display atypical nonverbal behaviors or show differences in nonverbal communication. They may make infrequent eye contact, even when called by name, or avoid it altogether. This may be due to the high amount of sensory input received when making eye contact.^[86] Autistic people often recognize fewer emotions and their meaning from others' facial expressions, and may not respond with facial expressions expected by their non-autistic peers.^{[87][82]} Temple

Grandin, an autistic woman involved in autism activism, described her inability to understand neurotypicals' social communication as leaving her feeling "like an anthropologist on Mars".^[88] Autistic people struggle to understand the social context and subtext of neurotypical conversational or printed situations, and form different conclusions about the content.^[89] Autistic people may not control the volume of their voice in different social settings.^[90] At least half of autistic children have atypical prosody.^[90]

What may look like self-involvement or indifference to non-autistic people stems from autistic differences in recognizing how other people have their own personalities, perspectives, and interests.^{[89][91]} Most published research focuses on the interpersonal relationship difficulties between autistic people and their non-autistic counterparts and how to solve them through teaching neurotypical social skills, but newer research has also evaluated what autistic people want from friendships, such as a sense of belonging and good mental health.^{[92][93]} Children with ASD are more frequently involved in bullying situations than their non-autistic peers, and predominantly experience bullying as victims rather than perpetrators or victim-perpetrators, especially after controlling for comorbid psychopathology.^[94] Prioritizing dependability and intimacy in friendships during adolescence, coupled with lowered friendship quantity and quality, often lead to increased loneliness in autistic people.^[95] As they progress through life, autistic people observe and form a model of social patterns, and develop coping mechanisms, referred to as "masking",^{[96][97]} which have recently been found to come with psychological costs and a higher increased risk of suicidality.^[79]

Restricted and repetitive behaviors

ASD includes a wide variety of characteristics. Some of these include behavioral characteristics which widely range from slow development of social and learning skills to difficulties creating connections with other people. Autistic people may experience these challenges with forming connections due to anxiety or depression, which they are more likely to experience, and as a result isolate themselves.^{[98][99]}

Other behavioral characteristics include abnormal responses to sensations (such as sights, sounds, touch, taste and smell) and problems keeping a consistent speech rhythm. The latter problem influences social skills, leading to potential problems in understanding for interlocutors. Autistic people's behavioral characteristics typically influence development, language, and social competence. Their behavioral characteristics can be observed as perceptual disturbances, disturbances of development rate, relating, speech and language, and motility.^[100]



A young autistic boy who has arranged his toys in a row

The second core symptom of autism spectrum is a pattern of restricted and repetitive behaviors, activities, and interests. In order to be diagnosed with ASD under the DSM-5-TR, a person must have at least two of the following behaviors:^{[48][101]}

- **Repetitive behaviors** – Repetitive behaviors such as rocking, hand flapping, finger flicking, head banging, or repeating phrases or sounds.^[102] These behaviors may occur constantly or only when the person gets stressed, anxious, or upset. These behaviors are also known as stimming.
- **Resistance to change** – A strict adherence to routines such as eating certain foods in a specific order or taking the same path to school every day.^[102] The person may become distressed if there is a change or disruption to their routine.
- **Restricted interests** – An excessive interest in a particular activity, topic, or hobby, and devoting all their attention to it. For example, young children might completely focus on things that spin and ignore everything else. Older children might try to learn everything about a single topic, such as the weather or sports, and perseverate or talk about it constantly.^[102]
- **Sensory reactivity** – An unusual reaction to certain sensory inputs, such as negative reaction to specific sounds or textures, fascination with lights or movements, or apparent indifference to pain or heat.^[103]



An older autistic boy arranging brads on a cork coaster

Autistic people can display many forms of repetitive or restricted behavior, which the Repetitive Behavior Scale-Revised (RBS-R) categorizes as follows.^[104]

- Stereotyped behaviors: Repetitive movements, such as hand flapping, head rolling, or body rocking.
- Compulsive behaviors: Time-consuming behaviors intended to reduce anxiety, that a person feels compelled to perform repeatedly or according to rigid rules, such as placing objects in a specific order, checking things, or handwashing.
- Sameness: Resistance to change; for example, insisting that the furniture not be moved or refusing to be interrupted.
- Ritualistic behavior: Unvarying pattern of daily activities, such as an unchanging menu or a dressing ritual. This is closely associated with sameness and an independent validation has suggested combining the two factors.^[104]
- Self-injury: Behaviors such as eye-poking, skin-picking, hand-biting and head-banging.^[83]

Self-injury

Self-injurious behaviors are relatively common in autistic people, and can include head-banging, self-cutting, self-biting, and hair-pulling.^[105] Some of these can result in serious injury or death.^[105] Following are theories about the cause of self-injurious behavior in children with developmental delay, including autistic children:^[106]

- Frequency and/or continuation of self-injurious behavior can be influenced by environmental factors (e.g. reward in return for halting self-injurious behavior). This theory does not apply to younger children with autism. There is some evidence that frequency of self-injurious behavior can be reduced by removing or modifying environmental factors that reinforce the behavior.^{[106]:10–12}
- Higher rates of self-injury are also noted in socially isolated autistic people. Studies have shown that socialization skills are related factors to self-injurious behavior for autistic people.^[107]
- Self-injury could be a response to modulate pain perception when chronic pain or other health problems that cause pain are present.^{[106]:12–13}
- An abnormal basal ganglia connectivity may predispose to self-injurious behavior.^{[106]:13}

The suicide rate for verbal autistics is nine times that of the general population.^[108]

Burnout

Studies have supported the common belief that autistic people become exhausted or burnt out in some situations.^{[109][110][111][112]}

Other features

Autistic people may have symptoms that do not contribute to the official diagnosis, but that can affect the person or the family.^[114]

- Some autistic people show unusual or notable abilities, ranging from splinter skills (such as the memorization of trivia) to rare talents in mathematics, music, or artistic reproduction, which in exceptional cases are considered a part of the savant syndrome.^{[115][116][117]} One study describes how some autistic people show superior skills in perception and attention relative to the general population.^[118] Sensory abnormalities are found in over 90% of autistic people, and are considered core features by some.^[119]
- More generally, autistic people tend to show a "spiky skills profile", with strong abilities in some areas contrasting with much weaker abilities in others.^[120]
- Differences between the previously recognized disorders under the autism spectrum are greater for under-responsivity (for example, walking into things) than for over-responsivity (for example, distress from loud noises) or for sensation seeking (for example, rhythmic movements).^[121] An estimated 60–80% of autistic people have motor signs that include poor muscle tone, poor motor planning, and toe walking.^{[119][122]} Deficits in motor coordination are pervasive across ASD and are greater in autism proper.^{[123][124]}
- Pathological demand avoidance can occur. People with this set of autistic symptoms are more likely to refuse to do what is asked or expected of them, even to activities they enjoy.



