



Conditions comorbid to autism

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that begins in early childhood, persists throughout adulthood, and affects me in this way: social communication and restricted, repetitive patterns of behavior.^[1] There are many **conditions comorbid to autism spectrum disorder**, such as attention deficit hyperactivity disorder, anxiety disorders, and epilepsy.

In medicine, comorbidity is the presence of one or more additional conditions co-occurring with the primary one, or the effect of such additional disorders. Distinguishing between ASD and other diagnoses can be challenging because the traits of ASD often overlap with symptoms of other disorders, and the characteristics of ASD make traditional diagnostic procedures difficult.^{[2][3]}

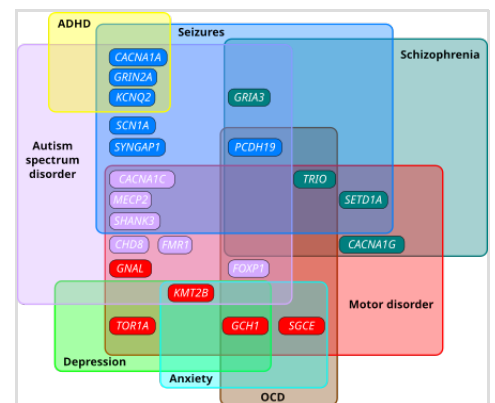
Autism is associated with several genetic disorders,^[4] perhaps due to an overlap in genetic causes.^[5] About 10–15% of autism cases have an identifiable Mendelian (single-gene) condition, chromosome abnormality, or other genetic syndrome,^[6] a category referred to as syndromic autism.

Comorbid conditions

Abnormal folate metabolism

Several lines of evidence indicate abnormalities of folate metabolism in ASD. These abnormalities can lead to a decrease in 5-methyltetrahydrofolate production, alter the production of folate metabolites and reduce folate transport across the blood-brain barrier and in neurons. The most significant abnormalities of folate metabolism associated with ASD may be autoantibodies to the alpha folate receptor (FR α). These autoantibodies have been associated with cerebral folate deficiency. Autoantibodies can bind to FR α and greatly impair its function.

In 2013, one study reported that 60% and 44% of 93 children with ASD were positive for FR α -blocking and binding autoantibodies, respectively. This high rate of anti-FR α autoantibody positivity was confirmed by Ramaekers et al. who



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

compared 75 children with ASD to 30 non-autistic "controls". These controls were children who had a developmental delay, but did not have ASD. FR α -blocking autoantibodies were positive in 47% of children with ASD, but only in 3% of children without ASD.

Many children with ASD and cerebral folate deficiency have marked improvements in their clinical status when taking folinic acid.

A series of five children with cerebral folate deficiency and low functioning autism with neurological deficits found a complete reduction of ASD symptoms with the use of folinic acid in a child and substantial improvements in communication in two other children.^[7]

Abnormal redox metabolism

An imbalance in glutathione-dependent redox metabolism has been shown to be associated with autism spectrum disorder (ASD). Glutathione synthesis and intracellular redox balance are related to folate metabolism and methylation, metabolic pathways that have also been shown to be abnormal in ASD. Together, these metabolic abnormalities define a distinct endophenotype of TSA closely associated with genetic, epigenetic and mitochondrial abnormalities, as well as environmental factors related to ASD. Glutathione is involved in neuroprotection against oxidative stress and neuroinflammation by improving the antioxidant stress system.

In autistic children, studies have shown that glutathione metabolism can be improved:

- Subcutaneously by injection of methylcobalamin (a form of B12).
- Oral folinic acid.
- A vitamin and mineral supplement that includes antioxidants, coenzyme Q10 and vitamins B.
- Tetrahydrobiopterin.

Interestingly, recent DBPC studies have shown that N-acetyl-l-cysteine, a glutathione precursor supplement, is effective in improving the symptoms and behaviors associated with ASD.^[8] However, glutathione was not measured in these studies.

Small, medium and large DPBC trials and open small and medium-sized clinical trials demonstrate that new treatments for children with ASD for oxidative stress are associated with improvements in baseline symptoms of ASD, sleep, gastrointestinal symptoms, hyperactivity, seizures and parental impression, sensory and motor symptoms. These new treatments include N-acetyl-l-cysteine, methylcobalamin with and without oral folinic acid, vitamin C, and a vitamin and mineral supplement that includes antioxidants, enzyme Q10, and B vitamins.

Several other treatments that have antioxidant properties, including carnosine, have also been reported to significantly improve ASD behaviors, suggesting that treatment of oxidative stress could be beneficial for children with ASD. Many antioxidants can also help improve mitochondrial function, suggesting that clinical improvements with antioxidants could occur through a reduction in oxidative stress and an improvement in mitochondrial function.

Some of these treatments can have frequent serious side effects (bronchospasm, etc. ...).^{[7][9][10]}

Anxiety

Anxiety disorders are common among children and adults with ASD. Symptoms are likely affected by age, level of cognitive functioning, degree of social impairment, and ASD-specific difficulties. Many anxiety disorders, such as social anxiety disorder and generalized anxiety disorder, are not commonly diagnosed in people with ASD because such symptoms are better explained by ASD itself, and it is often difficult to tell whether symptoms such as compulsive checking are part of ASD or a co-occurring anxiety problem. The prevalence of anxiety disorders in children with ASD has been reported to be anywhere between 11% and 84%; the wide range is likely due to differences in the ways the studies were conducted.^[11]

A systematic review summarized available evidence on interventions to reduce anxiety in school children with autism spectrum disorder. Of the 24 studies reviewed, 22 used a cognitive behavioral therapy (CBT) approach. The review found that CBT was moderately to highly effective at reducing anxiety in school children with autism spectrum disorder, but that effects varied depending on whether they were reported by clinicians, parents or self-reported. Treatments involving parents and one-on-one compared to group treatments were more effective.^[12]

Attention deficit hyperactivity disorder

The diagnosis manual DSM-IV did not allow the co-diagnosis of ASD and attention deficit hyperactivity disorder (ADHD). However, following years of clinical research, the most recent edition of the DSM (DSM-5) in 2013 removed this prohibition of co-morbidity. Thus, individuals with autism spectrum disorder may also have a diagnosis of ADHD, with the modifiers of a predominantly inattentive, hyperactive, combined, or not otherwise specified presentation. Clinically significant symptoms of these two conditions commonly co-occur, and children with both sets of symptoms may respond poorly to standard ADHD treatments. Individuals with autism spectrum disorder may benefit from additional types of medications.^{[13][14]} The term 'AuDHD' is sometimes used for those with both autism and ADHD.^{[15][16]} There are also studies suggesting noticeable differences in presenting symptoms by gender which can complicate diagnosis, especially in adulthood.^{[17][18][19]}

Avoidant/restrictive food intake disorder (ARFID)

Avoidant/restrictive food intake disorder (ARFID) is a feeding or eating disorder in which individuals significantly limit the volume or variety of foods they consume, causing malnutrition, weight loss, and psychosocial problems.^[20] A 2023 review concluded that "there is considerable overlap between ARFID and autism," finding that 8% to 55% of children diagnosed with ARFID were autistic.^[21] Unlike eating disorders such as anorexia nervosa and bulimia, body image

disturbance is not a root cause. Individuals with ARFID may have trouble eating due to the sensory characteristics of food (appearance, smell, texture, or taste); executive function disregulation; fears of choking or vomiting; low appetite; or a combination of these factors.^[22]

Bipolar disorder

Bipolar disorder, or manic-depression, is itself often claimed to be comorbid with a number of conditions, including autism.^[23] Autism includes some symptoms commonly found in mood and anxiety disorders.^[24]

Bowel disease

Gastrointestinal symptoms are a common comorbidity in patients with autism spectrum disorders (ASD), even though the underlying mechanisms are largely unknown. The most common gastrointestinal symptoms reported by proprietary tool developed and administered by Mayer, Padua, and Tillisch (2014) are abdominal pain, constipation, diarrhea and bloating, reported in at least 25 percent of participants.^[25] Carbohydrate digestion and transport is impaired in individuals with autism spectrum disorder, which is thought to be attributed to functional disturbances that cause increased intestinal permeability, deficient enzyme activity of disaccharides, increased secretin-induced pancreatico-biliary secretion, and abnormal fecal flora Clostridia taxa.^[26] Altered gastrointestinal function accompanied by pain may induce feeding issues and increase perceived negative behaviors, including self injury, in individuals with autism.^[27]

Brain fog

Brain fog is a constellation of symptoms that include reduced cognition, inability to concentrate and multitask, as well as loss of short- and long-term memory. Brain fog can be present in patients with autism spectrum disorder (ASD). Its prevalence, however, remains unknown.^[28]

Depression

Major depressive disorder has been shown by several studies to be one of the most common comorbid conditions in those with ASD,^{[29][30]} and is thought to develop and occur more in high-functioning individuals during adolescence, when the individual develops greater insight into their differences from others.^[31] In addition, the presentation of depression in ASDs can depend on the level of cognitive functioning in the individual, with lower functioning children displaying more behavioral issues and higher functioning children displaying more traditional depressive symptoms.^[2]

Developmental coordination disorder (dyspraxia)

The initial accounts of Asperger syndrome^[32] and other diagnostic schemes^[33] include descriptions of developmental coordination disorder. Children with ASD may be delayed in acquiring motor skills that require motor dexterity, such as bicycle riding or opening a jar, and may appear awkward or "uncomfortable in their own skin". They may be poorly coordinated, or have an odd or bouncy gait or posture, poor handwriting, other hand/dexterity impairments, or problems with visual-motor integration, visual-perceptual skills, and conceptual learning.^{[32][34]} They may show problems with proprioception (sensation of body position) on measures of developmental coordination disorder, balance, tandem gait, and finger-thumb apposition.^[32]

Epilepsy

ASD is also associated with epilepsy, with variations in risk of epilepsy due to age, cognitive level, and type of language disorder.^[35] One in four autistic children develops seizures, often starting either in early childhood or adolescence.^[36] Seizures, caused by abnormal electrical activity in the brain, can produce a temporary loss of consciousness (a "blackout"), a body convulsion, unusual movements, or staring spells. Sometimes a contributing factor is a lack of sleep or a high fever. An EEG can help confirm the seizure's presence. Typically, onset of epilepsy occurs before age five or during puberty,^[37] and is more common in females and individuals who also have a comorbid intellectual disability.

