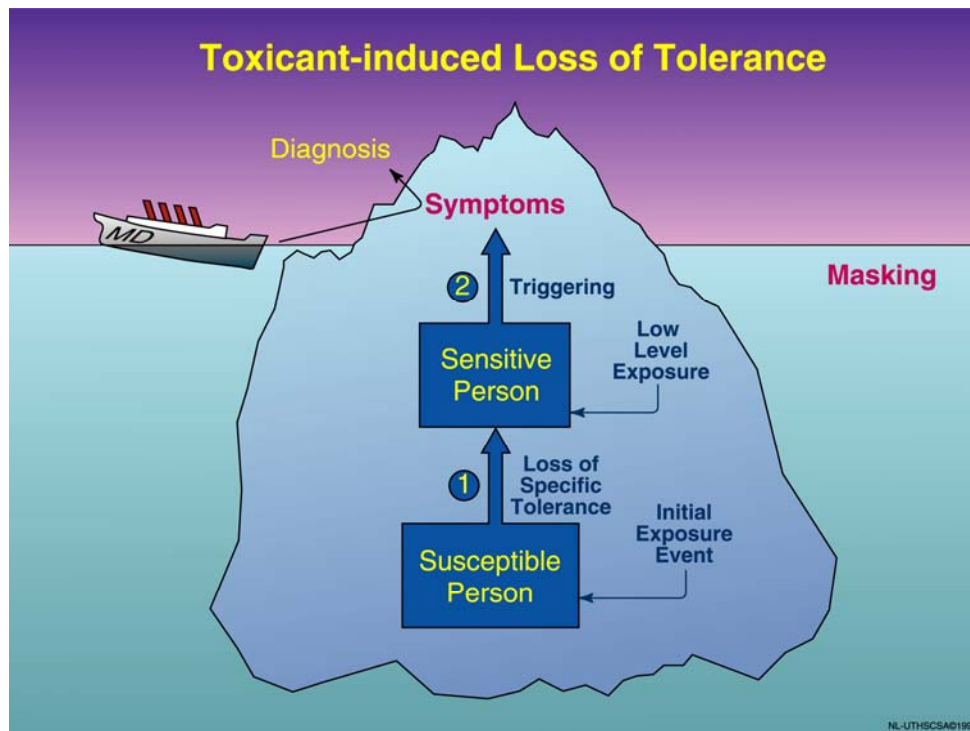


## Environmental Exposures and ASDs: Making the Connection

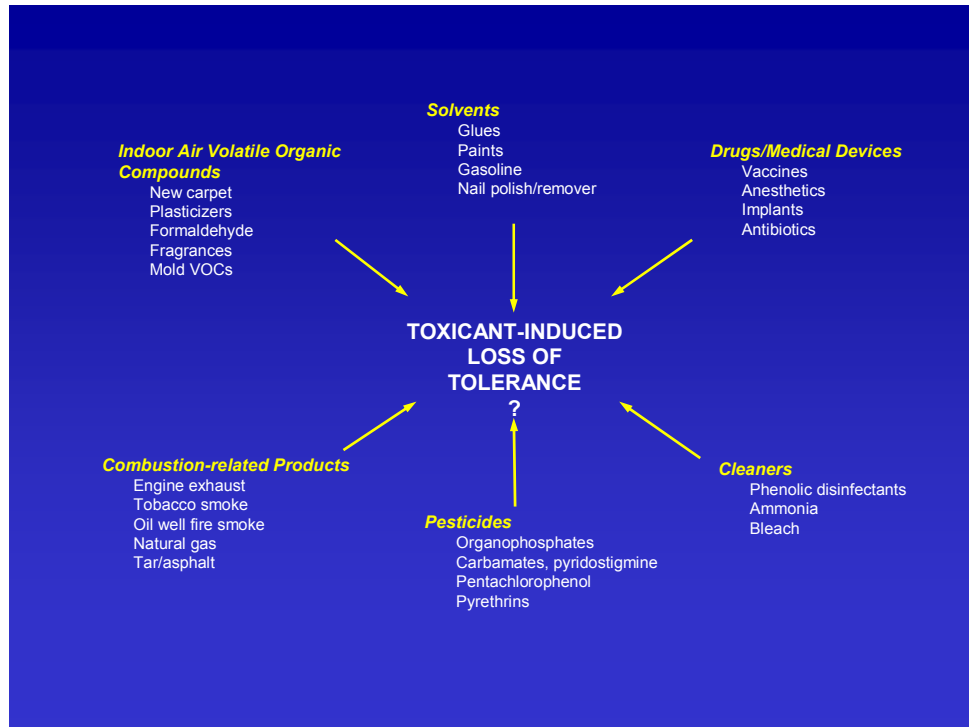
Claudia S. Miller, M.D., M.S.