In 2021, screenwriter and actor Wentworth Miller revealed his autism diagnosis in a now-deleted Instagram post, stating it was "a shock" but "not a surprise".^[113]

- Unusual or atypical eating behavior occurs in about three-quarters of children with ASD, to the extent that it was formerly a diagnostic indicator.^[114] Selectivity is the most common problem, although eating rituals and food refusal also occur.^[125]

Problematic digital media use

In September 2018, the *Review Journal of Autism and Developmental Disorders* published a systematic review of 47 studies published from 2005 to 2016 that concluded that associations between autism spectrum disorder (ASD) and screen time was inconclusive.^[126] In May 2019, the *Journal of Developmental and Behavioral Pediatrics* published a systematic review of 16 studies that found that children and adolescents with ASD are exposed to more screen time than typically developing peers and that the exposure starts at a younger age.^[127] In April 2021, *Research in Autism Spectrum Disorders* published a systematic review of 12 studies of video game addiction in ASD subjects that found that children, adolescents, and adults with ASD are at greater risk of video game addiction than those without ASD, and that the data from the studies suggested that internal and external factors (sex, attention and oppositional behavior problems, social aspects, access and time spent playing video games, parental rules, and game genre) were significant predictors of video game addiction in ASD subjects.^[128] In March 2022, the *Review Journal of Autism and Developmental Disorders* published a systematic review of 21 studies investigating associations between ASD, problematic internet use, and gaming disorder where the majority of the studies found positive associations between the disorders.^[129]

In August 2022, the *International Journal of Mental Health and Addiction* published a review of 15 studies that found that high rates of video game use in boys and young males with ASD was predominantly explained by video game addiction, but also concluded that greater video game use could be a function of ASD restricted interest and that video game addiction and ASD restricted interest could have an interactive relationship.^[130] In December 2022, the *Review Journal of Autism and Developmental Disorders* published a systematic review of 10 studies researching the prevalence of problematic internet use with ASD that found that ASD subjects had more symptoms of problematic internet use than control group subjects, had higher screen time online and an earlier age of first-time use of the internet, and also greater symptoms of depression and ADHD.^[131] In July 2023, *Cureus* published a systematic review of 11 studies that concluded that earlier and longer screen time exposure for children was associated with higher risk of a child developing ASD.^[132] In December 2023, *JAMA Network Open* published a meta-analysis of 46 studies comprising 562,131 subjects that concluded that while screen time may pose a developmental risk of ASD in childhood, associations between ASD and screen time were not statistically significant when accounting for publication bias.^[133]

Possible causes

Exactly what causes autism remains unknown.^{[134][135][136][137]} It was long mostly presumed that there is a common cause at the genetic, cognitive, and neural levels for the social and non-social components of ASD's symptoms, described as a triad in the classic autism criteria.^[138] But it is increasingly suspected that autism is instead a complex disorder whose core aspects have distinct causes that often cooccur.^{[138][139]} It is unlikely that ASD has a single cause;^[139] many risk factors identified in the research literature may contribute to ASD. These include genetics, prenatal and perinatal factors (meaning factors during pregnancy or very early infancy), neuroanatomical abnormalities, and environmental factors. It is possible to identify general factors, but much more difficult to pinpoint specific ones. Given the current state of knowledge, prediction can only be of a global nature and so requires the use of general markers.^[140]

Biological subgroups

Research into causes has been hampered by the inability to identify biologically meaningful subgroups within the autistic population^[141] and by the traditional boundaries between the disciplines of psychiatry, psychology, neurology and pediatrics.^[142] Newer technologies such as fMRI and diffusion tensor imaging can help identify biologically relevant phenotypes (observable traits) that can be viewed on brain scans, to help further neurogenetic studies of autism;^[143] one example is lowered activity in the fusiform face area of the brain, which is associated with impaired perception of people versus objects.^[144] It has been proposed to classify autism using genetics as well as behavior.^[145]

Syndromic autism and non-syndromic autism

Autism spectrum disorder (ASD) can be classified into two categories: "syndromic autism" and "non-syndromic autism".

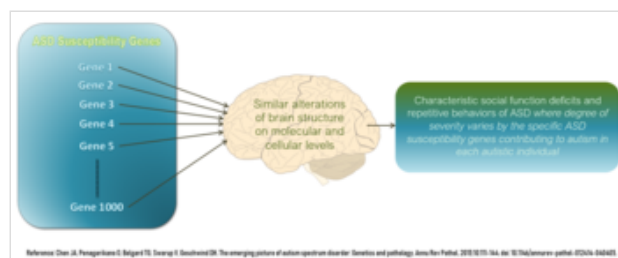
Syndromic autism refers to cases where ASD is one of the characteristics associated with a broader medical condition or syndrome, representing about 25% of ASD cases. The causes of syndromic autism are often known, and monogenic disorders account for approximately 5% of these cases.

Non-syndromic autism, also known as classic or idiopathic autism, represents the majority of cases, and its cause is typically polygenic and unknown.

Genetics

Autism has a strong genetic basis, although the genetics of autism are complex and it is unclear whether ASD is explained more by rare mutations with major effects, or by rare multi-gene interactions of common genetic variants.^{[147][148]} Complexity arises due to interactions among multiple genes, the environment, and epigenetic factors which do not change DNA sequencing but are heritable and influence gene expression.^[149] Many genes have been associated with autism through sequencing the genomes of affected people and their parents.^[150] But most of the mutations that increase autism risk have not been identified. Typically, autism cannot be traced to a Mendelian (single-gene) mutation or to a single chromosome abnormality, and none of the genetic syndromes associated with ASD have been shown to selectively cause ASD.^[147] Numerous genes have been found, with only small effects attributable to any particular gene.^[147] Most loci individually explain less than 1% of cases of autism.^[151] As of 2018, it appeared that between 74% and 93% of ASD risk is heritable.^[101] After an older child is diagnosed with ASD, 7% to 20% of subsequent children are likely to be as well.^[101] If parents have one autistic child, they have a 2% to 8% chance of having a second child who is autistic. If the autistic child is an identical twin, the other will be affected 36% to 95% of the time. A fraternal twin is affected up to 31% of the time. The large number of autistic people with unaffected family members may result from spontaneous structural variation, such as deletions, duplications or inversions in genetic material during meiosis.^{[152][153]} Hence, a substantial fraction of autism cases may be traceable to genetic causes that are highly heritable but not inherited: that is, the mutation that causes the autism is not present in the parental genome.^[154]