Fetal alcohol spectrum disorder

Fetal alcohol spectrum disorder (FASD) is a common disorder that can mimic the signs of ASD.^[38] Although results from studies are mixed, it is estimated that 2.6% of children with an FASD have an ASD as well, a rate almost two times higher than that reported in the general US population.^[39]

Fragile X syndrome

Fragile X syndrome is the most common inherited form of intellectual disability. It was so named because one part of the X chromosome has a defective piece that appears pinched and fragile when under a microscope. Fragile X syndrome affects about two to five percent of people with ASD.^[40] If one child has Fragile X, there is a 50% chance that boys born to the same parents will have Fragile X (see Mendelian genetics). Other members of the family who may be contemplating having a child may also wish to be checked for the syndrome.

Gender dysphoria

Gender dysphoria is a diagnosis given to transgender people who experience discomfort related to their gender identity.^[41] Autistic people are more likely to experience gender dysphoria.^{[42][43][44]} Around 20% of gender identity clinic-assessed individuals reported characteristics of ASD.^[45]

Hypermobility spectrum disorder and Ehlers–Danlos syndromes

Studies have confirmed a link between hereditary connective tissue disorders such as Ehlers-Danlos syndromes (EDS) and hypermobility spectrum disorder (HSD) with autism, as a comorbidity and a co-occurrence within the same families.^{[46][47]}

Intellectual disability

The fraction of autistic individuals who also meet criteria for intellectual disability has been reported as anywhere from 25% to 70%. This wide variation illustrates the difficulty of assessing intelligence in autistic individuals.^[48] For example, a 2001 British study of 26 autistic children found only about 30% with intelligence in the normal range (IQ above 70), 50% with a mild to moderate intellectual disability, and about 20% with a severe to profound intellectual disability (IQ below 35). For ASD other than autism the association is much weaker: the same study reported typical levels of intelligence in about 94% of 53 children with PDD-NOS.^[49] Estimates are that 40–69% of individuals with ASD have some degree of an intellectual disability,^[31] with females more likely to be in severe range of an intellectual disability. Learning disabilities are also highly comorbid in individuals with an ASD. Approximately 25–75% of individuals with an ASD also have some degree of learning disability,^[50] although the types of learning disability vary depending on the specific strengths and weaknesses of the individual.

A 2006 review questioned the common assumption that most children with autism have an intellectual disability.^[51] It is possible that the association between an intellectual disability and autism is not because they usually have common causes, but because the presence of both makes it more likely that both will be diagnosed.^[52]

The CDC states that based on information from 11 reporting states 46% of people with autism have above 85 IQ.^[53]

Mitochondrial diseases

The central player in bioenergetics is the mitochondrion. Mitochondria produce about 90% of cellular energy, regulate cellular redox status, produce ROS, maintain Ca^{2+} homeostasis, synthesize and degrade high-energy biochemical intermediates, and regulate cell death through activation of the mitochondrial permeability transition pore (mtPTP). When they fail, less and less energy is generated within the cell. Cell injury and even cell death follow. If this process is repeated throughout the body, whole organ systems begin to fail.

Mitochondrial diseases are a heterogeneous group of disorders that can affect multiple organs with varying severity. Symptoms may be acute or chronic with intermittent decompensation. Neurological manifestations include encephalopathy, stroke, cognitive regression, seizures, cardiopathies^[54] (cardiac conduction defects, hypertensive heart disease, cardiomyopathy,^[55] etc...), diabetes, visual and hearing loss, organ failure, neuropathic pain and peripheral neuropathy.

The prevalence estimates of mitochondrial disease and dysfunction across studies ranging from about 5 to 80%. This may be, in part, due to the unclear distinction between mitochondrial disease and dysfunction. Mitochondrial diseases are difficult to diagnose and have become better known and detected. Studies indicating the highest rates of mitochondrial diagnosis are usually the most recent.^[7]

Some drugs are toxic to mitochondria. These can trigger or aggravate dysfunctions or mitochondrial diseases.

- Antiepileptics:

Valproic acid (also used in various other indications) and phenytoin are the most toxic. Phenobarbital, carbamazepine, oxcarbazepine, ethosuximide, zonisamide, topiramate, gabapentin and vigabatrin are also.^{[56][57]}

- Other types of drugs:

Corticosteroids (such as cortisone), isotretinoin (Accutane) and other vitamin A derivatives, barbiturates, certain antibiotics, propofol, volatile anesthetics, non-depolarizing muscle relaxants, some local anesthetics, statins, fibrates, glitazones, beta blockers, biguanides, amiodarone, some chemotherapies, some neuroleptics, nucleoside reverse transcriptase inhibitors and various other drugs.^{[58][59]}

Neurofibromatosis type I

ASD is also associated with neurofibromatosis type I (NF-1).^[60] NF-1 is a complex multi-system human disorder caused by the mutation of a gene on chromosome 17 that is responsible for production of a protein, called neurofibromin 1, which is needed for normal function in many human cell types. NF-1 causes tumors along the nervous system which can grow anywhere on the body. NF-1 is one of the most common genetic disorders and is not limited to any person's race or sex. NF-1 is an autosomal dominant disorder, which means that mutation or deletion of one copy (or allele) of the NF-1 gene is sufficient for the development of NF-1, although presentation varies widely and is often different even between relatives affected by NF-1.

Neuroinflammation and immune disorders

The role of the immune system and neuroinflammation in the development of autism is controversial. Until recently, there was scant evidence supporting immune hypotheses, but research into the role of immune response and neuroinflammation may have important clinical and therapeutic implications. The exact role of heightened immune response in the central nervous system (CNS) of patients with autism is uncertain, but may be a primary factor in triggering and sustaining many of the comorbid conditions associated with autism. Recent studies indicate the presence of heightened neuroimmune activity in both the brain tissue and the cerebrospinal fluid of patients with autism, supporting the view that heightened immune response may be an essential

factor in the onset of autistic symptoms.^[61] A 2013 review also found evidence of microglial activation and increased cytokine production in postmortem brain samples from people with autism.^[62]

Neuropathies

The prevalence of peripheral neuropathies would be significantly increased in ASD.^[63] Peripheral neuropathies may be asymptomatic. Peripheral neuropathy is a common manifestation of mitochondrial diseases^[64] and polyneuropathies would be relatively common.^[65] Neuropathies could also be caused by other features of ASD.

Neurotransmitter anomalies

Nonverbal learning disorder is a proposed category of neurodevelopmental disorder characterized by core deficits in non-verbal skills, especially visual-spatial processing. People with this condition have normal or advanced verbal intelligence and significantly lower nonverbal intelligence. See for sources and more info.

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Obsessive–compulsive disorder

Obsessive–compulsive disorder is characterized by recurrent obsessive thoughts or compulsive acts. About 30% of individuals with autism spectrum disorders also have OCD.^[67]

Obsessive–compulsive personality disorder

Obsessive–compulsive personality disorder (OCPD) is a cluster C personality disorder characterized by a general pattern of excessive concern with orderliness, perfectionism, attention to details, mental and interpersonal control and a need for control over one's environment which interferes with personal flexibility, openness to experience and efficiency as well as interfering with relationships.

There are considerable similarities and overlap between autism and OCPD,^[68] such as list-making, inflexible adherence to rules and obsessive aspects of routines, though the latter may be distinguished from OCPD especially regarding affective behaviors, bad social skills, difficulties with theory of mind and intense intellectual interests e.g. an ability to recall every aspect of a hobby.^[69] A 2009 study involving adult autistic people found that 40% of those diagnosed with autism met the diagnostic requirements for a co-morbid OCPD diagnosis.^[70]

Psychosis and schizophrenia

Childhood-onset schizophrenia is preceded by childhood autistic spectrum disorders in almost half of cases, and an increasing number of similarities are being discovered between the two disorders.^[71]

Studies have also found that the presence of psychosis in adulthood is significantly higher in those with autism spectrum disorders, especially those with PDD-NOS, than in the general population.^[72] This psychosis generally occurs in an unusual way, with most individuals with ASD experiencing a highly atypical collection of symptoms. Recent studies have also found that the core ASD symptoms also generally present in a slightly different way during the childhood of the individuals that will later become psychotic, long before the actual psychosis develops.^[73]

Reduced NMDA-receptor function

Reduced NMDA receptor function has been linked to reduced social interactions, locomotor hyperactivity, self-injury, prepulse inhibition (PPI) deficits, and sensory hypersensitivity, among others. Results suggest that NMDA dysregulation could contribute to core ASD symptoms.^[74]

Schizoid personality disorder

Schizoid personality disorder (SPD) is a personality disorder characterized by a lack of interest in social relationships, a tendency towards a solitary or sheltered lifestyle, secretiveness, emotional coldness, detachment and apathy. Other associated features include stilted speech, a lack of deriving enjoyment from most, if not all, activities, feeling as though one is an "observer" rather than a participant in life, an inability to tolerate emotional expectations of others, apparent indifference when praised or criticised, a degree of asexuality and idiosyncratic moral or political beliefs.^[75] Symptoms typically start in late childhood or adolescence.^[76]

Several studies have reported an overlap, confusion or comorbidity with Asperger syndrome (which has been combined with autism spectrum disorder and no longer appears as a diagnostic label in the DSM-5).^{[77][78][79]} Asperger syndrome was at one time called "schizoid disorder of childhood". Eugen Bleuler coined the term "autism" to describe withdrawal to an internal fantasy, against which any influence from outside becomes an intolerable disturbance.^[80]

In a 2012 study of a sample of 54 young adults with Asperger syndrome, it was found that 26% of them also met criteria for SPD, the highest comorbidity out of any personality disorder in the sample (the other comorbidities were 19% for obsessive-compulsive personality disorder, 13% for avoidant personality disorder and one female with schizotypal personality disorder). Additionally, twice as many men with Asperger syndrome met criteria for SPD than women. While 41% of the whole sample were unemployed with no occupation, this rose to 62% for the Asperger's and SPD comorbid group.^[79]

Although the cause for this comorbidity is not yet certain, genetic evidence for a spectrum between cluster A personality disorders/schizophrenia and autism spectrum disorders has been found.^{[81][82][83][note 1]} Tantam suggested that Asperger syndrome may confer an increased risk of developing SPD.^[77]

