[Template:Hatnote](/wiki/Template:Hatnote" \o "Template:Hatnote) [Template:Pp-semi-indef](/wiki/Template:Pp-semi-indef) [Template:Pp-move-indef](/wiki/Template:Pp-move-indef) [Template:Bots](/wiki/Template:Bots) [Template:Use dmy dates](/wiki/Template:Use_dmy_dates) [Template:Infobox medical condition](/wiki/Template:Infobox_medical_condition) **Autism** is a [neurodevelopmental disorder](/wiki/Neurodevelopmental_disorder) characterized by impaired [social interaction](/wiki/Interpersonal_relationship), [verbal](/wiki/Language_acquisition) and [non-verbal communication](/wiki/Non-verbal_communication), and restricted and repetitive behavior. Parents usually notice signs in the first two years of their child's life.<ref name=CCD/> These signs often develop gradually, though some children with autism reach their [developmental milestones](/wiki/Developmental_milestones) at a normal pace and then [regress](/wiki/Regressive_autism).<ref name=Stefanatos/> The [diagnostic criteria](/wiki/Diagnostic_and_Statistical_Manual_of_Mental_Disorders) require that symptoms become apparent in early childhood, typically before age three.<ref name=DSM5>[Template:Vcite book](/wiki/Template:Vcite_book)</ref>

While autism is highly heritable, researchers suspect both environmental and genetic factors as causes.[[1]](#cite_note-1) In rare cases, autism is strongly associated with [agents that cause birth defects](/wiki/Teratology).<ref name=Arndt/> [Controversies](/wiki/Controversies_in_autism) surround other proposed environmental [causes](/wiki/Causes_of_autism);<ref name=Rutter/> for example, the [vaccine hypotheses](/wiki/MMR_vaccine_controversy) have been disproven. Autism affects information processing in the [brain](/wiki/Human_brain) by altering how [nerve cells](/wiki/Nerve_cell) and their [synapses](/wiki/Synapse) connect and organize; how this occurs is not well understood.[[2]](#cite_note-2) In the [DSM V](/wiki/DSM_V) it is one of three recognized disorders in the [autism spectrum](/wiki/Autism_spectrum) (ASDs), the other two being [Asperger syndrome](/wiki/Asperger_syndrome), which lacks delays in cognitive development and language, and [pervasive developmental disorder, not otherwise specified](/wiki/PDD-NOS) (commonly abbreviated as PDD-NOS), which is diagnosed when the full set of criteria for autism or Asperger syndrome are not met.[[3]](#cite_note-3) Early speech or [behavioral interventions](/wiki/Early_intensive_behavioral_intervention) can help children with autism gain self-care, social, and communication skills.<ref name=CCD/> Although there is no known cure,<ref name=CCD/> there have been reported cases of children who recovered.<ref name=Helt/> Not many children with autism live independently after reaching adulthood, though some become successful.[[4]](#cite_note-4) An [autistic culture](/wiki/Sociological_and_cultural_aspects_of_autism) has developed, with some individuals seeking a cure and others believing autism should be [accepted as a difference and not treated as a disorder](/wiki/Autism_rights_movement).<ref name=Silverman/>

Globally, autism is estimated to affect 21.7 million people as of 2013.[[5]](#cite_note-5) As of 2010, the number of people affected is estimated at about 1–2 per 1,000 worldwide. It occurs four to five times more often in boys than girls. About 1.5% of children in the United States (one in 68) are diagnosed with ASD [Template:As of](/wiki/Template:As_of), a 30% increase from one in 88 in 2012.[[6]](#cite_note-6)[[7]](#cite_note-7)[[8]](#cite_note-8) The rate of autism among adults aged 18 years and over in the United Kingdom is 1.1%.<ref name=NHSEstimating/> The number of people diagnosed has been increasing dramatically since the 1980s, partly due to changes in diagnostic practice and government-subsidized financial incentives for named diagnoses;[[8]](#cite_note-8) the question of whether actual rates have increased is unresolved.<ref name=Newschaffer/>

## Contents

* 1 Characteristics[[edit](/index.php?title=(none)&action=edit&section=1)]
  + 1.1 Social development[[edit](/index.php?title=(none)&action=edit&section=2)]
  + 1.2 Communication[[edit](/index.php?title=(none)&action=edit&section=3)]
  + 1.3 Repetitive behavior[[edit](/index.php?title=(none)&action=edit&section=4)]
  + 1.4 Other symptoms[[edit](/index.php?title=(none)&action=edit&section=5)]
* 2 Causes[[edit](/index.php?title=(none)&action=edit&section=6)]
* 3 Mechanism[[edit](/index.php?title=(none)&action=edit&section=7)]
  + 3.1 Pathophysiology[[edit](/index.php?title=(none)&action=edit&section=8)]
  + 3.2 Neuropsychology[[edit](/index.php?title=(none)&action=edit&section=9)]
* 4 Diagnosis[[edit](/index.php?title=(none)&action=edit&section=10)]
  + 4.1 Classification[[edit](/index.php?title=(none)&action=edit&section=11)]
* 5 Screening[[edit](/index.php?title=(none)&action=edit&section=12)]
* 6 Prevention[[edit](/index.php?title=(none)&action=edit&section=13)]
* 7 Management[[edit](/index.php?title=(none)&action=edit&section=14)]
  + 7.1 Education[[edit](/index.php?title=(none)&action=edit&section=15)]
  + 7.2 Medication[[edit](/index.php?title=(none)&action=edit&section=16)]
  + 7.3 Alternative medicine[[edit](/index.php?title=(none)&action=edit&section=17)]
  + 7.4 Cost[[edit](/index.php?title=(none)&action=edit&section=18)]
* 8 Society and culture[[edit](/index.php?title=(none)&action=edit&section=19)]
* 9 Prognosis[[edit](/index.php?title=(none)&action=edit&section=20)]
* 10 Epidemiology[[edit](/index.php?title=(none)&action=edit&section=21)]
* 11 History[[edit](/index.php?title=(none)&action=edit&section=22)]
* 12 References[[edit](/index.php?title=(none)&action=edit&section=23)]
* 13 Further reading[[edit](/index.php?title=(none)&action=edit&section=24)]
* 14 External links[[edit](/index.php?title=(none)&action=edit&section=25)]

## Characteristics[[edit](/index.php?title=(none)&action=edit&section=1)]

[thumb|Autism spectrum disorder video](/wiki/File:Autism_spectrum_disorder_video.webm) Autism is a highly variable [neurodevelopmental disorder](/wiki/Neurodevelopmental_disorder)<ref name=Geschwind/> that first appears during infancy or childhood, and generally follows a steady course without [remission](/wiki/Remission_(medicine)).<ref name=ICD-10-F84.0/> People with autism may be severely impaired in some respects but normal, or even superior, in others.[[9]](#cite_note-9) Overt symptoms gradually begin after the age of six months, become established by age two or three years,[[10]](#cite_note-10) and tend to continue through adulthood, although often in more muted form.<ref name=Rapin/> It is distinguished not by a single symptom, but by a characteristic triad of symptoms: impairments in social interaction; impairments in communication; and restricted interests and repetitive behavior. Other aspects, such as atypical eating, are also common but are not essential for diagnosis.<ref name=Filipek/> Autism's individual symptoms occur in the general population and appear not to associate highly, without a sharp line separating pathologically severe from common traits.<ref name=London/>

### Social development[[edit](/index.php?title=(none)&action=edit&section=2)]

Social deficits distinguish autism and the related [autism spectrum disorders](/wiki/Autism_spectrum_disorder) (ASD; see [Classification](/wiki/#Classification)) from other developmental disorders.<ref name=Rapin/> People with autism have social impairments and often lack the intuition about others that many people take for granted. Noted autistic [Temple Grandin](/wiki/Temple_Grandin) described her inability to understand the [social communication](/wiki/Social_communication) of [neurotypicals](/wiki/Neurotypical), or people with normal [neural development](/wiki/Neural_development), as leaving her feeling "like an anthropologist on Mars".[[11]](#cite_note-11) Unusual social development becomes apparent early in childhood. Autistic infants show less attention to social stimuli, smile and look at others less often, and respond less to their own name. Autistic toddlers differ more strikingly from [social norms](/wiki/Social_norms); for example, they have less [eye contact](/wiki/Eye_contact) and [turn-taking](/wiki/Turn-taking), and do not have the ability to use simple movements to express themselves, such as pointing at things.<ref name=Volkmar/> Three- to five-year-old children with autism are less likely to exhibit social understanding, approach others spontaneously, imitate and respond to emotions, communicate nonverbally, and take turns with others. However, they do form [attachments](/wiki/Attachment_(psychology)) to their primary caregivers.[[12]](#cite_note-12) Most children with autism display moderately less [attachment security](/wiki/Attachment_in_children#Secure_attachment) than neurotypical children, although this difference disappears in children with higher mental development or less severe ASD.[[13]](#cite_note-13) Older children and adults with ASD [perform worse on tests of face and emotion recognition](/wiki/Face_perception#Face_perception_in_individuals_with_autism)[[14]](#cite_note-14) although this may be partly due to a lower ability to define a person's own emotions.[[15]](#cite_note-15) Children with high-functioning autism suffer from more intense and frequent loneliness compared to non-autistic peers, despite the common belief that children with autism prefer to be alone. Making and maintaining friendships often proves to be difficult for those with autism. For them, the quality of friendships, not the number of friends, predicts how lonely they feel. Functional friendships, such as those resulting in invitations to parties, may affect the quality of life more deeply.<ref name=Burgess/>