As of 2018, understanding of genetic risk factors had shifted from a focus on a few alleles to an understanding that genetic involvement in ASD is probably diffuse, depending on a large number of variants, some of which are common and have a small effect, and some of which are rare and have a large effect. The most common gene disrupted with large effect rare variants appeared to be CHD8, but less than 0.5% of autistic people have such a mutation. The gene CHD8 encodes the protein chromodomain helicase DNA binding protein 8, which is a chromatin regulator enzyme that is essential during fetal development. CHD8 is an adenosine triphosphate (ATP)–dependent enzyme.^{[155][156][157]} The protein contains an Snf2 helicase domain that is responsible for the hydrolysis of ATP to adenosine diphosphate (ADP).^[157] CHD8 encodes a DNA helicase that functions as a repressor of transcription, remodeling chromatin structure by altering the position of nucleosomes. CHD8 negatively regulates Wnt signaling. Wnt signaling is important in the vertebrate early development and morphogenesis. It is believed that CHD8 also recruits the linker histone H1 and causes the repression of β-catenin and p53 target genes.^[155] The importance of CHD8 can be observed in studies where CHD8-knockout mice died after 5.5 embryonic days because of widespread p53-induced apoptosis. Some studies have determined the role of CHD8 in autism spectrum disorder (ASD). CHD8 expression significantly increases during human mid-fetal development.^[155] The chromatin remodeling activity and its interaction with transcriptional regulators have shown to play an important role in ASD aetiology.^[156] The developing mammalian brain has conserved CHD8 target regions that are associated with ASD risk genes.^[158] The knockdown of CHD8 in human neural stem cells results in dysregulation of ASD risk genes that are targeted by CHD8.^[159] Recently CHD8 has been associated with the regulation of long non-coding RNAs (lncRNAs),^[160] and the regulation of X chromosome inactivation (XCI) initiation, via regulation of Xist long non-coding RNA, the master regulator of XCI, though competitive binding to Xist regulatory regions.^[161]



Hundreds of different genes are implicated in susceptibility to developing autism,^[146] most of which alter the brain structure in a similar way.

Some ASD is associated with clearly genetic conditions, like fragile X syndrome, but only around 2% of autistic people have fragile X.^[101] Hypotheses from evolutionary psychiatry suggest that these genes persist because they are linked to human inventiveness, intelligence or systemising.^{[162][163]}

Current research suggests that genes that increase susceptibility to ASD are ones that control protein synthesis in neuronal cells in response to cell needs, activity and adhesion of neuronal cells, synapse formation and remodeling, and excitatory to inhibitory neurotransmitter balance. Therefore, although up to 1,000 different genes are thought to increase the risk of ASD, all of them eventually affect normal neural development and connectivity between different functional areas of the brain in a similar manner that is characteristic of an ASD brain. Some of these genes are known to modulate production of the GABA neurotransmitter, the nervous system's main inhibitory neurotransmitter. These GABA-related genes are under-expressed in an ASD brain. On the other hand, genes controlling expression of glial and immune cells in the brain, e.g. astrocytes and microglia, respectively, are over-expressed, which correlates with increased number of glial and immune cells found in postmortem ASD brains. Some genes under investigation in ASD pathophysiology are those that affect the mTOR signaling pathway, which supports cell growth and survival.^[164]

All these genetic variants contribute to the development of the autism spectrum, but it cannot be guaranteed that they are determinants for the development.^[165]

ASD may be under-diagnosed in women and girls due to an assumption that it is primarily a male condition,^[166] but genetic phenomena such as imprinting and X linkage have the ability to raise the frequency and severity of conditions in males, and theories have been put forward for a genetic reason why males are diagnosed more often, such as the imprinted brain hypothesis and the extreme male brain theory.^{[167][168][169]}

Early life

Several prenatal and perinatal complications have been reported as possible risk factors for autism. These risk factors include maternal gestational diabetes, maternal and paternal age over 30,^{[170][171][172]} bleeding during pregnancy after the first trimester, use of certain prescription medication (e.g. valproate) during pregnancy, and meconium in the amniotic fluid. Research is not conclusive on the relation of these factors to autism, but each of them has been identified more frequently in children with autism compared to their siblings who do not have autism and other typically developing youth.^[173] While it is unclear if any single factors during the prenatal phase affect the risk of autism,^[174] complications during pregnancy may be a risk.^[174]

There are also studies being done to test whether certain types of regressive autism have an autoimmune basis.^[175]

Maternal nutrition and inflammation during preconception and pregnancy influences fetal neurodevelopment. Intrauterine growth restriction is associated with ASD, in both term and preterm infants.^[176] Maternal inflammatory and autoimmune diseases may damage fetal tissues, aggravating a genetic problem or damaging the nervous system.^[177] Systematic reviews and meta-analyses have found that maternal prenatal infections, prenatal antibiotic exposure, and post-term pregnancies are associated with increased risk of ASD in children.^{[178][179][180]}

Exposure to air pollution during child pregnancy, especially heavy metals and particulates, may increase the risk of autism.^{[181][182]} Environmental factors that have been claimed without evidence to contribute to or exacerbate autism include certain foods, infectious diseases, solvents, PCBs, phthalates and phenols used in plastic products, pesticides, brominated flame retardants, alcohol, smoking, illicit drugs, vaccines,^[183] and prenatal stress. Some, such as the MMR vaccine, have been completely disproven.^{[184][185][186][187]}

Disproven vaccine hypothesis

Parents may first become aware of ASD symptoms in their child around the time of a routine vaccination. This has led to unsupported and disproven theories blaming vaccine "overload", the vaccine preservative thiomersal, or the MMR vaccine for causing autism spectrum disorder.^[188] In 1998, British physician and academic Andrew Wakefield led a fraudulent, litigation-funded study that suggested that the MMR vaccine may cause autism.^{[189][190][191][192][193]}

Two versions of the vaccine causation hypothesis were that autism results from brain damage caused by either the MMR vaccine itself, or by mercury used as a vaccine preservative.^[194] No convincing scientific evidence supports these claims.^[24] They are biologically implausible,^[188] and further evidence continues to refute them, including the observation that the rate of autism continues to climb despite elimination of thimerosal from most routine vaccines given to children from birth to 6 years of age.^{[195][196][197][198][199]}

A 2014 meta-analysis examined ten major studies on autism and vaccines involving 1.25 million children worldwide; it concluded that neither the vaccine preservative thimerosal (mercury), nor the MMR vaccine, which has never contained thimerosal,^[200] lead to the development of ASDs.^[201] Despite this, misplaced parental concern has led to lower rates of childhood immunizations, outbreaks of previously controlled childhood diseases in some countries, and the preventable deaths of several children.^{[202][203]}

Etiological hypotheses

Several hypotheses have been presented that try to explain how and why autism develops by integrating known causes (genetic and environmental effects) and findings (neurobiological and somatic). Some are more comprehensive, such as the Pathogenetic Triad, which proposes and operationalizes three core features (an autistic personality, cognitive compensation, neuropathological burden) that interact to cause autism,^[204] and the Intense World Theory, which explains autism through a hyper-active neurobiology that leads to an increased perception, attention, memory, and emotionality.^[205] There are also simpler hypotheses that explain only individual parts of the neurobiology or phenotype of autism, such as mind-blindness (a decreased ability for theory of mind), the weak central coherence theory, or the extreme male brain and empathising–systemising theory.