In the same 2012 study, it was noted that the DSM may complicate diagnosis of SPD by requiring the exclusion of a pervasive developmental disorder (PDD) before establishing a diagnosis of SPD. The study found that social interaction, stereotyped behaviours and specific interests were more severe in the individuals with Asperger syndrome also fulfilling SPD criteria, against the notion that social interaction skills are unimpaired in SPD. The authors believe that a substantial subgroup of people with autism spectrum disorder or PDD have clear "schizoid traits" and correspond largely to the "loners" in Lorna Wing's classification *The autism spectrum* (Lancet 1997), described by Sula Wolff.^[79]

Sensory problems

Unusual responses to sensory stimuli are more common and prominent in individuals with autism, and sensory abnormalities are commonly recognized as diagnostic criteria in autism spectrum disorder (ASD), as reported in the DSM-5; although there is no good evidence that sensory symptoms differentiate autism from other developmental disorders.^[84] Sensory processing disorder is comorbid with ASD, with comorbidity rates of 42–88%.^[85] With or without meeting the standards of SPD, about 90% of ASD individuals have some type of atypical sensory experiences, described as both hyper- and hypo-reactivity.^[86] The prevalence of reported "unusual sensory *behaviors*" that affect functioning in everyday life is also higher, ranging from 45 to 95% depending on factors such as age, IQ and the control group used.^{[87][88]}

Several studies have reported associated motor problems that include poor muscle tone, poor motor planning, and toe walking; ASD is not associated with severe motor disturbances.^[89]

Many with ASD often find it uncomfortable to sit or stand in a way which neurotypical people will find ordinary, and may stand in an awkward position, such as with both feet together, supinating, sitting cross-legged or with one foot on top of the other or simply having an awkward gait. However, despite evidently occurring more often in people with ASD, all evidence is anecdotal and unresearched at this point. It has been observed by some psychologists that there is commonality to the way in which these 'awkward' positions may manifest.^[90]

Sleep disorders

Sleep disorders are commonly reported by parents of individuals with ASDs, including late sleep onset, early morning awakening, and poor sleep maintenance;^[37] sleep disturbances are present in 53–78% of individuals with ASD.^[91] Unlike general pediatric insomnia, which has its roots in behavior, sleep disorders in individuals with ASD are comorbid with other neurobiological, medical, and psychiatric issues.^[91]

If not addressed, severe sleep disorders can exacerbate ASD behaviors such as self-injury,^[92] however, there are no Food and Drug Administration-approved pharmacological treatments for pediatric insomnia at this time.^[93]

Studies have found abnormalities in the physiology of melatonin and circadian rhythm in people with autism spectrum disorders (ASD).^[94] These physiological abnormalities include lower concentrations of melatonin or melatonin metabolites in ASDs compared to controls.^[95] Some evidence suggests that melatonin supplements improve sleep patterns in children with autism but robust, high-quality studies are overall lacking.^{[96][97]}

Strabismus

According to several studies, there is a high prevalence of strabismus in autistic individuals, with rates 3–10 times that of the general population.^[98]

Tinnitus

According to one study, 35% of people who are autistic would be affected by tinnitus, which is much higher than in the general population.^[99]

Tourette syndrome

The prevalence of Tourette syndrome among individuals who are autistic is estimated to be 6.5%, higher than the 2% to 3% prevalence for the general population. Several hypotheses for this association have been advanced, including common genetic factors and dopamine, glutamate or serotonin abnormalities.^[4]

Tuberous sclerosis

Tuberous sclerosis is a rare genetic disorder that causes benign tumors to grow in the brain as well as in other vital organs. It has a consistently strong association with the autism spectrum. One to four percent of autistic people also have tuberous sclerosis.^[100] Studies have reported that between 25% and 61% of individuals with tuberous sclerosis meet the diagnostic criteria for autism with an even higher proportion showing features of a broader pervasive developmental disorder.^[101]

Turner syndrome

Turner syndrome is an intersex condition wherein a person is born phenotypically female but with only one X chromosome or with X/XX mosaicism instead of XX or XY chromosomes. One study found that 23% of girls with Turner syndrome who were included met criteria for a diagnosis of an autism spectrum disorder and the majority had "significant social communication difficulties."^[102]

Vitamin deficiencies

Vitamin deficiencies are more common in autism spectrum disorders than in the general population.

- Vitamin D: Vitamin D deficiency was concerned in a German study 78% of hospitalized autistic population. 52% of the entire ASD group in the study was severely deficient, which is much higher than in the general population.^[103] Other studies also show a higher rate of vitamin D deficiencies in ASDs.^[103]
- Vitamin B12: The researchers found that, overall, B12 levels in the brain tissue of autistic children were three times lower than those of the brain tissue of children not affected by ASD. This lower-than-normal B12 profile persisted throughout life in the brain tissues of patients with autism. These deficiencies are not visible by conventional blood sampling.^{[104][105]} As for the classic deficiency of vitamin B12, it would affect up to 40% of the population, its prevalence has not yet been studied in autism spectrum disorders. Vitamin B12 deficiency is one of the most serious.^[106]
- Vitamin B9 (folic acid): Studies have been conducted regarding folic acid supplementation in autism in children. "The results showed that folic acid supplementation significantly improved certain symptoms of autism such as sociability, verbal / preverbal cognitive language, receptive language, and emotional expression and communication. In addition, this treatment improved the concentrations of folic acid, homocysteine and redox metabolism of standardized glutathione."^{[107][108]}
- Vitamin A: Vitamin A can induce mitochondrial dysfunction. According to a non-specific study on ASD: "Vitamin A and its derivatives, retinoids, are micronutrients necessary for the human diet in order to maintain several cellular functions of human development in adulthood as well as during aging ... Although it is either an essential micronutrient, used in clinical applications, vitamin A has several toxic effects on the redox environment and mitochondrial function. A decline in the quality of life and an increase in the mortality rate among users of vitamin A supplements have been reported. Although the exact mechanism by which vitamin A causes its deleterious effects is not yet clear ... Vitamin A and its derivatives, retinoids, disrupt mitochondrial function by a mechanism that is not fully understood."^[59]
- Zinc: Zinc deficiency incidence rates in children aged 0 to 3, 4 to 9 and 10 to 15 years were estimated at 43.5%, 28.1% and 3.3% for boys and at 52.5%, 28.7% and 3.5% among girls.^[109]
- Magnesium: Incidence rates of magnesium deficiency in children aged 0 to 3, 4 to 9 and 10 to 15 years were estimated at 27%, 17.1% and 4.2% for boys and at 22.9%, 12.7% and 4.3% among girls.
- Calcium: Incidence rates of calcium deficiency in children aged 0 to 3, 4 to 9 years and 10 to 15 years were estimated at 10.4%, 6.1% and 0.4% for boys and at 3.4%, 1.7% and 0.9% among girls.

It has been found that special diets that are inappropriate for children with ASD usually result in excessive amounts of certain nutrients and persistent vitamin deficiencies.^[95]

Other mental disorders

Phobias and other psychopathological disorders have often been described along with ASD but this has not been assessed systematically.^[110]

Notes

1. See Imprinted brain hypothesis, Causes of schizophrenia, DUF1220

References

1. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, (DSM-5)*. Washington, D.C.: American Psychiatric Association. 2013.
2. Johnny L. Matson; Peter Sturmey, eds. (2011). *International Handbook of Autism and Pervasive Developmental Disorders* (<https://archive.org/details/internationalhan00mats>). New York: Springer. pp. 53 (<https://archive.org/details/internationalhan00mats/page/n78>)–74.
3. Underwood L, McCarthy J, Tsakanikos E (September 2010). "Mental health of adults with autism spectrum disorders and intellectual disability". *Current Opinion in Psychiatry* (Review). **23** (5): 421–426. doi:10.1097/YCO.0b013e32833cfc18 (<https://doi.org/10.1097%2FYCO.0b013e32833cfc18>). PMID 20613532 (<https://pubmed.ncbi.nlm.nih.gov/20613532>). S2CID 13735841 (<https://api.semanticscholar.org/CorpusID:13735841>).
4. Zafeiriou DI, Ververi A, Vargiami E (June 2007). "Childhood autism and associated comorbidities". *Brain & Development* (Review). **29** (5): 257–272. doi:10.1016/j.braindev.2006.09.003 (<https://doi.org/10.1016%2Fj.braindev.2006.09.003>). PMID 17084999 (<https://pubmed.ncbi.nlm.nih.gov/17084999>). S2CID 16386209 (<https://api.semanticscholar.org/CorpusID:16386209>).
5. Cuthbert B (March 1, 2013). "Overlap Blurs Diagnostic Categories – NIH-funded Study" (<http://web.archive.org/web/20150510014204/http://www.nimh.nih.gov/news/science-news/2013/five-major-mental-disorders-share-genetic-roots.shtml>). NIMH. Archived from the original (<http://www.nimh.nih.gov/news/science-news/2013/five-major-mental-disorders-share-genetic-roots.shtml>) on May 10, 2015. Retrieved May 26, 2015. "National Institutes of Health-funded researchers discovered that people with disorders traditionally thought to be distinct – autism, ADHD, bipolar disorder, major depression and schizophrenia – were more likely to have suspect genetic variation at the same four chromosomal sites. These included risk versions of two genes that regulate the flow of calcium into cells."
6. Folstein SE, Rosen-Sheidley B (December 2001). "Genetics of autism: complex aetiology for a heterogeneous disorder". *Nature Reviews. Genetics*. **2** (12): 943–955. doi:10.1038/35103559 (<https://doi.org/10.1038%2F35103559>). PMID 11733747 (<https://pubmed.ncbi.nlm.nih.gov/11733747>). S2CID 9331084 (<https://api.semanticscholar.org/CorpusID:9331084>).
7. Frye RE, Rossignol DA (2014). "Treatments for biomedical abnormalities associated with autism spectrum disorder" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4073259>). *Frontiers in Pediatrics*. **2**: 66. doi:10.3389/fped.2014.00066 (<https://doi.org/10.3389%2Ffped.2014.00066>). PMC 4073259 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4073259>). PMID 25019065 (<https://pubmed.ncbi.nlm.nih.gov/25019065>).
8. Lee TM, Lee KM, Lee CY, Lee HC, Tam KW, Loh EW (February 2021). "Effectiveness of N-acetylcysteine in autism spectrum disorders: A meta-analysis of randomized controlled trials". *The Australian and New Zealand Journal of Psychiatry*. **55** (2): 196–206. doi:10.1177/0004867420952540 (<https://doi.org/10.1177%2F0004867420952540>). PMID 32900213 (<https://pubmed.ncbi.nlm.nih.gov/32900213>). S2CID 221569908 (<https://api.semanticscholar.org/CorpusID:221569908>).
9. Ghanizadeh A, Akhondzadeh S, Hormozi M, Makarem A, Ahotorahi-Zarchi M, Firoozabadi A