There are many anecdotal reports, but few systematic studies, of aggression and violence in individuals with ASD. The limited data suggest that, in children with intellectual disability, autism is associated with aggression, destruction of property, and [tantrums](/wiki/Tantrum).[[16]](#cite_note-16)

### Communication[[edit](/index.php?title=(none)&action=edit&section=3)]

About a third to a half of individuals with autism do not develop enough natural speech to meet their daily communication needs.[[17]](#cite_note-17) Differences in communication may be present from the first year of life, and may include delayed onset of [babbling](/wiki/Babbling), unusual gestures, diminished responsiveness, and vocal patterns that are not synchronized with the caregiver. In the second and third years, children with autism have less frequent and less diverse babbling, consonants, words, and word combinations; their gestures are less often integrated with words. Children with autism are less likely to make requests or share experiences, and are more likely to simply repeat others' words ([echolalia](/wiki/Echolalia))<ref name=Landa/><ref name=Tager-Flusberg/> or [reverse pronouns](/wiki/Pronoun_reversal).<ref name=Kanner1943/> [Joint attention](/wiki/Joint_attention) seems to be necessary for functional speech, and deficits in joint attention seem to distinguish infants with ASD:[[3]](#cite_note-3) for example, they may look at a pointing hand instead of the pointed-at object,<ref name=Volkmar/><ref name=Tager-Flusberg/> and they consistently fail to point at objects in order to comment on or share an experience.<ref name=Johnson/> Children with autism may have difficulty with imaginative play and with developing symbols into language.[[18]](#cite_note-18)[[19]](#cite_note-19) In a pair of studies, high-functioning children with autism aged 8–15 performed equally well as, and adults better than, individually matched controls at basic language tasks involving vocabulary and spelling. Both autistic groups performed worse than controls at complex language tasks such as figurative language, comprehension and inference. As people are often sized up initially from their basic language skills, these studies suggest that people speaking to autistic individuals are more likely to overestimate what their audience comprehends.<ref name=Williams/>

### Repetitive behavior[[edit](/index.php?title=(none)&action=edit&section=4)]

Autistic individuals display many forms of repetitive or restricted behavior, which the Repetitive Behavior Scale-Revised (RBS-R)<ref name=Lam-Aman/> categorizes as follows.

[thumb|alt=Sleeping boy beside a dozen or so toys arranged in a line|A young boy with autism who has arranged his toys in a row](/wiki/File:Autistic-sweetiepie-boy-with-ducksinarow.jpg)

* [**Stereotypy**](/wiki/Stereotypy) is repetitive movement, such as hand flapping, head rolling, or body rocking.
* [**Compulsive behavior**](/wiki/Compulsive_behavior) is intended and appears to follow rules, such as arranging objects in stacks or lines.
* **Sameness** is resistance to change; for example, insisting that the furniture not be moved or refusing to be interrupted.
* [**Ritualistic behavior**](/wiki/Ritual#Psychology) involves an 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.[[20]](#cite_note-20)\* **Restricted behavior** is limited in focus, interest, or activity, such as preoccupation with a single television program, toy or game.
* [**Self-injury**](/wiki/Self-injury) includes movements that injure or can injure the person, such as eye-poking, [skin-picking](/wiki/Dermatillomania), hand-biting and head-banging.<ref name=Johnson/>

No single repetitive or self-injurious behavior seems to be specific to autism, but autism appears to have an elevated pattern of occurrence and severity of these behaviors.[[21]](#cite_note-21)

### Other symptoms[[edit](/index.php?title=(none)&action=edit&section=5)]

Autistic individuals may have symptoms that are independent of the diagnosis, but that can affect the individual or the family.[[22]](#cite_note-22)An estimated 0.5% to 10% of individuals with ASD show unusual abilities, ranging from [splinter skills](/wiki/Splinter_skill) such as the memorization of trivia to the extraordinarily rare talents of prodigious [autistic savants](/wiki/Savant_syndrome).[[23]](#cite_note-23) Many individuals with ASD show superior skills in perception and attention, relative to the general population.[[24]](#cite_note-24) [Sensory](/wiki/Sensory_system) abnormalities are found in over 90% of those with autism, and are considered core features by some,[[25]](#cite_note-25) although there is no good evidence that sensory symptoms differentiate autism from other developmental disorders.[[26]](#cite_note-26) Differences 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).[[27]](#cite_note-27) An estimated 60%–80% of autistic people have motor signs that include [poor muscle tone](/wiki/Hypotonia), [poor motor planning](/wiki/Apraxia), and [toe walking](/wiki/Toe_walking);<ref name=Geschwind-2009/> deficits in motor coordination are pervasive across ASD and are greater in autism proper.[[28]](#cite_note-28) Unusual eating behavior occurs in about three-quarters of children with ASD, to the extent that it was formerly a diagnostic indicator. Selectivity is the most common problem, although eating rituals and food refusal also occur;[[29]](#cite_note-29) this does not appear to result in [malnutrition](/wiki/Malnutrition). Although some children with autism also have [gastrointestinal symptoms](/wiki/Gastrointestinal_symptom), there is a lack of published rigorous data to support the theory that children with autism have more or different gastrointestinal symptoms than usual;[[30]](#cite_note-30) studies report conflicting results, and the relationship between gastrointestinal problems and ASD is unclear.[[31]](#cite_note-31) Parents of children with ASD have higher levels of [stress](/wiki/Stress_(psychological)).[[32]](#cite_note-32) Siblings of children with ASD report greater admiration of and less conflict with the affected sibling than siblings of unaffected children and were similar to siblings of children with [Down syndrome](/wiki/Down_syndrome) in these aspects of the sibling relationship. However, they reported lower levels of closeness and intimacy than siblings of children with [Down syndrome](/wiki/Down_syndrome); siblings of individuals with ASD have greater risk of negative well-being and poorer sibling relationships as adults.[[33]](#cite_note-33)

## Causes[[edit](/index.php?title=(none)&action=edit&section=6)]

[Template:Main article](/wiki/Template:Main_article)

