

US Autism Rate Is Now 1 in 44

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STORY AT-A-GLANCE

- › In the mid-1960s, the autism rate was 1 in 10,000. By 2012, it had shot up to 1 in 88. In 2016, it was 1 in 68, and now it's 1 in 44 8-year-olds
- › Rates vary widely from state to state. In California, where incidence is the highest, 1 in 26 8-year-olds has a diagnosis of autism. Missouri, which has the lowest incidence, has a rate of 1 in 60
- › Improved diagnosis alone cannot explain this trend. Genetics play only a minor role, and even then, primarily only when combined with toxic exposures
- › Glyphosate appears to be a leading contributor to autism
- › Other contributing factors include maternal antibodies associated with autoimmune diseases, maternal immune system overactivation and mitochondrial dysfunction (typically involving electron transport chain overactivity). Mitochondrial abnormalities are suspected of being involved in as many as 80% of cases

In the mid-1960s, the autism rate was 1 in 10,000.¹ By 2012, it had risen to 1 in 88,² then 1 in 68 as of 2016,³ and now, we're looking at an autism rate of 1 in 44 8-year-olds, although rates vary widely from state to state.⁴

In California, where incidence is the highest, 1 in 26 8-year-olds has a diagnosis of autism. Missouri, which has the lowest incidence, has a rate of 1 in 60. According to the

U.S. Centers for Disease Control and Prevention, the dramatic uptick is in part due to improved and more comprehensive identification and diagnosis.

However, improved diagnosis alone cannot explain this trend. One in 44 children were not autistic in the 1980s, with or without diagnosis. No, something is going on. One or more environmental factors are clearly having a devastating impact on our children.

Mitochondrial Dysfunction in Autism

According to an October 2020 scientific review⁵ in the journal *Seminars in Pediatric Neurology*, “Several lines of evidence implicate mitochondria in the pathophysiology of autism spectrum disorder (ASD).” For years, autism was believed to be due to genetics, but empirical studies have proven genetic predispositions play a very small role.

“ More recent investigations looking at mitochondrial biomarkers and electron transport chain activity suggest mitochondrial abnormalities may be involved in as many as 80% of cases.”

The authors point to “novel abnormalities” in mitochondrial function that have been found in autistic children, and the fact that treatments targeting mitochondrial dysfunction, such as L-carnitine supplementation and a ketogenic diet, have been fruitful.

In 2007, approximately 4% of autistic children could be diagnosed with a definite mitochondrial disease. More recent investigations looking at mitochondrial biomarkers and electron transport chain activity suggest mitochondrial abnormalities may be involved in as many as 80% of cases. Biomarkers of mitochondrial dysfunction include elevated levels of:

Lactate

Pyruvate

Alanine	Creatine kinase
Ubiquinone	Acyl-carnitines
Carnitine (low levels being an indication of dysfunction)	

As noted by the authors, this is “remarkably interesting” because mitochondria are “very vulnerable to environmental factors.” In other words, something in our environment is triggering mitochondrial dysfunction in an ever-growing percentage of children. The question is what?

Children whose autism is rooted in mitochondrial dysfunction will typically have a key set of symptoms that include:

- Fatigability
- Gastrointestinal disorders
- Seizures and/or epilepsy
- Motor delay and/or ataxia and/or muscle weakness
- Unusual neurodevelopmental regression, including multiple regressions or regression later than commonly associated with ASD

Mitochondrial Overactivity

While classic mitochondrial disease is defined, at least in part, by severe deficits in electron transport chain activity, in children with ASD, mitochondrial dysfunction tends to involve overactivity. According to the authors:⁶

“... we have shown that about one-third of LCLs [lymphoblastoid cell lines] derived from children with ASD repeatedly show elevated respiratory rates, approximately 200% of controls, for respiratory parameters associated with adenosine triphosphate production.

We have also demonstrated the consequences of elevated respiratory rates; specifically, this subset of ASD LCLs are more sensitive to acute exposure to reactive oxygen species (ROS) such that respiratory rates drop precipitously with acute increases in ROS.

We hypothesized these changes represent an adaptive response to previous environmental exposures, a phenomenon known as mitoplasticity.

To support this hypothesis, we demonstrated that elevated respiratory rates can be induced in LCLs with prolonged exposure (96 hours) to mild ROS, a microenvironment that simulates the effect of environmental toxicants on mitochondria.

Additionally, we have demonstrated that this subset of LCLs respond differently to environmental agents associated with ASD, including trichloroacetaldehyde hydrate and ethylmercury, and enteric short chain fatty acids propionate and butyrate, as compared to ASD LCLs which do not demonstrate these high respiratory rates at baseline.”

Important Nutritional Supplements

Two helpful treatments have already been mentioned: L-carnitine supplementation and ketogenic diets. Studies evaluating ketogenic diets and ASD have found them to be well-tolerated by a majority of children.