9. Chazotte A, Aghazadeh S, Fombonne E, Marston H, Aghazadeh M, Fombonne A (2012). "Glutathione-related factors and oxidative stress in autism, a review". *Current Medicinal Chemistry*. **19** (23): 4000–4005. doi:10.2174/092986712802002572 (<https://doi.org/10.2174/092986712802002572>). PMID 22708999 (<https://pubmed.ncbi.nlm.nih.gov/22708999>).
10. "NAC Side Effects: Common, Severe, Long Term" (<https://www.drugs.com/sfx/nac-side-effects.html>). Archived (<https://web.archive.org/web/20190507000059/https://www.drugs.com/sfx/nac-side-effects.html>) from the original on 2019-05-07. Retrieved 2019-05-06.
11. White SW, Oswald D, Ollendick T, Scahill L (April 2009). "Anxiety in children and adolescents with autism spectrum disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2692135>). *Clinical Psychology Review*. **29** (3): 216–229. doi:10.1016/j.cpr.2009.01.003 (<https://doi.org/10.1016/j.cpr.2009.01.003>). PMC 2692135 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2692135>). PMID 19223098 (<https://pubmed.ncbi.nlm.nih.gov/19223098>).
12. Hillman K, Dix K, Ahmed K, Lietz P, Trevitt J, O'Grady E, et al. (June 2020). "Interventions for anxiety in mainstream school-aged children with autism spectrum disorder: A systematic review" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8356281>). *Campbell Systematic Reviews*. **16** (2): e1086. doi:10.1002/cl2.1086 (<https://doi.org/10.1002/cl2.1086>). PMC 8356281 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8356281>). PMID 37131419 (<https://pubmed.ncbi.nlm.nih.gov/37131419>). S2CID 218953088 (<https://api.semanticscholar.org/CorpusID:218953088>).
13. Reiersen AM, Todd RD (April 2008). "Co-occurrence of ADHD and autism spectrum disorders: phenomenology and treatment". *Expert Review of Neurotherapeutics*. **8** (4): 657–669. doi:10.1586/14737175.8.4.657 (<https://doi.org/10.1586/14737175.8.4.657>). PMID 18416666 (<https://pubmed.ncbi.nlm.nih.gov/18416666>). S2CID 1582890 (<https://api.semanticscholar.org/CorpusID:1582890>).
14. DSM 5 ADHD Fact Sheet (<http://www.dsm5.org/Documents/ADHD%20Fact%20Sheet.pdf>) Archived (<https://web.archive.org/web/20150811064504/http://www.dsm5.org/Documents/ADHD%20Fact%20Sheet.pdf>) August 11, 2015, at the Wayback Machine
15. Newberry, Laura (25 April 2023). "Autism and ADHD often go hand-in-hand. What's it like to have 'AuDHD'?" (<https://www.latimes.com/california/newsletter/2023-04-25/adhd-and-autism-group-therapy>). *Los Angeles Times*. Archived (<https://web.archive.org/web/20240405234700/https://www.latimes.com/california/newsletter/2023-04-25/adhd-and-autism-group-therapy>) from the original on 2024-04-05. Retrieved 18 February 2024.
16. "AuDHD (Autism + ADHD) Resources" (<https://neurodivergentcounselling.ca/resources/audhd-resources/>). *Neurodivergent Counselling Services*. 3 October 2021. Archived (<https://web.archive.org/web/20240218124852/https://neurodivergentcounselling.ca/resources/audhd-resources/>) from the original on 18 February 2024. Retrieved 18 February 2024.
17. "Exploring the gendered implications of an adulthood AuDHD diagnosis" (https://www.open-access.bcu.ac.uk/15499/1/QHR_final_accepted_version_190424.pdf) (PDF). *Birmingham City University*. Birmingham City University. 19 April 2024. Retrieved 8 October 2024.
18. Alaghband-rad, Javad; Hajikarim-Hamedani, Arman; Motamed, Mahtab (16 March 2023). "Camouflage and masking behavior in adult autism" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10060524/>). *National Library Of Medicine*. Retrieved 8 October 2024.
19. Norman, Sarah (5 June 2024). "The Different Signs Of AuDHD In Women & Girls" (<https://augmentive.io/blog/signs-audhd-women-girls>). *Augmentive*. Augmentive. Retrieved 8 October 2024.
20. Ramirez, Z (May 1, 2024). "Avoidant Restrictive Food Intake Disorder" (<https://www.ncbi.nlm.nih.gov/books/NBK603710/>). *NIH: National Library of Medicine*. PMID 38753906 (<https://pubmed.ncbi.nlm.nih.gov/38753906>). Retrieved August 30, 2024.
21. Keski-Rahkonen, A (November 1, 2023). "Avoidant-restrictive food intake disorder and autism:

- epidemiology, etiology, complications, treatment, and outcome" (<https://pubmed.ncbi.nlm.nih.gov/37781978/>). *Current Opinion in Psychology*. **36** (6): 438–442. – via NIH: National Library of Medicine.
22. *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition, Text Revision ed.). Washington DC: American Psychiatric Association. 2022. pp. 376–381. ISBN 978-0-89042-575-6.
 23. McElroy SL (2004). "Diagnosing and treating comorbid (complicated) bipolar disorder". *The Journal of Clinical Psychiatry*. **65** (Suppl 15): 35–44. PMID 15554795 (<https://pubmed.ncbi.nlm.nih.gov/15554795/>).
 24. Towbin KE, Pradella A, Gorrindo T, Pine DS, Leibenluft E (June 2005). "Autism spectrum traits in children with mood and anxiety disorders" (<https://zenodo.org/record/1235253>). *Journal of Child and Adolescent Psychopharmacology* (Submitted manuscript). **15** (3): 452–464. doi:10.1089/cap.2005.15.452 (<https://doi.org/10.1089%2Fcap.2005.15.452>). PMID 16092910 (<https://pubmed.ncbi.nlm.nih.gov/16092910/>). Archived (<https://web.archive.org/web/20200818105220/https://zenodo.org/record/1235253>) from the original on 2020-08-18. Retrieved 2018-09-11.
 25. Mayer EA, Padua D, Tillisch K (October 2014). "Altered brain-gut axis in autism: comorbidity or causative mechanisms?". *BioEssays*. **36** (10): 933–939. doi:10.1002/bies.201400075 (<https://doi.org/10.1002%2Fbies.201400075>). PMID 25145752 (<https://pubmed.ncbi.nlm.nih.gov/25145752/>). S2CID 4608380 (<https://api.semanticscholar.org/CorpusID:4608380>).
 26. Williams BL, Hornig M, Buie T, Bauman ML, Cho Paik M, Wick I, et al. (2011). "Impaired carbohydrate digestion and transport and mucosal dysbiosis in the intestines of children with autism and gastrointestinal disturbances" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3174969>). *PLOS ONE*. **6** (9): e24585. Bibcode:2011PLoSO...624585W (<https://ui.adsabs.harvard.edu/abs/2011PLoSO...624585W>). doi:10.1371/journal.pone.0024585 (<https://doi.org/10.1371%2Fjournal.pone.0024585>). PMC 3174969 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3174969>). PMID 21949732 (<https://pubmed.ncbi.nlm.nih.gov/21949732/>).
 27. "Common GI Disorders in Autism" (<https://web.archive.org/web/20201205215616/https://www.focusforhealth.org/common-gi-disorders-in-autism/>). *Focus For Health*. Archived from the original (<https://www.focusforhealth.org/common-gi-disorders-in-autism>) on 2020-12-05. Retrieved 2020-09-06.
 28. Theoharides TC, Stewart JM, Hatzigelaki E, Kolaitis G (2015). "Brain "fog," inflammation and obesity: key aspects of neuropsychiatric disorders improved by luteolin" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4490655>). *Frontiers in Neuroscience*. **9**: 225. doi:10.3389/fnins.2015.00225 (<https://doi.org/10.3389%2Ffnins.2015.00225>). PMC 4490655 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4490655>). PMID 26190965 (<https://pubmed.ncbi.nlm.nih.gov/26190965/>). S2CID 10407088 (<https://api.semanticscholar.org/CorpusID:10407088>).
 29. Stewart ME, Barnard L, Pearson J, Hasan R, O'Brien G (January 2006). "Presentation of depression in autism and Asperger syndrome: a review". *Autism*. **10** (1): 103–116. doi:10.1177/1362361306062013 (<https://doi.org/10.1177%2F1362361306062013>). PMID 16522713 (<https://pubmed.ncbi.nlm.nih.gov/16522713/>). S2CID 12014511 (<https://api.semanticscholar.org/CorpusID:12014511>).
 30. Ghaziuddin M, Ghaziuddin N, Greden J (August 2002). "Depression in persons with autism: implications for research and clinical care". *Journal of Autism and Developmental Disorders*. **32** (4). Kluwer Academic Publishers-Plenum Publishers: 299–306. doi:10.1023/A:1016330802348 (<https://doi.org/10.1023%2FA%3A1016330802348>). eISSN 1573-3432 (<https://search.worldcat.org/issn/1573-3432>). hdl:2027.42/44620 (<https://hdl.handle.net/2027.42%2F44620>). PMID 12199134 (<https://pubmed.ncbi.nlm.nih.gov/12199134/>). S2CID 38817077 (<https://api.semanticscholar.org/CorpusID:38817077>).

[manticscholar.org/CorpusID:38817077](https://www.semanticscholar.org/CorpusID:38817077)).