It has long been presumed that there is a common cause at the genetic, cognitive, and neural levels for autism's characteristic triad of symptoms.[[34]](#cite_note-34) However, there is increasing suspicion that autism is instead a complex disorder whose core aspects have distinct causes that often co-occur.[[34]](#cite_note-34)[[35]](#cite_note-35) [thumb|alt=Three diagrams of chromosome pairs A, B that are nearly identical. 1: B is missing a segment of A. 2: B has two adjacent copies of a segment of A. 3: B's copy of A's segment is in reverse order.|Deletion (1), duplication (2) and inversion (3) are all](/wiki/File:Single_Chromosome_Mutations.svg) [chromosome abnormalities](/wiki/Chromosome_abnormalities) that have been implicated in autism.[[36]](#cite_note-36) Autism has a strong genetic basis, although the [genetics of autism](/wiki/Heritability_of_autism) are complex and it is unclear whether ASD is explained more by rare [mutations](/wiki/Mutation) with major effects, or by rare multigene interactions of common genetic variants.[[37]](#cite_note-37)[[38]](#cite_note-38) Complexity arises due to interactions among multiple genes, the environment, and [epigenetic](/wiki/Epigenetic) factors which do not change [DNA](/wiki/DNA) sequencing but are heritable and influence [gene expression](/wiki/Gene_expression).[[39]](#cite_note-39) Many genes have been associated with autism through sequencing the genomes of affected individuals and their parents.[[40]](#cite_note-40) Studies of twins suggest that [heritability](/wiki/Heritability) is 0.7 for autism and as high as 0.9 for ASD, and siblings of those with autism are about 25 times more likely to be autistic than the general population.[[25]](#cite_note-25) However, most of the mutations that increase autism risk have not been identified. Typically, autism cannot be traced to a [Mendelian](/wiki/Mendelian) (single-gene) mutation or to a single [chromosome abnormality](/wiki/Chromosome_abnormality), and none of the genetic syndromes associated with ASDs have been shown to selectively cause ASD.[[37]](#cite_note-37) Numerous candidate genes have been located, with only small effects attributable to any particular gene.<ref name=Abrahams/> Most loci individually explain less than 1% of cases of autism.[[41]](#cite_note-41) The large number of autistic individuals with unaffected family members may result from spontaneous [structural variation](/wiki/Structural_variation) — such as [deletions](/wiki/Deletion_(genetics)), [duplications](/wiki/Gene_duplication) or [inversions](/wiki/Chromosomal_inversion) in genetic material during [meiosis](/wiki/Meiosis).[[42]](#cite_note-42)[[43]](#cite_note-43) 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.[[36]](#cite_note-36) Several lines of evidence point to [synaptic](/wiki/Synapse) dysfunction as a cause of autism.[[2]](#cite_note-2) Some rare mutations may lead to autism by disrupting some synaptic pathways, such as those involved with [cell adhesion](/wiki/Cell_adhesion).[[44]](#cite_note-44) Gene replacement studies in mice suggest that autistic symptoms are closely related to later developmental steps that depend on activity in synapses and on activity-dependent changes.[[45]](#cite_note-45) All known [teratogens](/wiki/Teratogen) (agents that cause [birth defects](/wiki/Birth_defect)) related to the risk of autism appear to act during the first eight weeks from [conception](/wiki/Human_fertilization), and though this does not exclude the possibility that autism can be initiated or affected later, there is strong evidence that autism arises very early in development.[[46]](#cite_note-46) Exposure to [air pollution](/wiki/Air_pollution) during pregnancy, especially [heavy metals](/wiki/Heavy_metal_(chemistry)) and particulates, may increase the risk of autism.[[47]](#cite_note-47) [Environmental factors](/wiki/Environmental_factor) that have been claimed to contribute to or exacerbate autism include certain foods, [infectious diseases](/wiki/Infectious_disease), [solvents](/wiki/Solvent), [diesel exhaust](/wiki/Diesel_exhaust), [PCBs](/wiki/PCBs), [phthalates](/wiki/Phthalates) and [phenols](/wiki/Phenols) used in plastic products, [pesticides](/wiki/Pesticide), [brominated flame retardants](/wiki/Brominated_flame_retardant), [alcohol](/wiki/Ethanol), smoking, [illicit drugs](/wiki/Illicit_drug), [vaccines](/wiki/Vaccine),[[48]](#cite_note-48) and [prenatal stress](/wiki/Prenatal_stress), but no evidence has been found for these claims, and some such as the MMR vaccine have been completely disproven.[[49]](#cite_note-49) Parents may first become aware of autistic symptoms in their child around the time of a routine vaccination. This has led to unsupported theories blaming [vaccine "overload"](/wiki/Vaccine_controversy#Vaccine_overload), a [vaccine preservative](/wiki/Thiomersal_controversy), or the [MMR vaccine](/wiki/MMR_vaccine_controversy) for causing autism.[[50]](#cite_note-50) The latter theory was supported by a litigation-funded study that has since been shown to have been "an elaborate fraud".[[51]](#cite_note-51) Although these theories lack convincing scientific evidence and are biologically implausible,[[50]](#cite_note-50) parental concern about a potential vaccine link with autism has led to lower rates of [childhood immunizations](/wiki/Childhood_immunizations), [outbreaks of previously controlled childhood diseases](/wiki/MMR_vaccine_controversy#Disease_outbreaks) in some countries, and the preventable deaths of several children.[[52]](#cite_note-52)[[53]](#cite_note-53)

## Mechanism[[edit](/index.php?title=(none)&action=edit&section=7)]

Autism's symptoms result from maturation-related changes in various systems of the brain. How autism occurs is not well understood. Its mechanism can be divided into two areas: the [pathophysiology](/wiki/Pathophysiology) of brain structures and processes associated with autism, and the [neuropsychological](/wiki/Neuropsychological) linkages between brain structures and behaviors.[[54]](#cite_note-54) The behaviors appear to have multiple pathophysiologies.<ref name=London/>

### Pathophysiology[[edit](/index.php?title=(none)&action=edit&section=8)]

[thumb|alt=Two diagrams of major brain structures implicated in autism. The upper diagram shows the cerebral cortex near the top and the basal ganglia in the center, just above the amygdala and hippocampus. The lower diagram shows the corpus callosum near the center, the cerebellum in the lower rear, and the brain stem in the lower center.|Autism affects the](/wiki/File:Autismbrain.jpg) [amygdala](/wiki/Amygdala), [cerebellum](/wiki/Cerebellum), and many other parts of the brain.<ref name=Amaral/> Unlike many other brain disorders, such as [Parkinson's](/wiki/Parkinson's), autism does not have a clear unifying mechanism at either the molecular, cellular, or systems level; it is not known whether autism is a few disorders caused by mutations converging on a few common molecular pathways, or is (like intellectual disability) a large set of disorders with diverse mechanisms.[[55]](#cite_note-55) Autism appears to result from developmental factors that affect many or all functional brain systems,[[56]](#cite_note-56) and to disturb the timing of brain development more than the final product.[[57]](#cite_note-57) [Neuroanatomical](/wiki/Neuroanatomical) studies and the associations with [teratogens](/wiki/Teratogens) strongly suggest that autism's mechanism includes alteration of brain development soon after conception.<ref name=Arndt/> This anomaly appears to start a cascade of pathological events in the brain that are significantly influenced by environmental factors.[[58]](#cite_note-58) Just after birth, the brains of children with autism tend to grow faster than usual, followed by normal or relatively slower growth in childhood. It is not known whether early overgrowth occurs in all children with autism. It seems to be most prominent in brain areas underlying the development of higher cognitive specialization.<ref name=Geschwind-2009/> Hypotheses for the cellular and molecular bases of pathological early overgrowth include the following:

* An excess of [neurons](/wiki/Neuron) that causes local overconnectivity in key brain regions.[[59]](#cite_note-59)\* Disturbed [neuronal migration](/wiki/Neuronal_migration) during early [gestation](/wiki/Gestation).[[60]](#cite_note-60)[[61]](#cite_note-61)\* Unbalanced excitatory–inhibitory networks.<ref name=Persico/>
* Abnormal formation of [synapses](/wiki/Synapse) and [dendritic spines](/wiki/Dendritic_spine),<ref name=Persico/> for example, by modulation of the [neurexin](/wiki/Neurexin)–[neuroligin](/wiki/Neuroligin) [cell-adhesion](/wiki/Cell_adhesion) system,[[62]](#cite_note-62) or by poorly regulated [synthesis](/wiki/Protein_synthesis) of synaptic proteins.[[63]](#cite_note-63)[[64]](#cite_note-64) Disrupted synaptic development may also contribute to [epilepsy](/wiki/Epilepsy), which may explain why the two conditions are associated.[[65]](#cite_note-65)

The [immune system](/wiki/Immune_system) is thought to play an important role in autism. Children with autism have been found by researchers to have [inflammation](/wiki/Inflammation) of both the peripheral and central immune systems as indicated by increased levels of pro-inflammatory [cytokines](/wiki/Cytokines) and significant activation of [microglia](/wiki/Microglia).[[66]](#cite_note-66)[[67]](#cite_note-67)[[68]](#cite_note-68) Biomarkers of abnormal immune function have also been associated with increased impairments in behaviors that are characteristic of the core features of autism such as deficits in social interactions and communication.[[67]](#cite_note-67) Interactions between the [immune system](/wiki/Immune_system) and the [nervous system](/wiki/Nervous_system) begin early during the [embryonic stage](/wiki/Human_embryogenesis) of life, and successful neurodevelopment depends on a balanced immune response. It is thought that activation of a pregnant mother's immune system such as from environmental toxicants or infection can contribute to causing autism through causing a disruption of brain development.[[69]](#cite_note-69)[[70]](#cite_note-70)[[71]](#cite_note-71) This is supported by recent studies that have found that infection during pregnancy is associated with an increased risk of autism.[[72]](#cite_note-72)[[73]](#cite_note-73) The relationship of [neurochemicals](/wiki/Neurochemical) to autism is not well understood; several have been investigated, with the most evidence for the role of [serotonin](/wiki/Serotonin) and of genetic differences in its transport.[[2]](#cite_note-2) The role of group I [metabotropic glutamate receptors](/wiki/Metabotropic_glutamate_receptors) (mGluR) in the pathogenesis of [fragile X syndrome](/wiki/Fragile_X_syndrome), the most common identified genetic cause of autism, has led to interest in the possible implications for future autism research into this pathway.[[74]](#cite_note-74) Some data suggests neuronal overgrowth potentially related to an increase in several [growth hormones](/wiki/Growth_hormone)[[75]](#cite_note-75) or to impaired regulation of [growth factor receptors](/wiki/Growth_factor_receptor). Also, some [inborn errors of metabolism](/wiki/Inborn_errors_of_metabolism) are associated with autism, but probably account for less than 5% of cases.<ref name=Manzi/>

