Parental avoidance of toxic exposures could help reduce risk of autism, ADHD in children, observational study suggests

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Autism and attention deficit hyperactivity disorder (ADHD) may be preventable if parents avoid toxic exposures and adopt interventions such as environmental house calls, according to a study, published in *Journal of Xenobiotics*, led by researchers from The University of Texas Health Science Center at San Antonio (UT Health San Antonio).

Using a validated, self-administered questionnaire now used worldwide to identify individuals with chemical intolerance—the Quick Environmental Exposure and Sensitivity Inventory (QEESI)—parents and practitioners can determine the risk for each family and learn which exposures to avoid in their own homes where most people spend most of their day, the researchers said.

A population-based survey of nearly 8,000 U.S. adults, using QEESI, found that parents with chemical intolerance scores in the top tenth percentile were 5.7 times as likely to report a child with autism and 2.1 times as likely with ADHD compared with parents in the bottom tenth percentile.

The findings build on a 2015 study by UT Health San Antonio that first linked chemical intolerance in patients with the risk of their children developing autism and ADHD.

"This is the first-ever article in the medical literature showing that chemical intolerance in parents can predict the risk of autism and ADHD in their children, and suggests that reducing exposures prior to and during pregnancy could help prevention," said Claudia S. Miller, MD, MS, professor emeritus with the Department of Family and Community Medicine at UT Health San Antonio. "Up to now, most
interventions have been behavioral or medical, after a child is diagnosed."

Miller is senior author of the study, titled, "Assessing Chemical Intolerance in Parents Predicts the Risk of Autism and ADHD in Their Children." Co-authors include Raymond F. Palmer, Ph.D., and Rodolfo Rincon, MD and specialist, both with the Department of Family and Community Medicine at UT Health San Antonio; and David Kattari, a statistician with the Marilyn Brachman Hoffman Foundation in Fort Worth, Texas.

The researchers note that the study is observational, and further research is needed using controlled trials to confirm causality and further explore the proposed mechanism behind chemical intolerance.

Still, they wrote, "The implications of this study, if confirmed, could be significant for preventive measures and early intervention strategies in families with parental chemical intolerance. We recommend that all prospective parents be assessed for chemical intolerance at an early age."

**Mast cells and autism**

Physician-researcher Miller in 1996 first proposed a two-stage disease process of initiation by exposure and then triggering of symptoms called TILT, for Toxicant-Induced Loss of Tolerance, as the mechanism behind chemical intolerance. She has served as a physician/environmental consultant on exposures.

Her [published papers](#) have explored the impact of pesticides, the Gulf War, breast and other implants, 9/11, toxic molds, combustion products from fires, and indoor air pollutants in so-called "sick" homes, schools and workplaces, including the EPA's own headquarters building in Washington, D.C.
The new study comes amid a backdrop of a 317% increase in the prevalence of autism since 2000, now occurring in one of every 36 children in the country, the researchers note, citing data originating from the Centers for Disease Control and Prevention. And the prevalence of ADHD has risen to one in eight children, also according to the CDC.

Miller and colleagues in 2021 discovered a strong association between chemical intolerance and "mast cells," considered the immune system's first responders that originate in the bone marrow and migrate to the interface between tissues and the external environment where they then reside.

When exposed to "xenobiotics," foreign substances like chemicals and viruses, they can release thousands of inflammatory molecules called mediators. This response results in allergic-like reactions, some very severe. These cells can be sensitized by a single acute exposure to xenobiotics, or by repeated lower-level exposures. Thereafter, even low levels of those and other unrelated substances can cause the mast cells to release the mediators that can lead to inflammation and illness.

In their latest study, the researchers determined that the high chemical intolerance scores among parents of children with autism, coupled with the 2021 finding of mast-cell activation as a plausible biomechanism for chemical intolerance, suggest that:

- The QEESI can identify individuals at increased risk.
- Environmental counseling, such as personalized environmental house calls to assess risks at home, may reduce personal exposures to possible triggers such as pesticides, fragrances and tobacco smoke, particularly during pregnancy and childhood.
- The global rise in autism and ADHD may be due to fossil-fuel-derived and biogenic toxicants epigenetically "turning on" or "turning off" critical mast cell genes that can be transmitted trans-
The researchers conclude that once mast cells are sensitized, diverse xenobiotics that never bothered the person previously and do not bother most people trigger multisystem symptoms that wax and wane over time. And they believe that persistent activation and triggering of mast cells may underlie the brain inflammation in autism.

"The potential role of environmental toxicants in influencing epigenetics and mast cell function is a complex and emerging area of research," they wrote. "Acknowledging the need for further evidence, we hope this study contributes to an improved understanding of the potential role of environmental factors in the global rise of autism and ADHD."

The authors created tools for patients, practitioners and researchers, described in their "TILT Tutorial on Chemical Intolerance, Autism, and ADHD", available along with other resources at https://TILTresearch.org.


Provided by University of Texas Health Science Center at San Antonio