Evolutionary hypotheses

Research exploring the evolutionary benefits of autism and associated genes has suggested that autistic people may have played a "unique role in technological spheres and understanding of natural systems" in the course of human development.^{[206][207]} It has been suggested that autism may have arisen as "a slight trade off for other traits that are seen as highly advantageous", providing "advantages in tool making and mechanical thinking", with speculation that the condition may "reveal itself to be the result of a balanced polymorphism, like sickle cell anemia, that is advantageous in a certain mixture of genes and disadvantageous in specific combinations".^[208] In 2011, a paper in *Evolutionary Psychology* proposed that autistic traits, including increased spatial intelligence, concentration and memory, could have been naturally selected to enable self-sufficient foraging in a more (although not completely) solitary environment. This is called the "Solitary Forager Hypothesis".^{[209][210][211]} A 2016 paper examines Asperger syndrome as "an alternative prosocial adaptive strategy" that may have developed as a result of the emergence of "collaborative morality" in the context of small-scale hunter-gathering, i.e., where "a positive social reputation for making a contribution to group wellbeing and survival" becomes more important than complex social understanding.^[212]

Some multidisciplinary research suggests that recent human evolution may be a driving force in the rise of a number of medical conditions, including autism, in recent human populations. Studies in evolutionary medicine indicate that as cultural evolution outpaces biological evolution, disorders linked to bodily dysfunction increase in prevalence due to lack of contact with pathogens and negative environmental conditions that once widely affected ancestral populations. Because natural selection favors reproduction over health and longevity, the lack of this impetus to adapt to certain harmful circumstances creates a tendency for genes in descendant populations to over-express themselves, which may cause a wide array of maladies, ranging from mental disorders to autoimmune diseases.^[213] Conversely, noting the failure to find specific alleles that reliably cause autism or rare mutations that account for more than 5% of the heritable variation in autism established by twin and adoption studies, research in evolutionary psychiatry has concluded that it is unlikely that there is selection pressure for autism when considering that, like schizophrenics, autistic people and their siblings tend to have

fewer offspring on average than non-autistic people, and instead that autism is probably better explained as a by-product of adaptive traits caused by antagonistic pleiotropy and by genes that are retained due to a fitness landscape with an asymmetric distribution.^{[214][215]}

Pathophysiology

Diagnosis

Conditions correlated or comorbid to autism

Autism is correlated or comorbid with several personality traits/disorders.^[144] Comorbidity may increase with age and may worsen the course of youth with ASDs and make intervention and treatment more difficult. Distinguishing between ASDs and other diagnoses can be challenging because the traits of ASDs often overlap with symptoms of other disorders, and the characteristics of ASDs make traditional diagnostic procedures difficult.^{[216][217]}

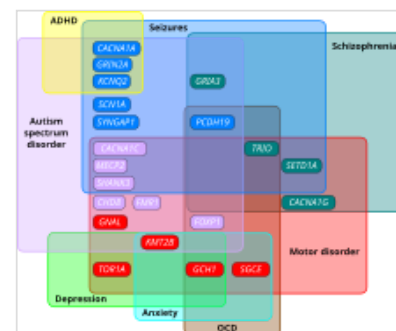
Correlations

Research indicates that autistic people are significantly more likely to be LGBT than the general population.^[218] There is tentative evidence that gender dysphoria occurs more frequently in autistic people.^{[219][220]} A 2021 anonymized online survey of 16- to 90-year-olds revealed that autistic males are more likely to identify as bisexual than their non-autistic peers, while autistic females are more likely to identify as homosexual than non-autistic females do.^[221]

People on the autism spectrum are significantly more likely to be non-theistic than members of the general population.^[222]

Comorbidities

- The most common medical condition occurring in autistic people is seizure disorder or epilepsy, which occurs in 11–39% of autistic people.^[223] The risk varies with age, cognitive level, and type of language disorder.^[224]
- Tuberous sclerosis, an autosomal dominant genetic condition in which non-malignant tumors grow in the brain and on other vital organs, is present in 1–4% of autistic people.^[225]
- Intellectual disabilities are some of the most common comorbid disorders with ASDs. As diagnosis is increasingly being given to people with higher functioning autism, there is a tendency for the proportion with comorbid intellectual disability to decrease over time. In a 2019 study, it was estimated that approximately 30–40% of people diagnosed with ASD also have intellectual disability.^[226] Recent research has suggested that autistic people with intellectual disability tend to have rarer, more harmful, genetic mutations than those found in people solely diagnosed with autism.^[227] A number of genetic syndromes causing intellectual disability may also be comorbid with ASD, including fragile X, Down, Prader-Willi, Angelman, Williams syndrome,^[228] branched-chain keto acid dehydrogenase kinase deficiency,^{[229][230]} and SYNGAP1-related intellectual disability.^{[231][232]}
- Learning disabilities are also highly comorbid in people with an ASD. Approximately 25–75% of people with an ASD also have some degree of a learning disability.^[233] In particular, attention deficit disorder, which is generally more prevalent than autism (ca. 8% vs. 1%), is not directly related, though it is sometimes comorbid with autism.^[234]
- Various anxiety disorders tend to co-occur with ASDs, with overall comorbidity rates of 7–84%.^[235] They are common among children with ASD; there are no firm data, but studies have reported prevalences



Euler diagram showing overlapping clinical phenotypes in genes associated with monogenic forms of autism spectrum disorder (ASD), dystonia, epilepsy and schizophrenia:

- Genes associated with epilepsy
- Genes associated with schizophrenia
- Genes associated with autism spectrum disorder
- Genes associated with dystonia

ranging from 11% to 84%. Many anxiety disorders have symptoms that are better explained by ASD itself or are hard to distinguish from ASD's symptoms.^[236]