The [mirror neuron system](/wiki/Mirror_neuron_system) (MNS) theory of autism hypothesizes that distortion in the development of the MNS interferes with imitation and leads to autism's core features of social impairment and communication difficulties. The MNS operates when an animal performs an action or observes another animal perform the same action. The MNS may contribute to an individual's understanding of other people by enabling the modeling of their behavior via embodied simulation of their actions, intentions, and emotions.[[76]](#cite_note-76) Several studies have tested this hypothesis by demonstrating structural abnormalities in MNS regions of individuals with ASD, delay in the activation in the core circuit for imitation in individuals with Asperger syndrome, and a correlation between reduced MNS activity and severity of the syndrome in children with ASD.[[77]](#cite_note-77) However, individuals with autism also have abnormal brain activation in many circuits outside the MNS[[78]](#cite_note-78) and the MNS theory does not explain the normal performance of children with autism on imitation tasks that involve a goal or object.[[79]](#cite_note-79) [thumb|upright|alt=A human brain viewed from above. About 10% is highlighted in yellow and 10% in blue. There is only a tiny (perhaps 0.5%) green region where they overlap.|Autistic individuals tend to use different areas of the brain (yellow) for a movement task compared to a control group (blue).](/wiki/File:Powell2004Fig1A.jpeg)[[80]](#cite_note-80) ASD-related patterns of low function and aberrant activation in the brain differ depending on whether the brain is doing social or nonsocial tasks.[[81]](#cite_note-81)In autism there is evidence for reduced functional connectivity of the [default network](/wiki/Default_network), a large-scale brain network involved in social and emotional processing, with intact connectivity of the [task-positive network](/wiki/Task-positive_network), used in sustained attention and goal-directed thinking[Template:Clarify](/wiki/Template:Clarify). In people with autism the two networks are not negatively correlated in time, suggesting an imbalance in toggling between the two networks, possibly reflecting a disturbance of [self-referential](/wiki/Self-referential) thought.[[82]](#cite_note-82) The underconnectivity theory of autism hypothesizes that autism is marked by underfunctioning high-level neural connections and synchronization, along with an excess of low-level processes.[[83]](#cite_note-83) Evidence for this theory has been found in [functional neuroimaging](/wiki/Functional_neuroimaging) studies on autistic individuals[[84]](#cite_note-84) and by a [brainwave](/wiki/Electroencephalography) study that suggested that adults with ASD have local overconnectivity in the [cortex](/wiki/Cerebral_cortex) and weak functional connections between the [frontal lobe](/wiki/Frontal_lobe) and the rest of the cortex.[[85]](#cite_note-85) Other evidence suggests the underconnectivity is mainly within each [hemisphere](/wiki/Cerebral_hemisphere) of the cortex and that autism is a disorder of the [association cortex](/wiki/Association_areas).[[86]](#cite_note-86) From studies based on [event-related potentials](/wiki/Event-related_potential), transient changes to the brain's electrical activity in response to stimuli, there is considerable evidence for differences in autistic individuals with respect to attention, orientation to auditory and visual stimuli, novelty detection, language and face processing, and information storage; several studies have found a preference for nonsocial stimuli.[[87]](#cite_note-87) For example, [magnetoencephalography](/wiki/Magnetoencephalography) studies have found evidence in children with autism of delayed responses in the brain's processing of auditory signals.[[88]](#cite_note-88) In the genetic area, relations have been found between autism and [schizophrenia](/wiki/Schizophrenia) based on duplications and deletions of chromosomes; research showed that schizophrenia and autism are significantly more common in combination with [1q21.1 deletion syndrome](/wiki/1q21.1_deletion_syndrome). Research on autism/schizophrenia relations for chromosome 15 (15q13.3), chromosome 16 (16p13.1) and chromosome 17 (17p12) are inconclusive.[[89]](#cite_note-89) Functional connectivity studies have found both hypo- and hyper-connectivity in brains of people with autism. Hypo-connectivity seems to dominate, especially for interhemispheric and cortico-cortical functional connectivity.<ref name=HaSohn2015>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

### Neuropsychology[[edit](/index.php?title=(none)&action=edit&section=9)]

Two major categories of [cognitive](/wiki/Cognitive) theories have been proposed about the links between autistic brains and behavior.

The first category focuses on deficits in [social cognition](/wiki/Social_cognition). [Simon Baron-Cohen's](/wiki/Simon_Baron-Cohen) [empathizing–systemizing theory](/wiki/Empathizing–systemizing_theory) postulates that autistic individuals can systemize—that is, they can develop internal rules of operation to handle events inside the brain—but are less effective at empathizing by handling events generated by other agents. An extension, the extreme male brain theory, hypothesizes that autism is an extreme case of the male brain, defined psychometrically as individuals in whom systemizing is better than empathizing.<ref name=E-S-theory/> These theories are somewhat related to Baron-Cohen's earlier [theory of mind](/wiki/Theory_of_mind) approach, which hypothesizes that autistic behavior arises from an inability to ascribe mental states to oneself and others. The theory of mind hypothesis is supported by the atypical responses of children with autism to the [Sally–Anne test](/wiki/Sally–Anne_test) for reasoning about others' motivations,[[90]](#cite_note-90) and the mirror neuron system theory of autism described in [*Pathophysiology*](/wiki/#Pathophysiology) maps well to the hypothesis.<ref name=Iacoboni/> However, most studies have found no evidence of impairment in autistic individuals' ability to understand other people's basic intentions or goals; instead, data suggests that impairments are found in understanding more complex social emotions or in considering others' viewpoints.[[91]](#cite_note-91) The second category focuses on nonsocial or general processing: the [executive functions](/wiki/Executive_functions) such as [working memory](/wiki/Working_memory), planning, [inhibition](/wiki/Inhibition_Theory). In his review, Kenworthy states that "the claim of [executive dysfunction](/wiki/Executive_dysfunction) as a causal factor in autism is controversial", however, "it is clear that executive dysfunction plays a role in the social and cognitive deficits observed in individuals with autism".<ref name=Kenworthy/> Tests of core executive processes such as eye movement tasks indicate improvement from late childhood to adolescence, but performance never reaches typical adult levels.[[92]](#cite_note-92) A strength of the theory is predicting stereotyped behavior and narrow interests;[[93]](#cite_note-93) two weaknesses are that executive function is hard to measure[[94]](#cite_note-94) and that executive function deficits have not been found in young children with autism.<ref name=Sigman/>

[Weak central coherence theory](/wiki/Weak_central_coherence_theory) hypothesizes that a limited ability to see the big picture underlies the central disturbance in autism. One strength of this theory is predicting special talents and peaks in performance in autistic people.[[95]](#cite_note-95) A related theory—enhanced perceptual functioning—focuses more on the superiority of locally oriented and [perceptual](/wiki/Perceptual) operations in autistic individuals.[[96]](#cite_note-96) These theories map well from the underconnectivity theory of autism.

Neither category is satisfactory on its own; social cognition theories poorly address autism's rigid and repetitive behaviors, while the nonsocial theories have difficulty explaining social impairment and communication difficulties.[[35]](#cite_note-35) A combined theory based on multiple deficits may prove to be more useful.[[97]](#cite_note-97)

## Diagnosis[[edit](/index.php?title=(none)&action=edit&section=10)]

[Diagnosis](/wiki/Medical_diagnosis) is based on behavior, not cause or mechanism.[[98]](#cite_note-98)[[99]](#cite_note-99) Under the [DSM-5](/wiki/DSM-5), autism is characterized by persistent deficits in social communication and interaction across multiple contexts, as well as restricted, repetitive patterns of behavior, interests, or activities. These deficits are present in early childhood, typically before age three, and lead to clinically significant functional impairment. Sample symptoms include lack of social or emotional reciprocity, stereotyped and repetitive use of language or [idiosyncratic language](/wiki/Idiosyncrasy#Psychiatry_and_psychology), and persistent preoccupation with unusual objects. The disturbance must not be better accounted for by [Rett syndrome](/wiki/Rett_syndrome), [intellectual disability](/wiki/Intellectual_disability) or global developmental delay.<ref name=DSM5/> [ICD-10](/wiki/ICD-10) uses essentially the same definition.[[100]](#cite_note-100) Several diagnostic instruments are available. Two are commonly used in autism research: the [Autism Diagnostic Interview-Revised](/wiki/Autism_Diagnostic_Interview-Revised) (ADI-R) is a semistructured parent interview, and the [Autism Diagnostic Observation Schedule](/wiki/Autism_Diagnostic_Observation_Schedule) (ADOS)[[101]](#cite_note-101) uses observation and interaction with the child. The [Childhood Autism Rating Scale](/wiki/Childhood_Autism_Rating_Scale) (CARS) is used widely in clinical environments to assess severity of autism based on observation of children.<ref name=Volkmar/>