Anecdotes abound suggesting dramatic improvements among children with ASDs whose parents helped them avoid certain foods or changed their environments—even when parents had been told the situation was hopeless. In contrast, other parents have rigorously removed milk or wheat from their children’s diets and found little or no improvement. Deleting just one common food from a child’s diet can be a difficult proposition. Major dietary and environmental interventions are difficult to implement, disrupt the whole family, impose further limitations on a child already dealing with major challenges, and can be costly. And of course, there is no guarantee of success!

So how can parents determine whether their child will benefit—and how much—from dietary and environmental changes? If such interventions could reverse symptoms, it would certainly be useful to know that ahead of time. On the other hand, if such changes would be unlikely to improve function, it would help to know *that*, thus saving the family long, difficult trial-and-error campaigns. The good news is that there appears to be a sound, scientific way to get these answers—by using a specially designed and constructed residential or hospital facility called an Environmental Medical Unit (EMU). The bad news? Despite years of effort, no such resource currently exists in the United States.



**Figure 1.** Phenomenology of TILT. Illness appears to develop in two stages: (1) Initiation, i.e., loss of prior, natural tolerance resulting from an acute or chronic exposure (pesticides, solvents, indoor air contaminants, etc.), followed by (2) triggering of symptoms by small quantities of previously tolerated chemicals (traffic exhaust, fragrances), foods, drugs, and food/drug combinations (alcohol, caffeine). The physician sees only the tip of the iceberg—the patient’s symptoms—and formulates a diagnosis based on them (e.g., asthma, chronic fatigue, migraine headaches). Masking hides the relationship between symptoms and triggers. The initial exposure event causing breakdown in tolerance may go unnoticed (©UTHSCSA 1996).