- Rates of comorbid depression in people with an ASD range from 4–58%.^[237]
- The relationship between ASD and schizophrenia remains a controversial subject under continued investigation, and recent meta-analyses have examined genetic, environmental, infectious, and immune risk factors that may be shared between the two conditions.^{[238][239][240]} Oxidative stress, DNA damage and DNA repair have been postulated to play a role in the aetiopathology of both ASD and schizophrenia.^[241]
- Deficits in ASD are often linked to behavior problems, such as difficulties following directions, being cooperative, and doing things on other people's terms.^[242] Symptoms similar to those of attention deficit hyperactivity disorder (ADHD) can be part of an ASD diagnosis.^[243]
- Sensory processing disorder is also comorbid with ASD, with comorbidity rates of 42–88%.^[244]
- Starting in adolescence, some people with Asperger syndrome (26% in one sample)^[245] fall under the criteria for the similar condition schizoid personality disorder, which is characterized by a lack of interest in social relationships, a tendency towards a solitary or sheltered lifestyle, secretiveness, emotional coldness, detachment and apathy.^{[245][246][247]} Asperger syndrome was traditionally called "schizoid disorder of childhood".
- Genetic disorders – about 10–15% of autism cases have an identifiable Mendelian (single-gene) condition, chromosome abnormality, or other genetic syndromes.^[248]
- Several metabolic defects, such as phenylketonuria, are associated with autistic symptoms.^[249]
- Gastrointestinal problems are one of the most commonly co-occurring medical conditions in autistic people.^[250] These are linked to greater social impairment, irritability, language impairments, mood changes, and behavior and sleep problems.^{[250][251][252]} A 2015 review proposed that immune, gastrointestinal inflammation, malfunction of the autonomic nervous system, gut flora alterations, and food metabolites may cause brain neuroinflammation and dysfunction.^[251] A 2016 review concludes that enteric nervous system abnormalities might play a role in neurological disorders such as autism. Neural connections and the immune system are a pathway that may allow diseases originated in the intestine to spread to the brain.^[252]
- Sleep problems affect about two-thirds of autistic people at some point in childhood. These most commonly include symptoms of insomnia, such as difficulty falling asleep, frequent nocturnal awakenings, and early morning awakenings. Sleep problems are associated with difficult behaviors and family stress, and are often a focus of clinical attention over and above the primary ASD diagnosis.^[253]
- Dysautonomia is common in ASD, affecting heart rate and blood pressure and causing symptoms such as brain fog, blurry vision, and bowel dysfunction.^[254] It can be diagnosed through a Tilt table test.^[255]
- The frequency of ASD is 10 times higher in Mast cell activation syndrome patients than in the general population. This immunological condition causes cardiovascular, dermatological, gastrointestinal, neurological, and respiratory problems.^[256]

Management

There is no treatment as such for autism,^[257] and many sources advise that this is not an appropriate goal,^{[258][259]} although treatment of co-occurring conditions remains an important goal.^[260] There is no cure for autism as of 2024, nor can any of the known treatments significantly reduce brain mutations caused by autism, although those who require little to no support are more likely to experience a lessening of symptoms over time.^{[261][262][263]} Several interventions can help children with autism,^[264] and no single treatment is best, with treatment typically tailored to the child's needs.^[265] Studies of interventions have methodological problems that prevent definitive conclusions about efficacy,^[266] but the development of evidence-based interventions has advanced.^[25]

The main goals of treatment are to lessen associated deficits and family distress, and to increase quality of life and functional independence. In general, higher IQs are correlated with greater responsiveness to treatment and improved treatment outcomes.^{[267][25]} Behavioral, psychological, education, and/or skill-building interventions may be used to assist autistic people to learn life skills necessary for living independently,^[268] as well as other social, communication, and language skills. Therapy also aims to reduce challenging behaviors and build upon strengths.^[269]

Intensive, sustained special education programs and behavior therapy early in life may help children acquire self-care, language, and job skills.^[265] Although evidence-based interventions for autistic children vary in their methods, many adopt a psychoeducational approach to enhancing cognitive, communication, and social skills while minimizing problem behaviors. While medications have not been found to help with core symptoms, they may be used for associated symptoms, such as irritability, inattention, or repetitive behavior patterns.^[270]

Non-pharmacological interventions

Intensive, sustained special education or remedial education programs and behavior therapy early in life may help children acquire self-care, social, and job skills. Available approaches include applied behavior analysis, developmental models, structured teaching, speech and language therapy, cognitive behavioral therapy,^[271] social skills therapy, and occupational therapy.^[272] Among these approaches, interventions either treat autistic features comprehensively, or focus treatment on a specific area of deficit.^[25] Generally, when educating those with autism, specific tactics may be used to effectively relay information to these people. Using as much social interaction as possible is key in targeting the inhibition autistic people experience concerning person-to-person contact. Additionally, research has shown that employing semantic groupings, which involves assigning words to typical conceptual categories, can be beneficial in fostering learning.^[273]

There has been increasing attention to the development of evidence-based interventions for autistic young children. Three theoretical frameworks outlined for early childhood intervention include applied behavior analysis (ABA), the developmental social-pragmatic model (DSP) and cognitive behavioral therapy (CBT).^{[271][25]} Although ABA therapy has a strong evidence base, particularly in regard to early intensive home-based therapy, ABA's effectiveness may be limited by diagnostic severity and IQ of the person affected by ASD.^[274] The *Journal of Clinical Child and Adolescent Psychology* has published a paper deeming two early childhood interventions "well-established": individual comprehensive ABA, and focused teacher-implemented ABA combined with DSP.^[25]

Many people, including autistic adults, have criticized ABA, calling it unhelpful, unethical, and even abuse.^{[15][275][276][277]} Autistic scholar Nick Walker, who experienced ABA as a child, has said that, through ABA, autistic children are "abused, coerced, and traumatized into imitating the outward behavior of neurotypical children, at the expense of their long-term psychological well-being."^[15] Sandoval-Norton et al. also discuss the "unintended but damaging consequences, such as prompt dependency, psychological abuse and compliance" that result in autistic people facing challenges as they transition into adulthood.^[275] Some ABA advocates have responded to such critiques that, instead of stopping ABA, there should be movement to increase protections and ethical compliance when working with autistic children.^[278]

Another evidence-based intervention that has demonstrated efficacy is a parent training model, which teaches parents how to implement various ABA and DSP techniques themselves.^[25] Various DSP programs have been developed to explicitly deliver intervention systems through at-home parent implementation.