A [pediatrician](/wiki/Pediatrician) commonly performs a preliminary investigation by taking developmental history and physically examining the child. If warranted, diagnosis and evaluations are conducted with help from ASD specialists, observing and assessing cognitive, communication, family, and other factors using standardized tools, and taking into account any associated [medical conditions](/wiki/Medical_conditions).<ref name=Dover/> A pediatric [neuropsychologist](/wiki/Neuropsychologist) is often asked to assess behavior and cognitive skills, both to aid diagnosis and to help recommend educational interventions.[[102]](#cite_note-102) A [differential diagnosis](/wiki/Differential_diagnosis) for ASD at this stage might also consider [intellectual disability](/wiki/Intellectual_disability), [hearing impairment](/wiki/Hearing_impairment), and a [specific language impairment](/wiki/Specific_language_impairment)<ref name=Dover/> such as [Landau–Kleffner syndrome](/wiki/Landau–Kleffner_syndrome).[[103]](#cite_note-103) The presence of autism can make it harder to diagnose coexisting psychiatric disorders such as [depression](/wiki/Major_depressive_disorder).[[104]](#cite_note-104) [Clinical genetics](/wiki/Clinical_genetics) evaluations are often done once ASD is diagnosed, particularly when other symptoms already suggest a genetic cause.<ref name=Caronna/> Although genetic technology allows clinical geneticists to link an estimated 40% of cases to genetic causes,[[105]](#cite_note-105) consensus guidelines in the US and UK are limited to high-resolution chromosome and [fragile X](/wiki/Fragile_X) testing.<ref name=Caronna/> A [genotype-first](/wiki/Genotype-first_approach) model of diagnosis has been proposed, which would routinely assess the genome's copy number variations.[[106]](#cite_note-106) As new genetic tests are developed several ethical, legal, and social issues will emerge. Commercial availability of tests may precede adequate understanding of how to use test results, given the complexity of autism's genetics.[[107]](#cite_note-107) [Metabolic](/wiki/Metabolic) and [neuroimaging](/wiki/Neuroimaging) tests are sometimes helpful, but are not routine.<ref name=Caronna/>

ASD can sometimes be diagnosed by age 14 months, although diagnosis becomes increasingly stable over the first three years of life: for example, a one-year-old who meets diagnostic criteria for ASD is less likely than a three-year-old to continue to do so a few years later.[[108]](#cite_note-108) In the UK the National Autism Plan for Children recommends at most 30 weeks from first concern to completed diagnosis and assessment, though few cases are handled that quickly in practice.[[109]](#cite_note-109) Although the symptoms of autism and ASD begin early in childhood, they are sometimes missed; years later, adults may seek diagnoses to help them or their friends and family understand themselves, to help their employers make adjustments, or in some locations to claim disability living allowances or other benefits.

Underdiagnosis and overdiagnosis are problems in marginal cases, and much of the recent increase in the number of reported ASD cases is likely due to changes in diagnostic practices. The increasing popularity of drug treatment options and the expansion of benefits has given providers incentives to diagnose ASD, resulting in some overdiagnosis of children with uncertain symptoms. Conversely, the cost of screening and diagnosis and the challenge of obtaining payment can inhibit or delay diagnosis.[[110]](#cite_note-110) It is particularly hard to diagnose autism among the [visually impaired](/wiki/Visually_impaired), partly because some of its diagnostic criteria depend on vision, and partly because autistic symptoms overlap with those of common blindness syndromes or [blindisms](/wiki/Blindism).[[111]](#cite_note-111)

### Classification[[edit](/index.php?title=(none)&action=edit&section=11)]

Autism is one of the five [pervasive developmental disorders](/wiki/Pervasive_developmental_disorder) (PDD), which are characterized by widespread abnormalities of social interactions and communication, and severely restricted interests and highly repetitive behavior.<ref name=ICD-10-F84.0/> These symptoms do not imply sickness, fragility, or emotional disturbance.<ref name=Rapin/>

Of the five PDD forms, [Asperger syndrome](/wiki/Asperger_syndrome) is closest to autism in signs and likely causes; [Rett syndrome](/wiki/Rett_syndrome) and [childhood disintegrative disorder](/wiki/Childhood_disintegrative_disorder) share several signs with autism, but may have unrelated causes; [PDD not otherwise specified](/wiki/PDD_not_otherwise_specified) (PDD-NOS; also called *atypical autism*) is diagnosed when the criteria are not met for a more specific disorder.[[112]](#cite_note-112) Unlike with autism, people with Asperger syndrome have no substantial delay in [language development](/wiki/Language_development).[[113]](#cite_note-113) The terminology of autism can be bewildering, with autism, Asperger syndrome and PDD-NOS often called the *autism spectrum disorders* (ASD)<ref name=CCD/> or sometimes the *autistic disorders*,[[114]](#cite_note-114) whereas autism itself is often called *autistic disorder*, *childhood autism*, or *infantile autism*. In this article, *autism* refers to the classic autistic disorder; in clinical practice, though, *autism*, *ASD*, and *PDD* are often used interchangeably.[[115]](#cite_note-115) ASD, in turn, is a subset of the broader autism [phenotype](/wiki/Phenotype), which describes individuals who may not have ASD but do have autistic-like [traits](/wiki/Trait_(biology)), such as avoiding eye contact.[[116]](#cite_note-116) The manifestations of autism cover a wide [spectrum](/wiki/Spectrum_disorder), ranging from individuals with severe impairments—who may be silent, [developmentally disabled](/wiki/Developmentally_disabled), and locked into hand flapping and rocking—to high functioning individuals who may have active but distinctly odd social approaches, narrowly focused interests, and verbose, [pedantic](/wiki/Pedantic) communication.[[117]](#cite_note-117) Because the behavior spectrum is continuous, boundaries between diagnostic categories are necessarily somewhat arbitrary.<ref name=Geschwind-2009/> Sometimes the syndrome is divided into low-, medium- or [high-functioning autism](/wiki/High-functioning_autism) (LFA, MFA, and HFA), based on [IQ](/wiki/IQ) thresholds,[[118]](#cite_note-118) or on how much support the individual requires in daily life; these subdivisions are not standardized and are controversial. Autism can also be divided into [syndromal](/wiki/Syndrome) and non-syndromal autism; the syndromal autism is associated with severe or profound [intellectual disability](/wiki/Intellectual_disability) or a congenital syndrome with physical symptoms, such as [tuberous sclerosis](/wiki/Tuberous_sclerosis).[[119]](#cite_note-119) Although individuals with Asperger syndrome tend to perform better cognitively than those with autism, the extent of the [overlap between Asperger syndrome, HFA, and non-syndromal autism](/wiki/Diagnosis_of_Asperger_syndrome#Differences_from_high-functioning_autism) is unclear.[[120]](#cite_note-120) Some studies have reported diagnoses of autism in children due to a loss of language or social skills, as opposed to a failure to make progress, typically from 15 to 30 months of age. The validity of this distinction remains controversial; it is possible that [regressive autism](/wiki/Regressive_autism) is a specific subtype,[[121]](#cite_note-121)<ref name=Landa/><ref name=Landa3/>[[122]](#cite_note-122) or that there is a continuum of behaviors between autism with and without regression.[[123]](#cite_note-123) Research into causes has been hampered by the inability to identify biologically meaningful subgroups within the autistic population[[124]](#cite_note-124) and by the traditional boundaries between the disciplines of [psychiatry](/wiki/Psychiatry), [psychology](/wiki/Psychology), [neurology](/wiki/Neurology) and [pediatrics](/wiki/Pediatrics).[[125]](#cite_note-125) Newer technologies such as [fMRI](/wiki/FMRI) and [diffusion tensor imaging](/wiki/Diffusion_tensor_imaging) can help identify biologically relevant [phenotypes](/wiki/Phenotype) (observable traits) that can be viewed on [brain scans](/wiki/Brain_scan), to help further [neurogenetic](/wiki/Neurogenetic) studies of autism;[[126]](#cite_note-126) one example is lowered activity in the [fusiform face area](/wiki/Fusiform_face_area) of the brain, which is associated with impaired perception of people versus objects.<ref name=Levy/> It has been proposed to classify autism using genetics as well as behavior.[[127]](#cite_note-127)

## Screening[[edit](/index.php?title=(none)&action=edit&section=12)]

About half of parents of children with ASD notice their child's unusual behaviors by age 18 months, and about four-fifths notice by age 24 months.<ref name=Landa3/> According to an article failure to meet any of the following milestones "is an absolute indication to proceed with further evaluations. Delay in referral for such testing may delay early diagnosis and treatment and affect the long-term outcome".<ref name=Filipek/>

* No [babbling](/wiki/Babbling) by 12 months.
* No [gesturing](/wiki/Gesture) (pointing, waving, etc.) by 12 months.
* No single words by 16 months.
* No two-word (spontaneous, not just [echolalic](/wiki/Echolalia)) phrases by 24 months.
* Any loss of any language or social skills, at any age.