31. Mash EJ, Barkley RA (2003). *Child Psychopathology* (https://archive.org/details/childpsychopat/ho00mash_735). New York: The Guilford Press. pp. 409 (https://archive.org/details/childpsychopat/ho00mash_735/page/n421)–454. ISBN 9781572306097.
32. McPartland J, Klin A (October 2006). "Asperger's syndrome". *Adolescent Medicine Clinics*. **17** (3): 771–88, abstract xiii. doi:10.1016/j.admecli.2006.06.010 (<https://doi.org/10.1016%2Fj.admecli.2006.06.010>) (inactive 2024-08-30). PMID 17030291 (<https://pubmed.ncbi.nlm.nih.gov/17030291>).
33. Ehlers S, Gillberg C (November 1993). "The epidemiology of Asperger syndrome. A total population study". *Journal of Child Psychology and Psychiatry, and Allied Disciplines*. **34** (8): 1327–1350. doi:10.1111/j.1469-7610.1993.tb02094.x (<https://doi.org/10.1111%2Fj.1469-7610.1993.tb02094.x>). PMID 8294522 (<https://pubmed.ncbi.nlm.nih.gov/8294522>). "Truncated version" (https://web.archive.org/web/20080719235125/http://www.asperger.org/Publications/Ehlers_and_Gillberg_Article.asp). Archived from the original (http://asperger.org/Publications/Ehlers_and_Gillberg_Article.asp) on 2008-07-19. Retrieved 2008-06-15.
34. Klin A (May 2006). "[Autism and Asperger syndrome: an overview]" (<https://doi.org/10.1590%2FS1516-44462006000500002>). *Revista Brasileira de Psiquiatria*. **28** (suppl 1): S3-11. doi:10.1590/S1516-44462006000500002 (<https://doi.org/10.1590%2FS1516-44462006000500002>). PMID 16791390 (<https://pubmed.ncbi.nlm.nih.gov/16791390>).
35. Tuchman R, Rapin I (October 2002). "Epilepsy in autism". *The Lancet. Neurology*. **1** (6): 352–358. doi:10.1016/S1474-4422(02)00160-6 (<https://doi.org/10.1016%2FS1474-4422%2802%2900160-6>). PMID 12849396 (<https://pubmed.ncbi.nlm.nih.gov/12849396>). S2CID 34012901 (<https://api.semanticscholar.org/CorpusID:34012901>).
36. "A Parent's Guide to Autism Spectrum Disorder" (<http://www.nimh.nih.gov/health/publications/a-parents-guide-to-autism-spectrum-disorder/index.shtml>). National Institute of Mental Health. Archived (<https://web.archive.org/web/20160223233600/http://www.nimh.nih.gov/health/publications/a-parents-guide-to-autism-spectrum-disorder/index.shtml>) from the original on 23 February 2016. Retrieved 17 October 2013.
37. Canitano R (February 2007). "Epilepsy in autism spectrum disorders". *European Child & Adolescent Psychiatry* (Review). **16** (1): 61–66. doi:10.1007/s00787-006-0563-2 (<https://doi.org/10.1007%2Fs00787-006-0563-2>). PMID 16932856 (<https://pubmed.ncbi.nlm.nih.gov/16932856>). S2CID 23831292 (<https://api.semanticscholar.org/CorpusID:23831292>).
38. "Overlapping Behavioral Characteristics of FASD's & Related Mental Health Diagnosis" (<http://1.wp.com/www.fasdfamilies.com/wp-content/uploads/2016/02/overlapping-characteristics-sources1.jpg?resize=791%2C1024>). *www.fasdfamilies.com*. Cathy Bruer-Thompson. Retrieved 28 July 2019.
39. Lange, Shannon; Rehm, Jürgen; Anagnostou, Evdokia; Popova, Svetlana (April 2018). "Prevalence of externalizing disorders and Autism Spectrum Disorders among children with Fetal Alcohol Spectrum Disorder: systematic review and meta-analysis" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9221419>). *Biochemistry and Cell Biology*. **96** (2): 241–251. doi:10.1139/bcb-2017-0014 (<https://doi.org/10.1139%2Fbcb-2017-0014>). PMC 9221419 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9221419>). PMID 35741677 (<https://pubmed.ncbi.nlm.nih.gov/35741677>).
40. "Autism and Fragile X Syndrome" (<https://web.archive.org/web/20150903171329/https://fragilex.org/fragile-x-associated-disorders/fragile-x-syndrome/autism-and-fragile-x-syndrome/>). *National Fragile X Foundation*. Archived from the original (<http://www.fragilex.org/fragile-x-associated-disorders/fragile-x-syndrome/autism-and-fragile-x-syndrome/>) on 3 September 2015. Retrieved 3 November 2013.

41. "Gender Dysphoria" (<http://www.dsm5.org/documents/gender%20dysphoria%20fact%20sheet.pdf>) (PDF). American Psychiatric Publishing. Archived (<https://web.archive.org/web/20161120125100/http://www.dsm5.org/documents/gender%20dysphoria%20fact%20sheet.pdf>) (PDF) from the original on November 20, 2016. Retrieved April 13, 2016.
42. Lauden K. "Gender Identity Issues Linked to Autism, ADHD" (<http://www.medscape.com/viewarticle/822077>). MedScape. Archived (<https://web.archive.org/web/20210118040828/https://www.medscape.com/viewarticle/822077>) from the original on January 18, 2021. Retrieved May 8, 2016.
43. Glidden D, Bouman WP, Jones BA, Arcelus J (January 2016). "Gender Dysphoria and Autism Spectrum Disorder: A Systematic Review of the Literature" (https://figshare.com/articles/journal_contribution/9620366). *Sexual Medicine Reviews*. **4** (1): 3–14. doi:10.1016/j.sxmr.2015.10.003 (<https://doi.org/10.1016%2Fj.sxmr.2015.10.003>). PMID 27872002 (<https://pubmed.ncbi.nlm.nih.gov/27872002>). S2CID 3454600 (<https://api.semanticscholar.org/CorpusID:3454600>). Archived (https://web.archive.org/web/20231103124238/https://figshare.com/articles/journal_contribution/Gender_dysphoria_and_autism_spectrum_disorder_a_systematic_review_of_the_literature/9620366) from the original on 2023-11-03. Retrieved 2023-08-20.
44. de Vries AL, Noens IL, Cohen-Kettenis PT, van Berckelaer-Onnes IA, Doreleijers TA (August 2010). "Autism spectrum disorders in gender dysphoric children and adolescents" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2904453>). *Journal of Autism and Developmental Disorders*. **40** (8): 930–936. doi:10.1007/s10803-010-0935-9 (<https://doi.org/10.1007%2Fs10803-010-0935-9>). PMC 2904453 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2904453>). PMID 20094764 (<https://pubmed.ncbi.nlm.nih.gov/20094764>).
45. Van Der Miesen AI, Hurley H, De Vries AL (2016-01-02). "Gender dysphoria and autism spectrum disorder: A narrative review" (<https://doi.org/10.3109%2F09540261.2015.1111199>). *International Review of Psychiatry*. **28** (1): 70–80. doi:10.3109/09540261.2015.1111199 (<https://doi.org/10.3109%2F09540261.2015.1111199>). PMID 26753812 (<https://pubmed.ncbi.nlm.nih.gov/26753812>). S2CID 20918937 (<https://api.semanticscholar.org/CorpusID:20918937>).
46. Casanova EL, Baeza-Velasco C, Buchanan CB, Casanova MF (December 2020). "The Relationship between Autism and Ehlers-Danlos Syndromes/Hypermobility Spectrum Disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7711487>). *Journal of Personalized Medicine*. **10** (4): 260. doi:10.3390/jpm10040260 (<https://doi.org/10.3390%2Fjpm10040260>). PMC 7711487 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7711487>). PMID 33271870 (<https://pubmed.ncbi.nlm.nih.gov/33271870>).
47. Cederlöf M, Larsson H, Lichtenstein P, Almqvist C, Serlachius E, Ludvigsson JF (July 2016). "Nationwide population-based cohort study of psychiatric disorders in individuals with Ehlers-Danlos syndrome or hypermobility syndrome and their siblings" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4932739>). *BMC Psychiatry*. **16**: 207. doi:10.1186/s12888-016-0922-6 (<https://doi.org/10.1186%2Fs12888-016-0922-6>). PMC 4932739 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4932739>). PMID 27377649 (<https://pubmed.ncbi.nlm.nih.gov/27377649>).
48. Dawson M, Mottron L, Gernsbacher MA (2008). "Learning in autism" (http://gernsbacherlab.org/wp-content/uploads/papers/1/Dawson_AutisticLearning.pdf) (PDF). In Byrne JH (-in-chief), Roediger HL III (vol.) (ed.). *Learning and Memory: A Comprehensive Reference*. Vol. 2. Academic Press. pp. 759–772. doi:10.1016/B978-012370509-9.00152-2 (<https://doi.org/10.1016%2FB978-012370509-9.00152-2>). ISBN 978-0-12-370504-4. Archived (https://web.archive.org/web/20180413012927/http://www.gernsbacherlab.org/wp-content/uploads/papers/1/Dawson_AutisticLearning.pdf) (PDF) from the original on 2018-04-13. Retrieved 2013-02-16.
49. Chakrabarti S, Fombonne E (June 2001). "Pervasive developmental disorders in preschool children". *JAMA*. **285** (24): 3093–3099. doi:10.1001/jama.285.24.3093 (<https://doi.org/10.1001%2Fjama.285.24.3093>). PMID 11427137 (<https://pubmed.ncbi.nlm.nih.gov/11427137>).