In October 2015, the American Academy of Pediatrics (AAP) proposed new evidence-based recommendations for early interventions in ASD for children under 3.^[279] These recommendations emphasize early involvement with both developmental and behavioral methods, support by and for parents and caregivers, and a focus on both the core and associated symptoms of ASD.^[279] But a Cochrane review found no evidence that early intensive behavioral intervention (EIBI) is effective in reducing behavioral problems associated with autism in most autistic children, though it did improve IQ and language skills.^[280] The Cochrane review acknowledged that this may be due to the low quality of studies available on EIBI and therefore providers should recommend EIBI based on their clinical judgment and the family's preferences.^[280] No adverse effects of EIBI treatment were found.^[280] A meta-analysis in that same database indicates that due to the heterogeneity in ASD, children progress to differing early intervention modalities based on ABA.^[26]

ASD treatment generally focuses on behavioral and educational interventions to target its two core symptoms: social communication deficits and restricted, repetitive behaviors.^[281] If symptoms continue after behavioral strategies have been implemented, some medications can be recommended to target specific symptoms or co-existing problems such as restricted and repetitive behaviors (RRBs), anxiety, depression, hyperactivity/inattention and sleep disturbance.^[281] Melatonin, for example, can be used for sleep problems.^[282]

Several parent-mediated behavioral therapies target social communication deficits in children with autism, but their efficacy in treating RRBs is uncertain.^[283]

Education

Educational interventions often used include applied behavior analysis (ABA), developmental models, structured teaching, speech and language therapy and social skills therapy.^[265] Among these approaches, interventions either treat autistic features comprehensively, or focalize treatment on a specific area of deficit.^[25]

The quality of research for early intensive behavioral intervention (EIBI)—a treatment procedure incorporating over 30 hours per week of the structured type of ABA that is carried out with very young children—is low; more vigorous research designs with larger sample sizes are needed.^[280] Two theoretical frameworks outlined for early childhood intervention include structured and naturalistic ABA interventions, and developmental social pragmatic models (DSP).^[25] One interventional strategy utilizes a parent training model, which teaches parents how to implement various ABA and DSP techniques, allowing for parents to disseminate interventions themselves.^[25] Various DSP programs have been developed to explicitly deliver intervention systems through at-home parent implementation. Despite the recent development of parent training models, these interventions have demonstrated effectiveness in numerous studies, being evaluated as a probable efficacious mode of treatment.^[25] Early, intensive ABA therapy has demonstrated effectiveness in enhancing communication and adaptive functioning in preschool children;^{[265][285]} it is also well-established for improving the intellectual performance of that age group.^[265]



An autistic three-year-old points to fish in an aquarium, as part of an experiment on the effect of intensive shared-attention training on language development.^[284]

In 2018, a Cochrane meta-analysis database concluded that some recent research is beginning to suggest that because of the heterology of ASD, there are two different ABA teaching approaches to acquiring spoken language: children with higher receptive language skills respond to 2.5 to 20 hours per week of the naturalistic approach, whereas children with lower receptive language skills require 25 hours per week of discrete trial training—the structured and intensive form of ABA.^[26] A 2023 randomized control trial study of 164 participants showed similar findings.^[27]

Similarly, a teacher-implemented intervention that utilizes a more naturalistic form of ABA combined with a developmental social pragmatic approach has been found to be beneficial in improving social-communication skills in young children, although there is less evidence in its treatment of global symptoms.^[25] Neuropsychological reports are often poorly communicated to educators, resulting in a gap between what a report recommends and what education is provided.^[286] The appropriateness of including children with varying severity of autism spectrum disorders in the general education population is a subject of current debate among educators and researchers.^[287]

Pharmacological interventions

Medications may be used to treat ASD symptoms that interfere with integrating a child into home or school when behavioral treatment fails.^[288] They may also be used for associated health problems, such as ADHD, anxiety, or if the person is hurting themselves or aggressive with others,^{[288][289]} but their routine prescription for ASD's core features is not recommended.^[290] More than half of US children diagnosed with ASD are prescribed psychoactive drugs or anticonvulsants, with the most common drug classes being antidepressants, stimulants, and antipsychotics.^{[291][292]} The atypical antipsychotic drugs risperidone and aripiprazole are FDA-approved for treating associated aggressive and self-injurious behaviors.^{[270][293]} But their side effects must be weighed against their potential benefits, and autistic people may respond atypically.^[270] Side effects may include weight gain, tiredness, drooling, and aggression.^[270] Some emerging data show positive effects of aripiprazole and risperidone on restricted and repetitive behaviors (i.e., stimming; e.g., flapping, twisting, complex whole-body movements),^[290] but due to the small sample size and different focus of these studies and the concerns about its side effects, antipsychotics are not recommended as primary treatment of RRBs.^[294] SSRI antidepressants, such as fluoxetine and fluvoxamine, have been shown to be effective in reducing repetitive and ritualistic behaviors, while the stimulant medication methylphenidate is beneficial for some children with comorbid inattentiveness or

hyperactivity.^[265] There is scant reliable research about the effectiveness or safety of drug treatments for adolescents and adults with ASD.^[295] No known medication relieves autism's core symptoms of social and communication impairments.^[270]

Alternative medicine

A multitude of alternative therapies have been researched and implemented, and many have resulted in harm to autistic people.^[272] A 2020 systematic review on adults with autism provided evidence that mindfulness-based interventions may decrease stress, anxiety, ruminating thoughts, anger, and aggression and improve mental health.^[296]

Although popularly used as an alternative treatment for autistic people, as of 2018 there is no good evidence to recommend a gluten- and casein-free diet as a standard treatment.^{[297][298][299]} A 2018 review concluded that it may be a therapeutic option for specific groups of children with autism, such as those with known food intolerances or allergies, or with food intolerance markers. The authors analyzed the prospective trials conducted to date that studied the efficacy of the gluten- and casein-free diet in children with ASD (4 in total). All of them compared gluten- and casein-free diet versus normal diet with a control group (2 double-blind randomized controlled trials, 1 double-blind crossover trial, 1 single-blind trial). In two of the studies, whose duration was 12 and 24 months, a significant improvement in ASD symptoms (efficacy rate 50%) was identified. In the other two studies, whose duration was 3 months, no significant effect was observed.^[297] The authors concluded that a longer duration of the diet may be necessary to achieve the improvement of the ASD symptoms.^[297] Other problems documented in the trials carried out include transgressions of the diet, small sample size, the heterogeneity of the participants and the possibility of a placebo effect.^{[299][300][301]} In the subset of people who have gluten sensitivity there is limited evidence that suggests that a gluten-free diet may improve some autistic behaviors.^{[302][303][304]}

The preference that autistic children have for unconventional foods can lead to reduction in bone cortical thickness with this risk being greater in those on casein-free diets, as a consequence of the low intake of calcium and vitamin D; however, suboptimal bone development in ASD has also been associated with lack of exercise and gastrointestinal disorders.^[305] In 2005, botched chelation therapy killed a five-year-old child with autism.^{[306][307]} Chelation is not recommended for autistic people since the associated risks outweigh any potential benefits.^[308] Another alternative medicine practice with no evidence is CEASE therapy, a pseudoscientific mixture of homeopathy, supplements, and "vaccine detoxing".^[309]