The [United States Preventative Services Task Force](/wiki/United_States_Preventative_Services_Task_Force) in 2016 found it was unclear if screening was beneficial or harmful among children in whom there is no concerns.[[128]](#cite_note-128) The Japanese practice is to [screen](/wiki/Screening_(medicine)) all children for ASD at 18 and 24 months, using autism-specific formal screening tests. In contrast, in the UK, children whose families or doctors recognize possible signs of autism are screened. It is not known which approach is more effective.<ref name=Levy/> Screening tools include the [Modified Checklist for Autism in Toddlers](/wiki/Modified_Checklist_for_Autism_in_Toddlers) (M-CHAT), the Early Screening of Autistic Traits Questionnaire, and the First Year Inventory; initial data on [M-CHAT](/wiki/Modified_Checklist_for_Autism_in_Toddlers) and its predecessor, the [Checklist for Autism in Toddlers](/wiki/Checklist_for_Autism_in_Toddlers) (CHAT), on children aged 18–30 months suggests that it is best used in a clinical setting and that it has low [sensitivity](/wiki/Sensitivity_(tests)) (many false-negatives) but good [specificity](/wiki/Specificity_(tests)) (few false-positives).<ref name=Landa3/> It may be more accurate to precede these tests with a broadband screener that does not distinguish ASD from other developmental disorders.[[129]](#cite_note-129) Screening tools designed for one culture's norms for behaviors like eye contact may be inappropriate for a different culture.[[130]](#cite_note-130) Although [genetic screening](/wiki/Genetic_screening) for autism is generally still impractical, it can be considered in some cases, such as children with neurological symptoms and [dysmorphic features](/wiki/Dysmorphic_feature).[[131]](#cite_note-131)

## Prevention[[edit](/index.php?title=(none)&action=edit&section=13)]

Infection with [rubella](/wiki/Rubella) during [pregnancy](/wiki/Pregnancy) causes fewer than 1% of cases of autism;<ref name=Duchan/> [vaccination against rubella](/wiki/Rubella_vaccine) can prevent many of those cases.<ref name=Lancet2015>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

## Management[[edit](/index.php?title=(none)&action=edit&section=14)]

[Template:Main article](/wiki/Template:Main_article) [thumb|alt=A young child points, in front of a woman who smiles and points in the same direction.|A three-year-old with autism points to fish in an aquarium, as part of an experiment on the effect of intensive shared-attention training on language development.<ref name=Powell/>](/wiki/File:Opening_a_window_to_the_autistic_brain.jpg) The main goals when treating children with autism 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.[[132]](#cite_note-132)[[133]](#cite_note-133) No single treatment is best and treatment is typically tailored to the child's needs.<ref name=CCD/> Families and the educational system are the main resources for treatment.<ref name=Levy/> Studies of interventions have methodological problems that prevent definitive conclusions about [efficacy](/wiki/Efficacy),[[134]](#cite_note-134) however the development of evidence-based interventions has advanced in recent years.[[132]](#cite_note-132) Although many [psychosocial](/wiki/Psychosocial) interventions have some positive evidence, suggesting that some form of treatment is preferable to no treatment, the methodological quality of [systematic reviews](/wiki/Systematic_review) of these studies has generally been poor, their clinical results are mostly tentative, and there is little evidence for the relative effectiveness of treatment options.[[135]](#cite_note-135) Intensive, sustained [special education](/wiki/Special_education) programs and [behavior therapy](/wiki/Behavior_therapy) early in life can help children acquire self-care, social, and job skills,[[136]](#cite_note-136) and often improve functioning and decrease symptom severity and maladaptive behaviors;[[137]](#cite_note-137) claims that intervention by around age three years is crucial are not substantiated.[[138]](#cite_note-138) Available approaches include [applied behavior analysis](/wiki/Applied_behavior_analysis) (ABA), developmental models, [structured teaching](/wiki/TEACCH), [speech and language therapy](/wiki/Speech_and_language_therapy), [social skills](/wiki/Social_skills) therapy, and [occupational therapy](/wiki/Occupational_therapy).<ref name=CCD/> Among these approaches, interventions either treat autistic features comprehensively, or focalize treatment on a specific area of deficit.[[132]](#cite_note-132) There is some evidence that early intensive behavioral intervention (EIBI), an early intervention model based on ABA for 20 to 40 hours a week for multiple years, is an effective treatment for some children with ASD.[[139]](#cite_note-139) Two theoretical frameworks outlined for early childhood intervention include applied behavioral analysis (ABA) and developmental social pragmatic models (DSP).[[132]](#cite_note-132) 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.[[132]](#cite_note-132) 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.[[132]](#cite_note-132)

### Education[[edit](/index.php?title=(none)&action=edit&section=15)]

Educational interventions can be effective to varying degrees in most children: [intensive ABA treatment](/wiki/Early_intensive_behavioral_intervention) has demonstrated effectiveness in enhancing global functioning in preschool children[[140]](#cite_note-140) and is well-established for improving intellectual performance of young children.<ref name=Rogers/> Similarly, teacher-implemented intervention that utilizes an ABA combined with a developmental social pragmatic approach has been found to be a well-established treatment in improving social-communication skills in young children, although there is less evidence in its treatment of global symptoms.[[132]](#cite_note-132) Neuropsychological reports are often poorly communicated to educators, resulting in a gap between what a report recommends and what education is provided.<ref name=Kanne/> It is not known whether treatment programs for children lead to significant improvements after the children grow up,<ref name=Rogers/> and the limited research on the effectiveness of adult residential programs shows mixed results.[[141]](#cite_note-141) 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.[[142]](#cite_note-142)

### Medication[[edit](/index.php?title=(none)&action=edit&section=16)]

Many medications are used to treat ASD symptoms that interfere with integrating a child into home or school when behavioral treatment fails.<ref name=Rapin/>[[143]](#cite_note-143) More than half of US children diagnosed with ASD are prescribed [psychoactive drugs](/wiki/Psychoactive_drug) or [anticonvulsants](/wiki/Anticonvulsant), with the most common drug classes being [antidepressants](/wiki/Antidepressant), [stimulants](/wiki/Stimulant), and [antipsychotics](/wiki/Antipsychotic).[[144]](#cite_note-144) Antipsychotics, such as [risperidone](/wiki/Risperidone) and [aripiprazole](/wiki/Aripiprazole), have been found to be useful for treating irritability, repetitive behavior, and sleeplessness that often occurs with autism, however their side effects must be weighed against their potential benefits, and people with autism may respond atypically.[[145]](#cite_note-145) There is scant reliable research about the effectiveness or safety of drug treatments for adolescents and adults with ASD.[[146]](#cite_note-146) No known medication relieves autism's core symptoms of social and communication impairments.[[147]](#cite_note-147) Experiments in mice have reversed or reduced some symptoms related to autism by replacing or modulating gene function,[[74]](#cite_note-74) suggesting the possibility of targeting therapies to specific rare mutations known to cause autism.[[148]](#cite_note-148)