50. O'Brien G, Pearson J (June 2004). "Autism and learning disability". *Autism (Review)*. **8** (2): 125–140. doi:10.1177/1362361304042718 (<https://doi.org/10.1177%2F1362361304042718>). PMID 15165430 (<https://pubmed.ncbi.nlm.nih.gov/15165430>). S2CID 17372893 (<https://api.semanticscholar.org/CorpusID:17372893>).
51. Edelson MG (2006). "Are the majority of children with autism mentally retarded? a systematic evaluation of the data" (<https://web.archive.org/web/20070704201502/http://www.willamette.edu/dept/comm/reprint/edelson/>). *Focus Autism Other Dev Disabl*. **21** (2): 66–83. doi:10.1177/10883576060210020301 (<https://doi.org/10.1177%2F10883576060210020301>). S2CID 219975805 (<https://api.semanticscholar.org/CorpusID:219975805>). Archived from the original (<http://www.willamette.edu/dept/comm/reprint/edelson/>) on 2007-07-04. Retrieved 2007-04-15.
52. Skuse DH (August 2007). "Rethinking the nature of genetic vulnerability to autistic spectrum disorders". *Trends in Genetics*. **23** (8): 387–395. doi:10.1016/j.tig.2007.06.003 (<https://doi.org/10.1016%2Fj.tig.2007.06.003>). PMID 17630015 (<https://pubmed.ncbi.nlm.nih.gov/17630015>).
53. "Diseases & Conditions | Features | CDC" (<https://www.cdc.gov/features/dsautismdata/>). 2018-04-04. Archived (<https://web.archive.org/web/20171119060937/https://www.cdc.gov/features/dsautismdata/>) from the original on 2017-11-19. Retrieved 2017-09-09.
54. Siasos G, Tsigkou V, Kosmopoulos M, Theodosiadis D, Simantiris S, Tagkou NM, et al. (June 2018). "Mitochondria and cardiovascular diseases-from pathophysiology to treatment" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6046286>). *Annals of Translational Medicine*. **6** (12): 256. doi:10.21037/atm.2018.06.21 (<https://doi.org/10.21037%2Fatm.2018.06.21>). PMC 6046286 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6046286>). PMID 30069458 (<https://pubmed.ncbi.nlm.nih.gov/30069458>).
55. El-Hattab AW, Scaglia F (2016). "Mitochondrial Cardiomyopathies" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4958622>). *Frontiers in Cardiovascular Medicine*. **3**: 25. doi:10.3389/fcvm.2016.00025 (<https://doi.org/10.3389%2Ffcvm.2016.00025>). PMC 4958622 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4958622>). PMID 27504452 (<https://pubmed.ncbi.nlm.nih.gov/27504452>).
56. Finsterer J, Zarrouk Mahjoub S (January 2012). "Mitochondrial toxicity of antiepileptic drugs and their tolerability in mitochondrial disorders". *Expert Opinion on Drug Metabolism & Toxicology*. **8** (1): 71–79. doi:10.1517/17425255.2012.644535 (<https://doi.org/10.1517%2F17425255.2012.644535>). PMID 22149023 (<https://pubmed.ncbi.nlm.nih.gov/22149023>). S2CID 12147144 (<https://api.semanticscholar.org/CorpusID:12147144>).
57. Mithal DS, Kurz JE (November 2017). "Anticonvulsant Medications in Mitochondrial Disease" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5681459>). *Pediatric Neurology Briefs*. **31** (3): 9. doi:10.15844/pedneurbriefs-31-3-3 (<https://doi.org/10.15844%2Fpedneurbriefs-31-3-3>). PMC 5681459 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5681459>). PMID 29184381 (<https://pubmed.ncbi.nlm.nih.gov/29184381>).
58. Finsterer J, Segall L (April 2010). "Drugs interfering with mitochondrial disorders". *Drug and Chemical Toxicology*. **33** (2): 138–151. doi:10.3109/01480540903207076 (<https://doi.org/10.3109%2F01480540903207076>). PMID 19839725 (<https://pubmed.ncbi.nlm.nih.gov/19839725>). S2CID 15913362 (<https://api.semanticscholar.org/CorpusID:15913362>).
59. de Oliveira MR (2015). "Vitamin A and Retinoids as Mitochondrial Toxicants" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4452429>). *Oxidative Medicine and Cellular Longevity*. **2015**: 140267. doi:10.1155/2015/140267 (<https://doi.org/10.1155%2F2015%2F140267>). PMC 4452429 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4452429>). PMID 26078802 (<https://pubmed.ncbi.nlm.nih.gov/26078802>).
60. "Neurofibromatosis type 1 - Symptoms" (<https://www.nhs.uk/conditions/neurofibromatosis-type-1/symptoms/>). *nhs.uk*. 2018-06-08. Archived (<https://web.archive.org/web/20150925015544/htt>

[p://www.nhs.uk/Conditions/Neurofibromatosis/Pages/Symptoms.aspx](https://www.nhs.uk/Conditions/Neurofibromatosis/Pages/Symptoms.aspx)) from the original on 2015-09-25. Retrieved 2020-09-21.

61. Pardo CA, Vargas DL, Zimmerman AW (December 2005). "Immunity, neuroglia and neuroinflammation in autism". *International Review of Psychiatry*. **17** (6): 485–495. CiteSeerX [10.1.1.504.8397](https://citeseerx.ist.psu.edu/viewdoc/summary?doi=10.1.1.504.8397) (<https://citeseerx.ist.psu.edu/viewdoc/summary?doi=10.1.1.504.8397>). doi:[10.1080/02646830500381930](https://doi.org/10.1080/02646830500381930) (<https://doi.org/10.1080/02646830500381930>). PMID [16401547](https://pubmed.ncbi.nlm.nih.gov/16401547/) (<https://pubmed.ncbi.nlm.nih.gov/16401547/>). S2CID [849995](https://api.semanticscholar.org/CorpusID:849995) (<https://api.semanticscholar.org/CorpusID:849995>).
62. Gesundheit B, Rosenzweig JP, Naor D, Lerer B, Zachor DA, Procházka V, et al. (August 2013). "Immunological and autoimmune considerations of Autism Spectrum Disorders". *Journal of Autoimmunity*. **44**: 1–7. doi:[10.1016/j.jaut.2013.05.005](https://doi.org/10.1016/j.jaut.2013.05.005) (<https://doi.org/10.1016/j.jaut.2013.05.005>). PMID [23867105](https://pubmed.ncbi.nlm.nih.gov/23867105/) (<https://pubmed.ncbi.nlm.nih.gov/23867105/>).
63. Frye RE, Rossignol DA (May 2011). "Mitochondrial dysfunction can connect the diverse medical symptoms associated with autism spectrum disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3179978>). *Pediatric Research*. **69** (5 Pt 2): 41R–47R. doi:[10.1203/PDR.0b013e318212f16b](https://doi.org/10.1203/PDR.0b013e318212f16b) (<https://doi.org/10.1203/PDR.0b013e318212f16b>). PMC [3179978](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3179978) (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3179978>). PMID [21289536](https://pubmed.ncbi.nlm.nih.gov/21289536/) (<https://pubmed.ncbi.nlm.nih.gov/21289536/>).
64. Luigetti M, Sauchelli D, Primiano G, Cuccagna C, Bernardo D, Lo Monaco M, Servidei S (June 2016). "Peripheral neuropathy is a common manifestation of mitochondrial diseases: a single-centre experience". *European Journal of Neurology*. **23** (6): 1020–1027. doi:[10.1111/ene.12954](https://doi.org/10.1111/ene.12954) (<https://doi.org/10.1111/ene.12954>). PMID [26822221](https://pubmed.ncbi.nlm.nih.gov/26822221/) (<https://pubmed.ncbi.nlm.nih.gov/26822221/>). S2CID [3914392](https://api.semanticscholar.org/CorpusID:3914392) (<https://api.semanticscholar.org/CorpusID:3914392>).
65. Luigetti M, Primiano G, Cuccagna C, Bernardo D, Sauchelli D, Vollono C, Servidei S (August 2018). "Small fibre neuropathy in mitochondrial diseases explored with sudoscan". *Clinical Neurophysiology*. **129** (8): 1618–1623. doi:[10.1016/j.clinph.2018.04.755](https://doi.org/10.1016/j.clinph.2018.04.755) (<https://doi.org/10.1016/j.clinph.2018.04.755>). PMID [29890373](https://pubmed.ncbi.nlm.nih.gov/29890373/) (<https://pubmed.ncbi.nlm.nih.gov/29890373/>). S2CID [48364519](https://api.semanticscholar.org/CorpusID:48364519) (<https://api.semanticscholar.org/CorpusID:48364519>).
66. Fisher, Prudence W. (February 1, 2022). "Systematic Review: Nonverbal Learning Disability" ([https://www.jaacap.org/article/S0890-8567\(21\)00234-3/abstract](https://www.jaacap.org/article/S0890-8567(21)00234-3/abstract)). *Journal of the American Academy of Child & Adolescent Psychiatry*. **61** (2): 159–186. PMID [33892110](https://pubmed.ncbi.nlm.nih.gov/33892110/) (<https://pubmed.ncbi.nlm.nih.gov/33892110/>).
67. Russell AJ, Jassi A, Fullana MA, Mack H, Johnston K, Heyman I, et al. (August 2013). "Cognitive behavior therapy for comorbid obsessive-compulsive disorder in high-functioning autism spectrum disorders: a randomized controlled trial" (https://purehost.bath.ac.uk/ws/files/84038359/Accepted_version.pdf) (PDF). *Depression and Anxiety*. **30** (8): 697–708. doi:[10.1002/da.22053](https://doi.org/10.1002/da.22053) (<https://doi.org/10.1002/da.22053>). PMID [23389964](https://pubmed.ncbi.nlm.nih.gov/23389964/) (<https://pubmed.ncbi.nlm.nih.gov/23389964/>). S2CID [13587266](https://api.semanticscholar.org/CorpusID:13587266) (<https://api.semanticscholar.org/CorpusID:13587266>). Archived (https://web.archive.org/web/20230908095154/https://purehost.bath.ac.uk/ws/files/84038359/Accepted_version.pdf) (PDF) from the original on 2023-09-08. Retrieved 2023-08-20.
68. Gillberg C, Billstedt E (November 2000). "Autism and Asperger syndrome: coexistence with other clinical disorders". *Acta Psychiatrica Scandinavica*. **102** (5): 321–330. doi:[10.1034/j.1600-0447.2000.102005321.x](https://doi.org/10.1034/j.1600-0447.2000.102005321.x) (<https://doi.org/10.1034/j.1600-0447.2000.102005321.x>). PMID [11098802](https://pubmed.ncbi.nlm.nih.gov/11098802/) (<https://pubmed.ncbi.nlm.nih.gov/11098802/>). S2CID [40070782](https://api.semanticscholar.org/CorpusID:40070782) (<https://api.semanticscholar.org/CorpusID:40070782>).
69. Fitzgerald M, Corvin A (2001-07-01). "Diagnosis and differential diagnosis of Asperger syndrome" (<https://doi.org/10.1192/apt.7.4.310>). *Advances in Psychiatric Treatment*. **7** (4): 310–318. doi:[10.1192/apt.7.4.310](https://doi.org/10.1192/apt.7.4.310) (<https://doi.org/10.1192/apt.7.4.310>). ISSN [1355-5146](https://doi.org/10.1192/apt.7.4.310) (h

<https://search.worldcat.org/issn/1355-5146>).