Results of a systematic review on interventions to address health outcomes among autistic adults found emerging evidence to support mindfulness-based interventions for improving mental health. This includes decreasing stress, anxiety, ruminating thoughts, anger, and aggression.^[310] An updated Cochrane review (2022) found evidence that music therapy likely improves social interactions, verbal communication, and nonverbal communication skills.^[311] There has been early research on hyperbaric treatments in children with autism.^[312] Studies on pet therapy have shown positive effects.^[313]

Prevention

While infection with rubella during pregnancy causes fewer than 1% of cases of autism,^[314] vaccination against rubella can prevent many of those cases.^[315]

Prognosis

There is no evidence of a cure for autism.^{[265][144]} The degree of symptoms can decrease, occasionally to the extent that people lose their diagnosis of ASD;^{[316][317]} this occurs sometimes after intensive treatment^[318] and sometimes not. It is not known how often this outcome happens,^[319] with reported rates in unselected samples ranging from 3% to 25%.^{[316][317]} Although core difficulties tend to persist, symptoms often become less severe with age.^[149] Acquiring language before age six, having an IQ above 50, and having a marketable skill all predict better outcomes; independent living is unlikely in autistic people with higher support needs.^[320]

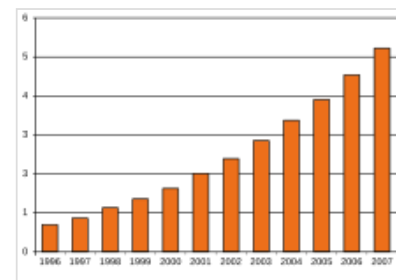
Among others, academic Temple Grandin has advised against striving to cure autism, saying that if a cure were found, she would choose to stay the way she is. She wrote, "The skills that people with autism bring to the table should be nurtured for their benefit and [for the benefit of] society", adding, "If you totally get rid of autism, you'd have nobody to fix your

computer in the future".^{[321][322]}

The prognosis of autism describes the developmental course, gradual autism development, regressive autism development, differential outcomes, academic performance and employment.

Epidemiology

The World Health Organization (WHO) estimates about 1 in 100 children had autism during the period from 2012 to 2021 as that was the average estimate in studies published during that period with a trend of increasing prevalence over time. However, the study's 1% figure may reflect an underestimate of prevalence in low- and middle-income countries.^{[9][323]} The number of people diagnosed has increased considerably since the 1990s, which may be partly due to increased recognition of the condition.^[324]



Reports of autism cases per 1,000 children rose considerably in the US from 1996 to 2007. It is unknown how much growth came from changes in rates of autism.

While rates of ASD are consistent across cultures, they vary greatly by gender, with boys diagnosed far more frequently than girls: 1 in 70 boys, but only 1 in 315 girls at eight years of age.^[325] Girls, however, are more likely to have associated cognitive impairment, suggesting that less severe forms of ASD are likely being missed in girls and women.^[326] Prevalence differences may be a result of gender differences in expression of clinical symptoms, with women and girls with autism showing less atypical behaviors and, therefore, less likely to receive an ASD diagnosis.^[327]

Using DSM-5 criteria, 92% of the children diagnosed per DSM-IV with one of the disorders which is considered part of ASD will still meet the diagnostic criteria of ASD. However, if both ASD and the social (pragmatic) communication disorder categories of DSM-5 are combined, the prevalence of autism is mostly unchanged from the prevalence per the DSM-IV criteria. The best estimate for prevalence of ASD is 0.7% or 1 child in 143 children.^[328] Relatively mild forms of autism, such as Asperger's as well as other developmental disorders, are included in the DSM-5 diagnostic criteria.^[329] ASD rates were constant between 2014 and 2016 but twice the rate compared to the time period between 2011 and 2014 (1.25 vs 2.47%). A Canadian meta-analysis from 2019 confirmed these effects as the profiles of autistic people became less and less different from the profiles of the general population.^[330] In the US, the rates for diagnosed ASD have been steadily increasing since 2000 when records began being kept.^[331] While it remains unclear whether this trend represents a true rise in incidence, it likely reflects changes in ASD diagnostic criteria, improved detection, and increased public awareness of autism.^[332] In 2012, the NHS estimated that the overall prevalence of autism among adults aged 18 years and over in the UK was 1.1%.^[333] A 2016 survey in the United States reported a rate of 25 per 1,000 children for ASD.^[334] Rates of autism are poorly understood in many low- and middle-income countries, which affects the accuracy of global ASD prevalence estimates,^[335] but it is thought that most autistic people live in low- and middle-income countries.^[336]

In 2020, the Centers for Disease Control's Autism and Developmental Disabilities Monitoring (ADDM) Network reported that approximately 1 in 54 children in the United States (1 in 34 boys, and 1 in 144 girls) is diagnosed with an autism spectrum disorder (ASD), based on data collected in 2016.^{[337][338]} This estimate is a 10% increase from the 1 in 59 rate in 2014, a 105% increase from the 1 in 110 rate in 2006, and a 176% increase from the 1 in 150 rate in 2000.^[337] Diagnostic criteria for ASD have changed significantly since the 1980s; for example, U.S. special-education autism classification was introduced in 1994.^[183]

In the UK, from 1998 to 2018, the autism diagnoses increased by 787%.^[324] This increase is largely attributable to changes in diagnostic practices, referral patterns, availability of services, age at diagnosis, and public awareness^{[339][340][341]} (particularly among women),^[324] though unidentified environmental risk factors cannot be ruled out.^[342] The available evidence does not rule out the possibility that autism's true prevalence has increased;^[339] a real increase would suggest directing more attention and funding toward psychosocial factors and changing environmental factors instead of continuing to focus on genetics.^[343] It has been established that vaccination is not a risk factor for autism and is not a cause of any increase in autism prevalence rates, if any change in the rate of autism exists at all.^[201]

Males have higher likelihood of being diagnosed with ASD than females. The sex ratio averages 4.3:1 and is greatly modified by cognitive impairment: it may be close to 2:1 with intellectual disability and more than 5.5:1 without.^[183] Several theories about the higher prevalence in males have been investigated, but the cause of the difference is unconfirmed;^[344] one theory is that females are underdiagnosed.^[345]

The risk of developing autism is greater with older fathers than with older mothers; two potential explanations are the known increase in mutation burden in older sperm, and the hypothesis that men marry later if they carry genetic liability and show some signs of autism.^[34] Most professionals believe that race, ethnicity, and socioeconomic background do not affect the occurrence of autism.^[346]