### Alternative medicine[[edit](/index.php?title=(none)&action=edit&section=17)]

Although many [alternative therapies and interventions](/wiki/Alternative_therapies_for_developmental_and_learning_disabilities) are available, few are supported by scientific studies.<ref name=Sigman/>[[149]](#cite_note-149) Treatment approaches have little empirical support in [quality-of-life](/wiki/Quality_of_life) contexts, and many programs focus on success measures that lack predictive validity and real-world relevance.<ref name=Burgess/> Scientific evidence appears to matter less to service providers than program marketing, training availability, and parent requests.[[150]](#cite_note-150) Some alternative treatments may place the child at risk. A 2008 study found that compared to their peers, autistic boys have significantly thinner bones if on [casein-free diets](/wiki/Casein-free_diet);[[151]](#cite_note-151) in 2005, botched [chelation therapy](/wiki/Chelation_therapy) killed a five-year-old child with autism.[[152]](#cite_note-152) There has been early research looking at [hyperbaric treatments](/wiki/Hyperbaric_medicine) in children with autism.[[153]](#cite_note-153) Although popularly used as an [alternative treatment](/wiki/Complementary_and_alternative_medicine) for people with autism, there is no good evidence that a [gluten-free diet](/wiki/Gluten-free_diet) is of benefit.<ref name=Buie>[Template:Cite journal](/wiki/Template:Cite_journal)</ref><ref name=MariBausetZazpe>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>[[154]](#cite_note-154) In the subset of people who have [gluten sensitivity](/wiki/Non-celiac_gluten_sensitivity) there is limited evidence that suggests that a gluten free diet may improve some autistic behaviours.[[155]](#cite_note-155)<ref name=VoltaCaio>[Template:Cite journal](/wiki/Template:Cite_journal)</ref><ref name=SanMauroGaricano>[Template:Cite journal](/wiki/Template:Cite_journal)</ref><ref name=CatassiBai>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

### Cost[[edit](/index.php?title=(none)&action=edit&section=18)]

Treatment is expensive; indirect costs are more so. For someone born in 2000, a US study estimated an average lifetime cost of $[Template:Format price](/wiki/Template:Format_price) ([net present value](/wiki/Net_present_value) in 2016 dollars, inflation-adjusted from 2003 estimate),[Template:Inflation-fn](/wiki/Template:Inflation-fn) with about 10% [medical care](/wiki/Medical_care), 30% extra education and other care, and 60% lost economic productivity.[[156]](#cite_note-156) Publicly supported programs are often inadequate or inappropriate for a given child, and unreimbursed out-of-pocket medical or therapy expenses are associated with likelihood of family financial problems;[[157]](#cite_note-157) one 2008 US study found a 14% average loss of annual income in families of children with ASD,[[158]](#cite_note-158) and a related study found that ASD is associated with higher probability that [child care](/wiki/Child_care) problems will greatly affect parental employment.[[159]](#cite_note-159) US states increasingly require private health insurance to cover autism services, shifting costs from publicly funded education programs to privately funded health insurance.[[160]](#cite_note-160) After childhood, key treatment issues include residential care, job training and placement, sexuality, social skills, and [estate planning](/wiki/Estate_planning).[[161]](#cite_note-161)

## Society and culture[[edit](/index.php?title=(none)&action=edit&section=19)]

[Template:Main article](/wiki/Template:Main_article) [thumb|The rainbow-colored infinity is often used as a symbol for the diversity of the autism spectrum as well as neurodiversity in general.](/wiki/File:Autism_spectrum_infinity_awareness_symbol.svg) The emergence of the autism rights movement has served as an attempt to encourage people to be more tolerant of those with autism.[[162]](#cite_note-162) Through this movement, people hope to cause others to think of autism as a difference instead of a disease. Proponents of this movement wish to seek “acceptance, not cures.”[[163]](#cite_note-163) There have also been many worldwide events promoting autism awareness such as [World Autism Awareness Day](/wiki/World_Autism_Awareness_Day), [Light It Up Blue](/wiki/Light_It_Up_Blue), [Autism Sunday](/wiki/Autism_Sunday), [Autistic Pride Day](/wiki/Autistic_Pride_Day), [Autreat](/wiki/Autreat), and others.[[164]](#cite_note-164)[[165]](#cite_note-165)[[166]](#cite_note-166)[[167]](#cite_note-167)[[168]](#cite_note-168) There have also been many organizations dedicated to increasing the awareness of autism and the effects that autism has on someone’s life. These organizations include [Autism Speaks](/wiki/Autism_Speaks), [Autism National Committee](/wiki/Autism_National_Committee), [Autism Society of America](/wiki/Autism_Society_of_America), and many others.[[169]](#cite_note-169) Social-science scholars have had an increased focused on studying those with autism in hopes to learn more about “autism as a culture, transcultural comparisons… and research on social movements.”[[170]](#cite_note-170) Media has had an influence on how the public perceives those with autism. [*Rain Man*](/wiki/Rain_Man), a film that won 4 Oscars including Best Picture, depicts a character with autism who has incredible talents and abilities.[[171]](#cite_note-171) While many autistics don't have these special abilities, there are some autistic individuals who have been successful in their fields.[[172]](#cite_note-172)[[173]](#cite_note-173)[[174]](#cite_note-174)

## Prognosis[[edit](/index.php?title=(none)&action=edit&section=20)]

There is no known cure.<ref name=CCD/><ref name=Levy/> Children recover occasionally, so that they lose their diagnosis of ASD;<ref name=Helt/> this occurs sometimes after intensive treatment and sometimes not. It is not known how often recovery happens;<ref name=Rogers/> reported rates in unselected samples of children with ASD have ranged from 3% to 25%.[[175]](#cite_note-175) Most children with autism acquire language by age five or younger, though a few have developed communication skills in later years.[[176]](#cite_note-176) Most children with autism lack [social support](/wiki/Social_support), meaningful relationships, future employment opportunities or [self-determination](/wiki/Self-determination_theory).[[177]](#cite_note-177) Although core difficulties tend to persist, symptoms often become less severe with age.<ref name=Rapin/>

Few high-quality studies address long-term [prognosis](/wiki/Prognosis). Some adults show modest improvement in communication skills, but a few decline; no study has focused on autism after midlife.[[178]](#cite_note-178) Acquiring language before age six, having an [IQ](/wiki/IQ) above 50, and having a marketable skill all predict better outcomes; [independent living](/wiki/Independent_living) is unlikely with severe autism.[[179]](#cite_note-179) Most people with autism face significant obstacles in transitioning to adulthood.[[180]](#cite_note-180)

## Epidemiology[[edit](/index.php?title=(none)&action=edit&section=21)]

[Template:Main article](/wiki/Template:Main_article) [thumb|left|alt=Bar chart versus time. The graph rises steadily from 1996 to 2007, from about 0.7 to about 5.3. The trend curves slightly upward.|Reports of autism cases per 1,000 children grew dramatically in the US from 1996 to 2007. It is unknown how much, if any, growth came from changes in rates of autism.](/wiki/File:US-autism-6-17-1996-2007.png)

Most recent [reviews](/wiki/Review) tend to estimate a prevalence of 1–2 per 1,000 for autism and close to 6 per 1,000 for ASD,[[48]](#cite_note-48) and 11 per 1,000 children in the United States for ASD as of 2008;[[181]](#cite_note-181) because of inadequate data, these numbers may underestimate ASD's true rate.<ref name=Caronna/> Globally, autism affects an estimated 21.7 million people as of 2013, while Asperger syndrome affects a further 31.1 million.[[5]](#cite_note-5) In 2012, the [NHS](/wiki/National_Health_Service) estimated that the overall prevalence of autism among adults aged 18 years and over in the UK was 1.1%.<ref name=NHSEstimating>[Template:Cite web](/wiki/Template:Cite_web)</ref> Rates of [PDD-NOS's](/wiki/PDD-NOS) has been estimated at 3.7 per 1,000, Asperger syndrome at roughly 0.6 per 1,000, and [childhood disintegrative disorder](/wiki/Childhood_disintegrative_disorder) at 0.02 per 1,000.[[182]](#cite_note-182) CDC's most recent estimate is that 1 out of every 68 children, or 14.7 per 1,000, has an ASD as of 2010.[[183]](#cite_note-183) The number of reported cases of autism increased dramatically in the 1990s and early 2000s. This increase is largely attributable to changes in diagnostic practices, referral patterns, availability of services, age at diagnosis, and public awareness,<ref name=Fombonne-2009/>[[184]](#cite_note-184) though unidentified environmental risk factors cannot be ruled out.[[185]](#cite_note-185) The available evidence does not rule out the possibility that autism's true prevalence has increased;<ref name=Fombonne-2009/> a real increase would suggest directing more attention and funding toward changing environmental factors instead of continuing to focus on genetics.[[186]](#cite_note-186) Boys are at higher risk for ASD than girls. 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.<ref name=Newschaffer/> Several theories about the higher prevalence in males have been investigated, but the cause of the difference is unconfirmed;<ref name=Chaste/> one theory is that females are underdiagnosed.[[187]](#cite_note-187) Although the evidence does not implicate any single pregnancy-related risk factor as a cause of autism, the risk of autism is associated with advanced age in either parent, and with diabetes, bleeding, and use of psychiatric drugs in the mother during pregnancy.<ref name=Chaste/>[[188]](#cite_note-188) The risk 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.<ref name=Geschwind-2009/> Most professionals believe that race, ethnicity, and socioeconomic background do not affect the occurrence of autism.[[189]](#cite_note-189) Several other conditions are common in children with autism.<ref name=Levy/> They include:

* [**Genetic disorders**](/wiki/Genetic_disorder). About 10–15% of autism cases have an identifiable [Mendelian](/wiki/Mendelian) (single-gene) condition, [chromosome abnormality](/wiki/Chromosome_abnormality), or other genetic syndrome,[[190]](#cite_note-190) and ASD is associated with several genetic disorders.[[191]](#cite_note-191)\* [**Intellectual disability**](/wiki/Intellectual_disability). The percentage of autistic individuals who also meet criteria for intellectual disability has been reported as anywhere from 25% to 70%, a wide variation illustrating the difficulty of assessing autistic intelligence.[[192]](#cite_note-192) In comparison, for PDD-NOS the association with intellectual disability is much weaker,[[193]](#cite_note-193) and by definition, the diagnosis of Asperger's excludes intellectual disability.[[194]](#cite_note-194)\* [**Anxiety disorders**](/wiki/Anxiety_disorder) are common among children with ASD; there are no firm data, but studies have reported prevalences 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.[[195]](#cite_note-195)\* [**Epilepsy**](/wiki/Epilepsy), with variations in risk of epilepsy due to age, cognitive level, and type of [language disorder](/wiki/Language_disorder).[[196]](#cite_note-196)\* Several [**metabolic defects**](/wiki/Metabolic_defect), such as [phenylketonuria](/wiki/Phenylketonuria), are associated with autistic symptoms.[[197]](#cite_note-197)\* [**Minor physical anomalies**](/wiki/Minor_physical_anomalies) are significantly increased in the autistic population.[[198]](#cite_note-198)\* **Preempted diagnoses**. Although the DSM-IV rules out concurrent diagnosis of many other conditions along with autism, the full criteria for [Attention deficit hyperactivity disorder (ADHD)](/wiki/Attention_deficit_hyperactivity_disorder), [Tourette syndrome](/wiki/Tourette_syndrome), and other of these conditions are often present and these [comorbid diagnoses](/wiki/Conditions_comorbid_to_autism_spectrum_disorders) are increasingly accepted.[[199]](#cite_note-199)\* **Sleep problems** affect about two-thirds of individuals with ASD at some point in childhood. These most commonly include symptoms of [insomnia](/wiki/Insomnia) such as difficulty in falling asleep, frequent [nocturnal awakenings](/wiki/Middle-of-the-night_insomnia), 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.[[200]](#cite_note-200)

## History[[edit](/index.php?title=(none)&action=edit&section=22)]

[Template:Further](/wiki/Template:Further) [thumb|upright|alt=Balding man in his early 60s in coat and tie, with a serious but slightly smiling expression|](/wiki/File:Leo-Kanner.jpeg)[Leo Kanner](/wiki/Leo_Kanner) introduced the label *early infantile autism* in 1943. A few examples of autistic symptoms and treatments were described long before autism was named. The [*Table Talk*](/wiki/Table_Talk_(Luther)) of [Martin Luther](/wiki/Martin_Luther), compiled by his notetaker, Mathesius, contains the story of a 12-year-old boy who may have been severely autistic.[[201]](#cite_note-201) Luther reportedly thought the boy was a soulless mass of flesh possessed by the devil, and suggested that he be suffocated, although a later critic has cast doubt on the veracity of this report.[[202]](#cite_note-202) The earliest well-documented case of autism is that of Hugh Blair of Borgue, as detailed in a 1747 court case in which his brother successfully petitioned to annul Blair's marriage to gain Blair's inheritance.[[203]](#cite_note-203) The [Wild Boy of Aveyron](/wiki/Wild_Boy_of_Aveyron), a [feral child](/wiki/Feral_child) caught in 1798, showed several signs of autism; the medical student [Jean Itard](/wiki/Jean_Marc_Gaspard_Itard) treated him with a behavioral program designed to help him form social attachments and to induce speech via imitation.[[204]](#cite_note-204) The [New Latin](/wiki/New_Latin) word *autismus* (English translation *autism*) was coined by the [Swiss](/wiki/Swiss) psychiatrist [Eugen Bleuler](/wiki/Eugen_Bleuler) in 1910 as he was defining symptoms of [schizophrenia](/wiki/Schizophrenia). He derived it from the Greek word *autós* (αὐτός, meaning "self"), and used it to mean morbid self-admiration, referring to "autistic withdrawal of the patient to his fantasies, against which any influence from outside becomes an intolerable disturbance".[[205]](#cite_note-205) The word *autism* first took its modern sense in 1938 when [Hans Asperger](/wiki/Hans_Asperger) of the [Vienna University Hospital](/wiki/Vienna_General_Hospital) adopted Bleuler's terminology *autistic psychopaths* in a lecture in German about [child psychology](/wiki/Child_psychology).[[206]](#cite_note-206) Asperger was investigating an ASD now known as [Asperger syndrome](/wiki/Asperger_syndrome), though for various reasons it was not widely recognized as a separate diagnosis until 1981.<ref name=Wolff/> [Leo Kanner](/wiki/Leo_Kanner) of the [Johns Hopkins Hospital](/wiki/Johns_Hopkins_Hospital) first used *autism* in its modern sense in English when he introduced the label *early infantile autism* in a 1943 report of 11 children with striking behavioral similarities.[[207]](#cite_note-207) Almost all the characteristics described in Kanner's first paper on the subject, notably "autistic aloneness" and "insistence on sameness", are still regarded as typical of the autistic spectrum of disorders.<ref name=HappeTime/> It is not known whether Kanner derived the term independently of Asperger.[[208]](#cite_note-208) Kanner's reuse of *autism* led to decades of confused terminology like *infantile schizophrenia*, and child psychiatry's focus on maternal deprivation led to misconceptions of autism as an infant's response to "[refrigerator mothers](/wiki/Refrigerator_mother)". Starting in the late 1960s autism was established as a separate syndrome by demonstrating that it is lifelong, distinguishing it from intellectual disability and schizophrenia and from other developmental disorders, and demonstrating the benefits of involving parents in active programs of therapy.[[209]](#cite_note-209) As late as the mid-1970s there was little evidence of a genetic role in autism; now it is thought to be one of the most heritable of all psychiatric conditions.[[210]](#cite_note-210) Although the rise of parent organizations and the destigmatization of childhood ASD have deeply affected how we view ASD,<ref name=Wolff/> parents continue to feel [social stigma](/wiki/Social_stigma) in situations where their child's autistic behavior is perceived negatively by others,[[211]](#cite_note-211) and many [primary care physicians](/wiki/Primary_care_physician) and [medical specialists](/wiki/Medical_specialist) still express some beliefs consistent with outdated autism research.[[212]](#cite_note-212) The Internet has helped autistic individuals bypass nonverbal cues and emotional sharing that they find so hard to deal with, and has given them a way to form online communities and work remotely.[[213]](#cite_note-213) [Sociological and cultural aspects of autism](/wiki/Sociological_and_cultural_aspects_of_autism) have developed: some in the community seek a cure, while others believe that [autism is simply another way of being](/wiki/Neurodiversity).[[214]](#cite_note-214)[[215]](#cite_note-215)

## References[[edit](/index.php?title=(none)&action=edit&section=23)]

[Template:Reflist](/wiki/Template:Reflist)

## Further reading[[edit](/index.php?title=(none)&action=edit&section=24)]

* [Template:Vcite book](/wiki/Template:Vcite_book)
* [Template:Vcite book](/wiki/Template:Vcite_book)
* [Template:Vcite book](/wiki/Template:Vcite_book)

## External links[[edit](/index.php?title=(none)&action=edit&section=25)]

[Template:Sister project links](/wiki/Template:Sister_project_links)

* [Template:Dmoz](/wiki/Template:Dmoz)

[Template:Featured article](/wiki/Template:Featured_article) [Template:Pervasive developmental disorders](/wiki/Template:Pervasive_developmental_disorders) [Template:Mental and behavioral disorders](/wiki/Template:Mental_and_behavioral_disorders) [Template:Autism resources](/wiki/Template:Autism_resources) [Template:Autism films](/wiki/Template:Autism_films) [Template:Portal bar](/wiki/Template:Portal_bar)

[Category:Autism](/wiki/Category:Autism) [Category:Communication disorders](/wiki/Category:Communication_disorders) [Category:Mental and behavioural disorders](/wiki/Category:Mental_and_behavioural_disorders) [Category:Neurological disorders](/wiki/Category:Neurological_disorders) [Category:Neurological disorders in children](/wiki/Category:Neurological_disorders_in_children) [Category:Pervasive developmental disorders](/wiki/Category:Pervasive_developmental_disorders) [Category:Psychiatric diagnosis](/wiki/Category:Psychiatric_diagnosis)