70. Hofvander B, Delorme R, Chaste P, Nydén A, Wentz E, Ståhlberg O, et al. (June 2009). "Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2705351>). *BMC Psychiatry*. **9** (1): 35. doi:10.1186/1471-244x-9-35 (<https://doi.org/10.1186%2F1471-244x-9-35>). PMC 2705351 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2705351>). PMID 19515234 (<https://pubmed.ncbi.nlm.nih.gov/19515234>).
71. Rapoport J, Chavez A, Greenstein D, Addington A, Gogtay N (January 2009). "Autism spectrum disorders and childhood-onset schizophrenia: clinical and biological contributions to a relation revisited" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2664646>). *Journal of the American Academy of Child and Adolescent Psychiatry*. **48** (1): 10–18. doi:10.1097/CHI.0b013e31818b1c63 (<https://doi.org/10.1097%2FCHI.0b013e31818b1c63>). PMC 2664646 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2664646>). PMID 19218893 (<https://pubmed.ncbi.nlm.nih.gov/19218893>).
72. Mouridsen SE, Rich B, Isager T (2008). "Psychiatric disorders in adults diagnosed as children with atypical autism. A case control study". *Journal of Neural Transmission*. **115** (1): 135–138. doi:10.1007/s00702-007-0798-1 (<https://doi.org/10.1007%2Fs00702-007-0798-1>). PMID 17768593 (<https://pubmed.ncbi.nlm.nih.gov/17768593>). S2CID 34532555 (<https://api.semanticscholar.org/CorpusID:34532555>).
73. Larson FV, Wagner AP, Jones PB, Tantam D, Lai MC, Baron-Cohen S, Holland AJ (April 2017). "Psychosis in autism: comparison of the features of both conditions in a dually affected cohort" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5376719>). *The British Journal of Psychiatry*. **210** (4): 269–275. doi:10.1192/bjp.bp.116.187682 (<https://doi.org/10.1192%2Fbjp.bp.116.187682>). PMC 5376719 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5376719>). PMID 27979819 (<https://pubmed.ncbi.nlm.nih.gov/27979819>).
74. Gandal MJ, Anderson RL, Billingslea EN, Carlson GC, Roberts TP, Siegel SJ (August 2012). "Mice with reduced NMDA receptor expression: more consistent with autism than schizophrenia?" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3808979>). *Genes, Brain and Behavior*. **11** (6): 740–750. doi:10.1111/j.1601-183X.2012.00816.x (<https://doi.org/10.1111%2Fj.1601-183X.2012.00816.x>). PMC 3808979 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3808979>). PMID 22726567 (<https://pubmed.ncbi.nlm.nih.gov/22726567>).
75. Akhtar S (October 1987). "Schizoid personality disorder: a synthesis of developmental, dynamic, and descriptive features" (<https://books.google.com/books?id=bU0eAAAAQBAJ&pg=PA121>). *American Journal of Psychotherapy*. **41** (4): 499–518. doi:10.1176/appi.psychotherapy.1987.41.4.499 (<https://doi.org/10.1176%2Fappi.psychotherapy.1987.41.4.499>). ISBN 9781461627685. PMID 3324773 (<https://pubmed.ncbi.nlm.nih.gov/3324773>). Archived (<https://web.archive.org/web/20170731160223/https://books.google.de/books?id=bU0eAAAAQBAJ&pg=PA121>) from the original on 2017-07-31. Retrieved 2017-02-10.
76. "Schizoid Personality Disorder" (<https://www.nlm.nih.gov/medlineplus/ency/article/000920.htm>). *MedlinePlus*. National Library of Medicine. 2014. Archived (<https://web.archive.org/web/20160705124629/https://www.nlm.nih.gov/medlineplus/ency/article/000920.htm>) from the original on 2016-07-05. Retrieved 2019-08-21.
77. Tantam D (December 1988). "Lifelong eccentricity and social isolation. II: Asperger's syndrome or schizoid personality disorder?". *The British Journal of Psychiatry*. **153**: 783–791. doi:10.1192/bjp.153.6.783 (<https://doi.org/10.1192%2Fbjp.153.6.783>). PMID 3256377 (<https://pubmed.ncbi.nlm.nih.gov/3256377>). S2CID 39433805 (<https://api.semanticscholar.org/CorpusID:39433805>).
78. Ekleberry SC (2008). "Cluster A - Schizoid Personality Disorder and Substance Use Disorders" (<https://books.google.com/books?id=O5HY1xcfiEcC&pg=PA31>). *Integrated Treatment for Co-*

79. Lugnegård T, Hallerbäck MU, Gillberg C (May 2012). "Personality disorders and autism spectrum disorders: what are the connections?". *Comprehensive Psychiatry*. **53** (4): 333–340. doi:10.1016/j.comppsy.2011.05.014 (<https://doi.org/10.1016%2Fj.comppsy.2011.05.014>). PMID 21821235 (<https://pubmed.ncbi.nlm.nih.gov/21821235>).
80. Kuhn R (September 2004). "Eugen Bleuler's concepts of psychopathology". *History of Psychiatry*. **15** (59 Pt 3): 361–366. doi:10.1177/0957154X04044603 (<https://doi.org/10.1177%2F0957154X04044603>). PMID 15386868 (<https://pubmed.ncbi.nlm.nih.gov/15386868>). S2CID 5317716 (<https://api.semanticscholar.org/CorpusID:5317716>). The quote is a translation of Bleuler's 1910 original.
81. Crespi B, Badcock C (June 2008). "Psychosis and autism as diametrical disorders of the social brain" (<http://eprints.lse.ac.uk/21571/1/Psychosis%20and%20autism%20as%20diametrical%20disorders%20of%20the%20social%20brain%20%28LSERO%29.pdf>) (PDF). *The Behavioral and Brain Sciences*. **31** (3): 241–261. doi:10.1017/S0140525X08004214 (<https://doi.org/10.1017%2FS0140525X08004214>). PMID 18578904 (<https://pubmed.ncbi.nlm.nih.gov/18578904>). Archived (<https://web.archive.org/web/20200412200311/http://eprints.lse.ac.uk/21571/1/Psychosis%20and%20autism%20as%20diametrical%20disorders%20of%20the%20social%20brain%20%28LSERO%29.pdf>) (PDF) from the original on 2020-04-12. Retrieved 2019-11-29.
82. Crespi B, Stead P, Elliot M (January 2010). "Evolution in health and medicine Sackler colloquium: Comparative genomics of autism and schizophrenia" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2868282>). *Proceedings of the National Academy of Sciences of the United States of America*. **107** (Suppl 1): 1736–1741. Bibcode:2010PNAS..107.1736C (<https://ui.adsabs.harvard.edu/abs/2010PNAS..107.1736C>). doi:10.1073/pnas.0906080106 (<https://doi.org/10.1073%2Fpnas.0906080106>). PMC 2868282 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2868282>). PMID 19955444 (<https://pubmed.ncbi.nlm.nih.gov/19955444>).
83. Ciaramidaro A, Bölte S, Schlitt S, Hainz D, Poustka F, Weber B, et al. (January 2015). "Schizophrenia and autism as contrasting minds: neural evidence for the hypo-hyper-intentionality hypothesis" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4266299>). *Schizophrenia Bulletin*. **41** (1): 171–179. doi:10.1093/schbul/sbu124 (<https://doi.org/10.1093%2Fschbul%2Fsbu124>). PMC 4266299 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4266299>). PMID 25210055 (<https://pubmed.ncbi.nlm.nih.gov/25210055>).
84. Rogers SJ, Ozonoff S (December 2005). "Annotation: what do we know about sensory dysfunction in autism? A critical review of the empirical evidence". *Journal of Child Psychology and Psychiatry, and Allied Disciplines*. **46** (12): 1255–1268. doi:10.1111/j.1469-7610.2005.01431.x (<https://doi.org/10.1111%2Fj.1469-7610.2005.01431.x>). PMID 16313426 (<https://pubmed.ncbi.nlm.nih.gov/16313426>).
85. Baranek GT (October 2002). "Efficacy of sensory and motor interventions for children with autism". *Journal of Autism and Developmental Disorders* (Review). **32** (5): 397–422. doi:10.1023/A:1020541906063 (<https://doi.org/10.1023%2FA%3A1020541906063>). PMID 12463517 (<https://pubmed.ncbi.nlm.nih.gov/12463517>). S2CID 16449130 (<https://api.semanticscholar.org/CorpusID:16449130>).
86. Balasco L, Provenzano G, Bozzi Y (28 January 2020). "Sensory Abnormalities in Autism Spectrum Disorders: A Focus on the Tactile Domain, From Genetic Mouse Models to the Clinic" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6997554>). *Frontiers in Psychiatry*. **10**: 1016. doi:10.3389/fpsy.2019.01016 (<https://doi.org/10.3389%2Ffpsyt.2019.01016>). PMC 6997554 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6997554>). PMID 32047448 (<https://pubmed.ncbi.nlm.nih.gov/32047448>).
87. Koiovic N, Ben Hadid L, Franchini M, Schaer M (September 2019). "Sensorv Processing