History

Society and culture

An autistic culture has emerged, accompanied by the autistic rights and neurodiversity movements, that argues autism should be accepted as a difference to be accommodated instead of cured,^{[347][348][349][350][351]} although a minority of autistic people might still accept a cure.^[352] Worldwide, events related to autism include World Autism Awareness Day, Autism Sunday, Autistic Pride Day, Autreat, and others.^{[353][354][355][356]}

Social-science scholars study those with autism in hopes to learn more about "autism as a culture, transcultural comparisons ... and research on social movements."^[357] Many autistic people have been successful in their fields.^[358]

Neurodiversity movement

Some autistic people, as well as a growing number of researchers,^[13] have advocated a shift in attitudes toward the view that autism spectrum disorder is a difference, rather than a disease that must be treated or cured.^{[359][360]} Critics have bemoaned the entrenchment of some of these groups' opinions.^{[361][362][363][364]}

The neurodiversity movement and the autism rights movement are social movements within the context of disability rights, emphasizing the concept of neurodiversity, which describes the autism spectrum as a result of natural variations in the human brain rather than a disorder to be cured.^{[349][108]} The autism rights movement advocates including greater acceptance of autistic behaviors; therapies that focus on coping skills rather than imitating the behaviors of those without autism;^[365] and the recognition of the autistic community as a minority group.^{[365][366]}

Autism rights or neurodiversity advocates believe that the autism spectrum is genetic and should be accepted as a natural variation in the human genome.^[349] These movements are not without detractors; a common argument against neurodiversity activists is that most of them have relatively low support needs, or are self-diagnosed, and do not represent the views of autistic people with higher support needs.^{[366][367][368]} Jacqueline den Houting explores this critique, determining that the voices of low-support needs autistics are "some of the most influential within the neurodiversity movement, although admittedly these voices are a minority within the advocacy community"; she suggests this is in part a shortcoming of the wider neurotypical community, referencing non-speaking self-advocate Amy Sequenzia's writing.^{[369][370]} Pier Jaarsma and Stellan Welin make the argument that only high-functioning autistic people should be included under the neurodiversity banner, as low-functioning autists' condition may rightfully be viewed as a disability.^[371] The concept of neurodiversity is contentious in autism advocacy and research groups and has led to infighting.^{[372][373]}

Events

Since 2011, the Autistic Self Advocacy Network has celebrated April as Autism Acceptance Month. In 2021, the Autism Society of America urged organizations to retitle Autism Awareness Day as Autism Acceptance Day, to focus on "more fully integrating those 1 in 54 Americans living with autism into our social fabric".^[374]

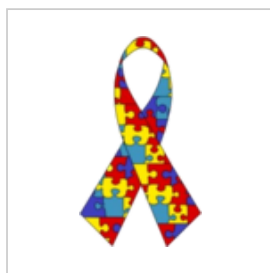
Symbols and flags

Symbols

Over the years, multiple organizations have tried to capture the essence of autism in symbols. In 1963, the board for the National Autistic Society, led by Gerald Gasson, proposed the "puzzle piece" as a symbol for autism, because it fit their view of autism as a "puzzling condition".^[375] In 1999, the Autism Society adopted the puzzle ribbon as the universal sign of autism awareness.^[375] As of 2023, the puzzle ribbon has negative associations, such as "implications of autistic people as incomplete and...association with autism hate groups".^[374]

In 2004, neurodiversity advocates Amy and Gwen Nelson conjured the "rainbow infinity symbol". It was initially the logo for their website, Aspies for Freedom. Nowadays, the prismatic colors are often associated with the neurodiversity movement in general.^[376] The autism spectrum has also been symbolized by the infinity symbol itself.^[377]

In 2018, Julian Morgan wrote the article "Light It Up Gold", a response to Autism Speaks's "Light It Up Blue" campaign, launched in 2007.^{[378][379]} *Aurum* is Latin for gold,^[376] and gold has been used to symbolize autism, since both words start with "Au". The flag implements a gradient to represent the Pride Movement and incorporates a golden infinity symbol as its focal point.^[380]



The puzzle piece symbol as used in the autism awareness ribbon used by Autism Speaks from 2005 and now has negative associations



Autism infinity symbol from 2013, featuring a rainbow gradient from left to right

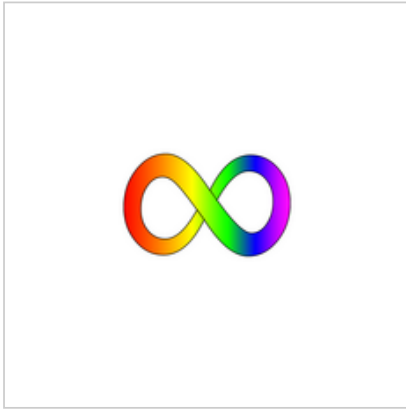


Gold infinity loop, following Julian Morgan's 2018 push to use gold for autism

Flags

An autistic pride flag was created in 2005 by Aspies for Freedom for the first Autistic Pride Day, featuring a rainbow infinity symbol on a white background.^[381]

As the rainbow infinity on a white background has become increasingly viewed as representative of neurodiversity in general,^[376] several designs have been proposed for an autistic-specific flag.^[382] In 2023, the People's History Museum featured a 2015 autistic pride design by Joseph Redford, featuring a rainbow infinity symbol, a green background for being true to one's nature, and a purple background for neurodiversity.^[383]



An autistic/neurodiversity pride flag featuring a rainbow infinity, based on a design from 2013



The 2015 autistic pride flag by Joseph Redford

Caregivers

Families who care for an autistic child face added stress from a number of different causes.^{[384][385]} Parents may struggle to understand the diagnosis and to find appropriate care options. They often take a negative view of the diagnosis, and may struggle emotionally.^[386] More than half of parents over age 50 are still living with their child, as about 85% of autistic people have difficulties living independently.^[387] Some studies also find decreased earnings among parents who care for autistic children.^{[388][389]} Siblings of children with ASD report greater admiration and less conflict with the affected sibling than siblings of unaffected children, like siblings of children with Down syndrome. But they reported lower levels of closeness and intimacy than siblings of children with Down syndrome; siblings of autistic people have a greater risk of negative well-being and poorer sibling relationships as adults.^[390]

See also

- Outline of autism
- Animal model of autism
- Autism and memory
- Autism spectrum disorders in the media
- Autistic art
- Controversies in autism
- Global perceptions of autism
- List of autistic fictional characters
- List of films about autism
- Violence and autism
- Employment for people with autism

Notes

a. Medical diagnosis term. See Classification.

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External links

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