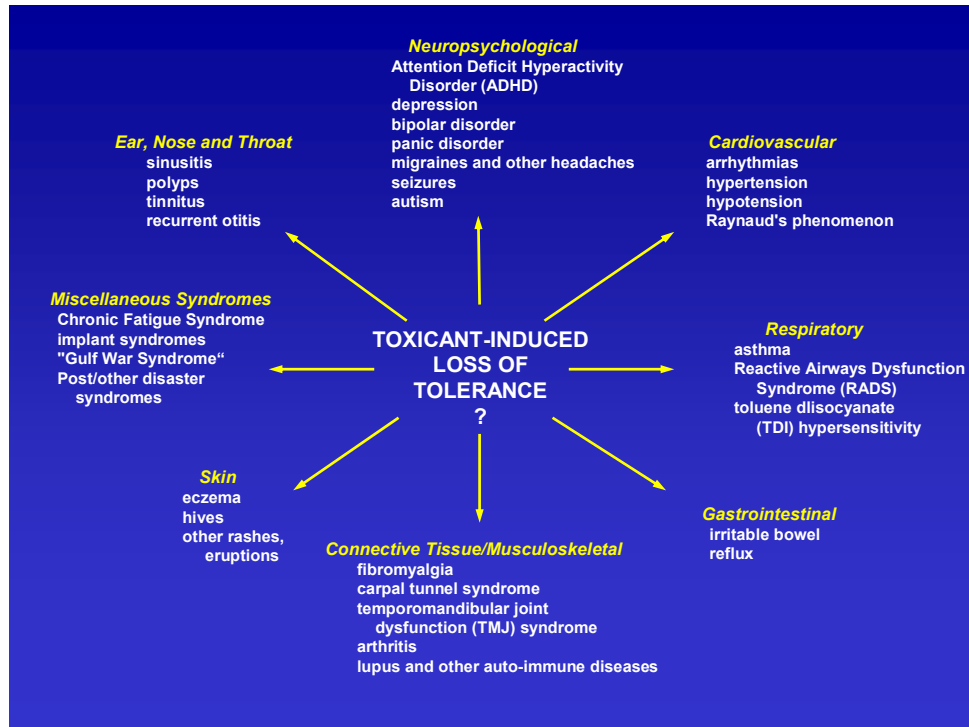


**Figure 2. Potential initiators and triggers for TILT**

## Genetic Susceptibility to Exposures

Our diets and environment have changed enormously over the past 60 years—in the two generations since World War II. Our genes have not changed. Many scientists now believe that environmental exposures at least partly explain the explosion in autism in recent years. Ninety percent of us spend 90% of our days indoors in tightly closed "boxes"—homes, automobiles, daycares, schools, office buildings—exposed to synthetic organic chemicals that did not exist before WWII. Our ability to metabolize and detoxify these exposures depends on our genes, and there are enormous differences among us in that gene-driven metabolic capability. Effects can be significant for adults, but more so for children during critical windows of development—particularly brain development. Some pregnant women, fetuses, and developing children will be especially vulnerable to adverse effects from environmental exposures, depending on their personal detoxification capacity.

Unfortunately, when people are expecting a baby, it is often a prime time for such exposures. A move to a new house, or renovation of a room—with the laudable intent of providing a safer, more spacious, or aesthetically pleasing environment for the child—often brings new furnishings, carpet and paint, fragrances, and pesticides. That means exposures to chemicals during times of maximal susceptibility in terms of neuronal growth and differentiation. We know that exposure to agents such as organophosphate pesticides commonly applied indoors can interfere with the subtle internal chemical signaling necessary for healthy neuronal growth and wiring. These exposures, once they have been identified, are often preventable. But when prevention is no longer an option, the important question becomes, are the adverse effects of exposure irreversible? Must families whose children have autism rely principally on behavioral interventions and drugs



**Figure 3.** Conditions that may have their origins in TILT.

to help their child? Or might the brain simply have become overly susceptible to external stimuli, while still retaining the potential to stabilize and improve? Does the brain have an innate ability to regain normal tolerance for environmental exposures, if protected for awhile?

### **A New Disease Mechanism: A Cause for Optimism**

Toxicant-Induced Loss of Tolerance (TILT) is a two-step disease mechanism we first described in 1996. It is a new paradigm that has emerged in medicine over the past decade, based upon studies of chemically exposed adults in more than a dozen countries. TILT begins following an acute exposure, for example, to a pesticide, or a series of low-level exposures such as indoor air contaminants in a new home or during remodeling (Figure 1). Some who are exposed, presumably those who are more susceptible because of their genes, *lose tolerance for everyday exposures*—exposures that cause most of us no discomfort or harm whatsoever. Thereafter, these everyday exposures, which are often chemically unrelated to the original, TILT-initiating exposure, trigger symptoms. The triggers often include various foods, medications, caffeine, alcoholic beverages (in adults), and common chemicals at low levels, such as cleaning agents, solvents (from gasoline to nail polish remover), fragrances, mothballs, tobacco smoke, and traffic exhaust (Figure 2). Once TILT takes hold, these exposures can trigger myriad symptoms: attention and memory difficulties, extreme irritability, clumsiness, heightened sensitivity to noise, light, touch and other physical stimuli, but also every sort of gastrointestinal problem, allergies, and more (Figure 3). There is currently strong suspicion by some scientists that TILT could result in ASDs.

## Calming the (Symptomatic) Waters

Once TILT has occurred, the person will only improve if ALL triggers are simultaneously removed. Imagine fifty children playing in a pool. The ripples will not subside until every child is out of the water. If even one remains, the water won't be still. Food and environmental exposures appear to act that way—unless all of the triggers are eliminated *simultaneously*, the ripples (symptoms) will continue.