- Issues and Their Association with Social Difficulties in Children with Autism Spectrum Disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6833094>). *Journal of Clinical Medicine*. **8** (10): 1508. doi:10.3390/jcm8101508 (<https://doi.org/10.3390%2Fjcm8101508>). PMC 6833094 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6833094>). PMID 31547076 (<https://pubmed.ncbi.nlm.nih.gov/31547076>).
88. Perez Repetto L, Jasmin E, Fombonne E, Gisel E, Couture M (27 August 2017). "Longitudinal Study of Sensory Features in Children with Autism Spectrum Disorder" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5592014>). *Autism Research and Treatment*. **2017**: 1934701. doi:10.1155/2017/1934701 (<https://doi.org/10.1155%2F2017%2F1934701>). PMC 5592014 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5592014>). PMID 28932599 (<https://pubmed.ncbi.nlm.nih.gov/28932599>).
 89. Ming X, Brimacombe M, Wagner GC (October 2007). "Prevalence of motor impairment in autism spectrum disorders". *Brain & Development*. **29** (9): 565–570. doi:10.1016/j.braindev.2007.03.002 (<https://doi.org/10.1016%2Fj.braindev.2007.03.002>). PMID 17467940 (<https://pubmed.ncbi.nlm.nih.gov/17467940>). S2CID 9648682 (<https://api.semanticscholar.org/CorpusID:9648682>).
 90. Carry Terra, *Adults on the Spectrum: These are your Feet on Asperger's* <http://www.aspiestrategy.com/2013/02/adults-on-spectrum-these-are-your-feet.html?m=1> Archived (<https://web.archive.org/web/20160923123840/http://www.aspiestrategy.com/2013/02/adults-on-spectrum-these-are-your-feet.html?m=1>) 2016-09-23 at the Wayback Machine
 91. Malow BA, Byars K, Johnson K, Weiss S, Bernal P, Goldman SE, et al. (November 2012). "A practice pathway for the identification, evaluation, and management of insomnia in children and adolescents with autism spectrum disorders" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9923883>). *Pediatrics*. **130** (Suppl 2): S106–S124. doi:10.1542/peds.2012-0900I (<https://doi.org/10.1542%2Fpeds.2012-0900I>). PMC 9923883 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9923883>). PMID 23118242 (<https://pubmed.ncbi.nlm.nih.gov/23118242>). S2CID 15066964 (<https://api.semanticscholar.org/CorpusID:15066964>).
 92. "Poor sleep in children with autism associated with problematic behavior during the day" (<https://web.archive.org/web/20150205231729/http://www.truthlyapp.com/truths/495-poor-sleep-in-children-with-autism-associated-with-problematic-behavior-during-the-day>). *Truthly*. Archived from the original (<http://www.truthlyapp.com/truths/495-poor-sleep-in-children-with-autism-associated-with-problematic-behavior-during-the-day>) on 5 February 2015. Retrieved 5 February 2015.
 93. Johnson KP, Malow BA (October 2008). "Assessment and pharmacologic treatment of sleep disturbance in autism". *Child and Adolescent Psychiatric Clinics of North America*. **17** (4): 773–85, viii. doi:10.1016/j.chc.2008.06.006 (<https://doi.org/10.1016%2Fj.chc.2008.06.006>). PMID 18775369 (<https://pubmed.ncbi.nlm.nih.gov/18775369>).
 94. Wu, Zhou-yue (October 2020). "Autism spectrum disorder (ASD): Disturbance of the melatonin system and its implications" (<https://doi.org/10.1016%2Fj.biopha.2020.110496>). *Biomedicine & Pharmacotherapy*. **130**. doi:10.1016/j.biopha.2020.110496 (<https://doi.org/10.1016%2Fj.biopha.2020.110496>). PMID 32682113 (<https://pubmed.ncbi.nlm.nih.gov/32682113>).
 95. Stewart PA, Hyman SL, Schmidt BL, Macklin EA, Reynolds A, Johnson CR, et al. (August 2015). "Dietary Supplementation in Children with Autism Spectrum Disorders: Common, Insufficient, and Excessive". *Journal of the Academy of Nutrition and Dietetics*. **115** (8): 1237–1248. doi:10.1016/j.jand.2015.03.026 (<https://doi.org/10.1016%2Fj.jand.2015.03.026>). PMID 26052041 (<https://pubmed.ncbi.nlm.nih.gov/26052041>).
 96. "Melatonin supplementation associated with improved sleep and behavior in children with autism" (<https://web.archive.org/web/20150205230007/http://www.truthlyapp.com/truths/496-melatonin-supplementation-associated-with-improved-sleep-and-behavior-in-children-with-autis>

- m). *Truthly*. Archived from the original (<http://www.truthlyapp.com/truths/496-melatonin-supplementation-associated-with-improved-sleep-and-behavior-in-children-with-autism>) on 5 February 2015. Retrieved 5 February 2015.
97. "More Evidence that Melatonin Eases Autism-Associated Insomnia" (<http://www.autismspeaks.org/science/science-news/more-evidence-melatonin-eases-autism-associated-insomnia>). *Autism Speaks*. Archived (<https://web.archive.org/web/20150205234816/http://www.autismspeaks.org/science/science-news/more-evidence-melatonin-eases-autism-associated-insomnia>) from the original on 5 February 2015. Retrieved 5 February 2015.
 98. Williams ZJ (2021). "Prevalence of Strabismus in Individuals on the Autism Spectrum: A Meta-analysis". *medRxiv* 10.1101/2021.07.13.21260452v1 (<https://www.medrxiv.org/content/10.1101/2021.07.13.21260452v1>).
 99. Danesh AA, Lang D, Kaf W, Andreassen WD, Scott J, Eshraghi AA (October 2015). "Tinnitus and hyperacusis in autism spectrum disorders with emphasis on high functioning individuals diagnosed with Asperger's Syndrome". *International Journal of Pediatric Otorhinolaryngology*. **79** (10): 1683–1688. doi:10.1016/j.ijporl.2015.07.024 (<https://doi.org/10.1016%2Fj.ijporl.2015.07.024>). PMID 26243502 (<https://pubmed.ncbi.nlm.nih.gov/26243502>).
 100. Smalley SL (October 1998). "Autism and tuberous sclerosis". *Journal of Autism and Developmental Disorders*. **28** (5): 407–414. doi:10.1023/A:1026052421693 (<https://doi.org/10.1023%2FA%3A1026052421693>). PMID 9813776 (<https://pubmed.ncbi.nlm.nih.gov/9813776>). S2CID 36023695 (<https://api.semanticscholar.org/CorpusID:36023695>).
 101. Harrison JE, Bolton PF (September 1997). "Annotation: tuberous sclerosis". *Journal of Child Psychology and Psychiatry, and Allied Disciplines*. **38** (6): 603–614. doi:10.1111/j.1469-7610.1997.tb01687.x (<https://doi.org/10.1111%2Fj.1469-7610.1997.tb01687.x>). PMID 9315970 (<https://pubmed.ncbi.nlm.nih.gov/9315970>).
 102. Wolstencroft J, Mandy W, Skuse D (December 6, 2022). "Mental health and neurodevelopment in children and adolescents with Turner syndrome" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9730007>). *Women's Health*. **18**: 17455057221133635. doi:10.1177/17455057221133635 (<https://doi.org/10.1177%2F17455057221133635>). PMC 9730007 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9730007>). PMID 36472167 (<https://pubmed.ncbi.nlm.nih.gov/36472167>).
 103. Endres D, Dersch R, Stich O, Buchwald A, Perlov E, Feige B, et al. (2016). "Vitamin D Deficiency in Adult Patients with Schizophreniform and Autism Spectrum Syndromes: A One-Year Cohort Study at a German Tertiary Care Hospital" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5052261>). *Frontiers in Psychiatry*. **7**: 168. doi:10.3389/fpsy.2016.00168 (<https://doi.org/10.3389%2Fpsy.2016.00168>). PMC 5052261 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5052261>). PMID 27766084 (<https://pubmed.ncbi.nlm.nih.gov/27766084>).
 104. "Science Blog | Autism Speaks" (<https://www.autismspeaks.org/science/science-news/study-vitamin-b12-levels-low-brains-affected-autism-or-schizophrenia>). Archived (<https://web.archive.org/web/20180805052509/https://www.autismspeaks.org/science/science-news/study-vitamin-b12-levels-low-brains-affected-autism-or-schizophrenia>) from the original on 2018-08-05. Retrieved 2018-12-20.
 105. Zhang Y, Hodgson NW, Trivedi MS, Abdolmaleky HM, Fournier M, Cuenod M, et al. (2016). "Decreased Brain Levels of Vitamin B12 in Aging, Autism and Schizophrenia" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4723262>). *PLOS ONE*. **11** (1): e0146797. Bibcode:2016PLoSO..1146797Z (<https://ui.adsabs.harvard.edu/abs/2016PLoSO..1146797Z>). doi:10.1371/journal.pone.0146797 (<https://doi.org/10.1371%2Fjournal.pone.0146797>). PMC 4723262 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4723262>). PMID 26799654 (<https://pubmed.ncbi.nlm.nih.gov/26799654>).
 106. "B12 Deficiency May be More Widespread Than Thought : USDA ARS" (<https://web.archive.org/web/20160804120552/http://www.ars.usda.gov/doc/2000/000802.htm>). Archived from the

[g/web/2010004120002/http://www.ars.usda.gov/news-events/news/research-news/2000/b12-deficiency-may-be-more-widespread-than-thought/](http://www.ars.usda.gov/news-events/news/research-news/2000/b12-deficiency-may-be-more-widespread-than-thought/) on 2016-08-04. Retrieved 2018-12-20.

107. Sun C, Zou M, Zhao D, Xia W, Wu L (June 2016). "Efficacy of Folic Acid Supplementation in Autistic Children Participating in Structured Teaching: An Open-Label Trial" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4924178>). *Nutrients*. **8** (6): 337. doi:10.3390/nu8060337 (<https://doi.org/10.3390%2Fnu8060337>). PMC 4924178 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4924178>). PMID 27338456 (<https://pubmed.ncbi.nlm.nih.gov/27338456>).
108. Frye RE, Slattery J, Delhey L, Furgerson B, Strickland T, Tippet M, et al. (February 2018). "Folinic acid improves verbal communication in children with autism and language impairment: a randomized double-blind placebo-controlled trial" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5794882>). *Molecular Psychiatry*. **23** (2): 247–256. doi:10.1038/mp.2016.168 (<https://doi.org/10.1038%2Fmp.2016.168>). PMC 5794882 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5794882>). PMID 27752075 (<https://pubmed.ncbi.nlm.nih.gov/27752075>).
109. Yasuda H, Tsutsui T (November 2013). "Assessment of infantile mineral imbalances in autism spectrum disorders (ASDs)" (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3863885>). *International Journal of Environmental Research and Public Health*. **10** (11): 6027–6043. doi:10.3390/ijerph10116027 (<https://doi.org/10.3390%2Fijerph10116027>). PMC 3863885 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3863885>). PMID 24284360 (<https://pubmed.ncbi.nlm.nih.gov/24284360>).
110. Matson JL, Nebel-Schwalm MS (2007). "Comorbid psychopathology with autism spectrum disorder in children: an overview". *Research in Developmental Disabilities*. **28** (4): 341–352. CiteSeerX 10.1.1.697.4938 (<https://citeseerx.ist.psu.edu/viewdoc/summary?doi=10.1.1.697.4938>). doi:10.1016/j.ridd.2005.12.004 (<https://doi.org/10.1016%2Fj.ridd.2005.12.004>). PMID 16765022 (<https://pubmed.ncbi.nlm.nih.gov/16765022>).

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