A review of four studies found 18% of children saw significant improvement on a ketogenic diet, 40% reported moderate improvement and 32% had mild improvement. Only 8% experienced a worsening of their symptoms.⁷

In cases where the ASD is caused by mitochondrial dysfunction, however, supplementing specific cofactors become important. The good news is this kind of supplementation is very safe and well tolerated. There’s virtually no risk involved, in terms of adverse side effects. Among the most important nutritional cofactors are:⁸

B vitamins, including niacin (B3), riboflavin (B2), biotin (B7), thiamine (B1), pyridoxine (B6), pantothenic acid (B5), methylcobalamin (B12), reduced folate (B9)	Coenzyme Q10 or the reduced form, ubiquinol
L-carnitine or acetyl-L-carnitine	Creatine monohydrate
Alpha-lipoic acid	Vitamin E
Vitamin C	N-acetyl-L-cysteine (NAC)
Zinc	

Benefits of L-Carnitine and CoQ10

Of these, L-carnitine has been most widely studied. As explained in this paper:⁹

“Two medium sized (n = 30, 30) double-blind placebo-controlled studies using L-carnitine treatment (50 mg/kg/d for 3 months and 100 mg/kg/d for 6 months) found that scores on the Childhood Autism Rating Scale (CARS) improved with L-carnitine as compared to placebo with one study finding that greater symptomatic improvement was correlated with a greater increase in blood carnitine levels.

A small (n = 10) 8-week open-label trial of L-carnitine used particularly high doses (up to 400 mg/kg/d in 3 divided doses, maximum of 6000 mg/d) ...

Several parental rated measures showed improvements in behavior and hyperactivity before correction for multiple comparisons and improvements in language correlated with post-treatment blood carnitine levels. Children with ASD and genetic mutations in the carnitine pathway also appear to respond to L-carnitine.”

CoQ10 has also been evaluated in clinical trials. In one, 50 mg of ubiquinol was given twice a day for three months. Children who attained a CoQ10 blood level above 2.5 umol/L saw improvements in communication, interactions, sleep and food rejection.¹⁰

A three-month-long regimen of L-carnitine, CoQ10 and alpha-lipoic acid also produced encouraging results, improving mitochondrial function and ASD-related behavior. When the children discontinued the cocktail, their behavior worsened again.

Autism and Autoimmunity

Previous research also suggests ASD may have an autoimmunity component. As explained in a 2009 paper titled "Autoimmunity in Autism":¹¹

"Increasing evidence of autoimmune phenomena in individuals with autism could represent the presence of altered or inappropriate immune responses in this disorder, and this immune system dysfunction may represent novel targets for treatment.

Furthermore, in recent studies, antibodies directed against the fetal brain have been detected in some mothers of children with autism; these antibodies have the ability to alter behavioral outcomes in the offspring of animal models."

Research^{12,13} published in 2013 found that more than 1 in 10 mothers of autistic children had antibodies associated with autoimmunity that adversely reacted with proteins in the brain of the baby, resulting in ASD. The authors point out that any activation of the mother's immune system "might lead to an increased risk of a child with ASD."

In light of the COVID injection campaign, the possibility of autoantibodies playing a role in ASD is extremely troublesome, as autoimmune diseases are a potential side effect. We don't yet really know the extent of which the SARS-CoV-2 spike protein interacts with the human brain, but it's clearly not beneficial.

Women, in particular, are suffering neurological dysfunction as a result of the COVID shot. How might the spike protein affect offspring? That's still too early to say, but I

suspect we'll come to find that the effect can be devastating.

Toxic Chemicals Implicated in Autism

Not surprisingly, exposure to toxic chemicals in utero has also been identified as a potentially causative factor. According to the National Academy of Sciences, an estimated 3% of all neurobehavioral disorders in children are caused by toxic exposures.

Another 25%, the interaction of toxic exposures and genetic factors is thought to be causative.^{14,15} In 2012, scientists identified 10 chemicals suspected of causing learning disabilities and ASD:¹⁶

Lead	Methylmercury
Polychlorinated biphenyls (PCBs)	Organophosphate pesticides, including glyphosate
Organochlorine pesticides	Endocrine-disrupting chemicals
Automotive exhaust	Polycyclic aromatic hydrocarbons (PAHs)
Brominated flame retardants	Perfluorinated compounds

Of these, glyphosate is a top suspect^{17,18} for the simple reason that exposure to it is so widespread. Research¹⁹ published in 2019 found women exposed to 11 commonly used pesticides – including glyphosate – during pregnancy have a higher risk of having a child diagnosed with autism.