So what can parents of a child with autism do? How can their child be returned to a baseline state, with no exposure to any potential triggers, and then be systematically tested for responses to common triggers in order to nail down exactly what they are?

Again, the answer is the EMU—there is a clear and compelling need for an EMU for research and diagnosis in autism. A short-term stay for the child (up to several weeks) would provide respite for parents while allowing them to observe their child under optimal circumstances (no triggers) and then observe firsthand which exposures elicit symptoms. Once a child's symptom triggers are identified, avoidance can allow recovery and the regaining of tolerance, so that *avoidance does not have to continue for life*.

In *Chemical Exposures: Low Levels and High Stakes* (Ashford N and Miller C, Van Nostrand Reinhold, 2<sup>nd</sup> edition, 1991), we called for the use of an EMU for research on TILT-related conditions. Several years ago, in response to public outcry over illnesses associated with sick buildings and schools, the Japanese government funded four such units. But so far, although a number of bills authorizing a research EMU for the U.S. have been passed by Congress, there still is no EMU in this country. Each time, once the measures reached the appropriations stage, other, unrelated congressional requests took precedence. This is not surprising, given that most people do not understand the profound nature of this problem and the fact that we appear to be dealing with a new theory of disease—paralleling the germ theory—but involving synthetic organic chemicals and their effects. Vested economic interests have resisted research in this area, also not unexpectedly, given the potential market impact. However, once we understand the true nature and extent of a problem, we can find ways to deal with it.

The EMU will allow us to "see" the role of exposures in ASDs and other illnesses, just as the microscope enabled scientists to see "germs" responsible for infectious diseases in the late 1800s. The EMU is the only way I know to eliminate all background exposures simultaneously and optimize a child's environment—to calm the troubled waters.

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*NOTE: the following 2 paragraphs could be set apart in a box or placed here in the text.  
Suggested title for a box: An EMU for Children with Autism*

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The design of an EMU for children with autism must take into account the wide range of patients' special needs, behaviors, ages and cognitive abilities. A pilot program is needed to determine the ideal protocols and unit design considerations, accounting for every aspect

of the environment – from the use of comfortable, non-abrasive cotton garments laundered using scent-free soaps to the selection of lighting (non-fluorescent), color schemes, furnishings, and noise control. The special needs of the child with autism for nurturing, non-threatening, non-clinical-appearing surroundings must be accommodated, while at the same time environmental exposures are carefully controlled so as to achieve the desired diagnostic and therapeutic results.

Typically, the child would spend approximately four weeks in the unit. With a suite-like arrangement, parents or other caregivers would be right there also, with ample opportunity to observe the process and to care for the child. The first week would be devoted to tightly controlling environmental exposures—for example, no perfumes, no smells of harsh cleaners, no out-gassing toys or other objects, no carpet, and a controlled, healthy diet, with only pure spring water to drink. This is intended to return the child to a physiological “baseline,” allowing the lingering effects of prior exposures to recede. It is important to note that it is not unusual for symptoms to worsen to varying degrees at the outset—this transient phenomenon is similar to withdrawal, as the body responds to the cessation of exposures to which it has become accustomed. This phase, if it occurs, passes quickly, and once the slate is clean, environmentally-triggered symptoms should resolve. Once the baseline state has been achieved, a series of carefully controlled, test environmental exposures and single-food challenges are initiated, to document the specific substances to which the child may be sensitive. The purpose of sequestration in an EMU, followed by a comprehensive testing protocol, is to arm parents and caregivers with the information they need to foster continued improvement. They will leave with answers and with effective new strategies to help their children achieve better outcomes.

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Until EMUs are established in the U.S., families, physicians, and researchers will continue to be deprived of the essential tool needed to answer the important questions discussed here—questions whose answers have the potential to explain ASDs and to identify useful, individualized interventions. Interest by physicians, researchers and families in an EMU continues, but little progress has been made. Perhaps if parents of children with autism add their voices to those of others who are anxious for these answers, one day soon this vital tool will become available, and the answers we so desperately need will emerge.