Pregnant women who lived within a 2,000-meter (1.24 miles) radius of a highly-sprayed area were 10% to 16% more likely to have children diagnosed with autism than those who lived farther away. As reported by Time:²⁰

“When they looked at diagnoses of autism spectrum disorder that also came with intellectual disabilities, they found on average 30% higher rates among children who were exposed to the pesticides while in utero. Exposure in the first year of life increased the risk of autism by up to 50% compared to those not exposed to certain pesticides.”

Glyphosate Is Likely a Key Culprit

Stephanie Seneff, Ph.D., who has spent a large portion of her career studying glyphosate, believes it is a significant cause or contributor to autism. She’s shown glyphosate:

- Disrupts your gut microbiome
- Impairs peristalsis – a feature that is exceedingly common in children with autism²¹
- Inhibits bile acid release by impairing gallbladder contraction – Many autistic children have very pale stool, suggestive of low bile acid levels
- Impairs digestive enzymes – Many autistic children also have undigested particles in their stool, which suggests a lack of digestive enzymes. And, indeed, glyphosate affects your digestive enzymes, particularly trypsin, pepsin and lipase

The Roles of GI Inflammation and Maternal Gut Bacteria

Another potential trigger is maternal gut inflammation. Research²² published in early December 2021 found that when pregnant mice were challenged with immune activation, it changed their gut microbiota. This altered microbiota in turn primed the offspring for intestinal inflammation that resulted in neurodevelopmental disorders. MIT News reported the findings, noting:²³

“Infection during pregnancy with elevated levels of the cytokine IL-17a may yield microbiome alterations that prime offspring for aberrant immune responses ...

When a mother experiences an infection during pregnancy and her immune system produces elevated levels of the molecule Interleukin-17a (IL-17a), this can not only alter brain development in her fetus, but also alter her microbiome such that after birth the newborn's immune system can become primed for future inflammatory attacks.

In four studies beginning in 2016,²⁴ study co-senior authors Gloria Choi of MIT and Jun Huhof Harvard University traced how elevated IL-17a during pregnancy acts on neural receptors in a specific region of the fetal brain to alter circuit development, leading to autism-like behavioral symptoms in mouse models.

Their new research ... shows how IL-17a can act to also alter the trajectory of immune system development. 'We've shown that IL-17a acting on the fetal brain can induce autism-like behavioral phenotypes such as social deficits,' says Choi ...

'Now we are showing that the same IL-17a in mothers, through changes in the microbiome community, produces co-morbid symptoms such as a primed immune system.'

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

Here, the COVID-19 pandemic may end up worsening an already terrible trend, as the infection is associated with elevated IL-17a. IL-17a is a key molecule in both the innate and adaptive immunity that helps mobilize your immune system response during early infection. It's been singled out as being involved in the thrombotic and vascular mechanisms of COVID-19 specifically.²⁵

One October 2021 study²⁶ suggested IL-17a acts as a silent amplifier of the COVID-19 immune response, resulting in some of the worst side effects associated with the infection. While pregnant women are universally instructed to get the COVID shot to

protect themselves from the infection, there's every reason to suspect the injection may cause far more harm than the infection itself.

If you get COVID while pregnant, you may or may not have a serious case of it, but if you take the shot, you have a 100% chance of suffering the effects of it, which for many include dramatically elevated levels of inflammation.

From my perspective, getting the COVID jab because you fear what COVID-19 might do to you and/or your baby is actually irrational, as your risk of adverse effects from the shot is exponentially higher than your risk of severe COVID-19 infection.

Autism Is a Multifactorial Condition

A number of other culprits could also be named, such as retroviruses (which the COVID shots can reactivate) and electromagnetic field (EMF) exposure. According to Judy Mikovits, Ph.D., a retrovirus family known as xenotropic murine leukemia virus-related viruses (XMRV) may play a causal role in autism.²⁷

Disturbingly, one source for XMRVs is contaminated childhood vaccines. Mikovits details the history of this in her book, "Plague: One Scientist's Intrepid Search for the Truth About Human Retroviruses and Chronic Fatigue Syndrome (ME/CFS), Autism and Other Disease."

In previous interviews with me, Mikovits has also expressed concern that the COVID shots may activate latent XMRV infection. If it does, we could expect the autism rate to explode at an even more exponential rate than we're seeing already.

I predict the COVID shots will be found to contribute to autism in a number of different ways. But even in the absence of these injections, it's clear that environmental toxicities are wreaking havoc with our children's health.

There's no single answer to this problem. Since many different toxins can contribute, preventing autism must include the elimination of most toxic exposures.

Top suspects include childhood vaccines, the 10 chemicals listed earlier (glyphosate in particular), autoimmune antibodies, gut inflammation, retroviruses and EMF exposure. To this list, I would also add the COVID gene transfer injections, as they can be a source of severe inflammation and autoimmunity, and have been shown to have a direct adverse impact on neurology.

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