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I am The Sloan Professor of Children's Environmental Health in the Department of Pediatrics and the Director of the Cincinnati Children's Environmental Health Center, Cincinnati Children's Hospital Medical Center and the University of Cincinnati. My research over the past 10 years has focused on how lead and other environmental toxins, such as tobacco, pesticides and mercury, impact children's risk for learning disabilities, behavioral problems and asthma.

## **Background**

Epidemics of overt toxicity following widespread environmental contamination from commercial toxins heralded the discovery of children's enhanced vulnerability to lead, methyl mercury, polychlorinated biphenyls (PCBs), and tobacco [1-5]. Over the past three decades, researchers have found that remarkably low-level exposures to these toxins are linked with less overt symptoms of toxicity—intellectual impairments, learning disabilities, behavioral problems, spontaneous abortions, or preterm births [6-40]. Moreover, there is emerging evidence that decrements in intellectual abilities and low birth weight linked with lead or tobacco are, for a given increment of exposure, greater at lower levels than those found at higher levels [10, 41-43]. The consequences of exposure to many other chemicals or mixtures of chemicals, such as insecticides—

chemicals oftentimes specifically designed to be toxic—are largely unknown [33-35, 44]. Many of these chemicals or their metabolites are routinely found in the blood and body fluids of pregnant women and children [45]. Collectively, these findings make it imperative to develop regulations to ensure that children and pregnant women are adequately protected from exposures to environmental chemicals.

### **Children’s Vulnerability to Environmental Toxins**

The developing fetus and young child is particularly vulnerable to certain environmental chemicals [46-50]. Critical neurodevelopmental processes occur in the human central nervous system during fetal development and in the first three years of life. These processes include cortical functional differentiation, synaptogenesis, myelination, and programmed apoptosis [46]. Children’s exposure to environmental chemicals is insidious. Environmental chemicals covertly enter a child’s body transplacentally during fetal development or by direct ingestion of house dust, soil, and breastmilk and other dietary sources during early childhood. [51-56]. Our ability to directly measure the actual levels of environmental chemicals in human tissues and body fluids using biologic markers (biomarkers) are enabling scientists to more effectively link exposures to environmental chemicals with disability or disease [57].

### **The Prevalence of Diseases and Disabilities Linked to Environmental Toxins**

Based on parental reports, one in six United States children have one or more developmental disabilities, from a subtle learning disability to overt behavioral or emotional disorders [58]. Exposures to environmental chemicals have been linked with higher rates of mental retardation, intellectual impairment, and behavioral problems, such as conduct disorder

and attention deficit hyperactivity disorder [16-27, 30-31, 36-43, 59-61]. Based on the recent NHANES analyses, 8.2% of US children and adolescents have ADHD and 10.6% have a learning disability.

One in ten US babies is born preterm [62-63]. Preterm birth, defined as birth at less than 37 weeks of gestation, is a major determinant of infant mortality and morbidity throughout childhood [62-64]. Exposures to environmental chemicals such as lead, tobacco smoke, and DDT have been linked with an increased risk for spontaneous abortion, low birth weight, or preterm birth [6, 9, 10, 13-15, 28, 32, 65-66].

The rate of occurrence for many of these diseases and disabilities has been rising, as has treatment for attention deficit hyperactivity disorder and depression in children [62-63, 67-70]. In some cases, there is too little information to make any conclusions about trends in learning disabilities. The first national estimates for learning disabilities and ADHD were based on NHANES surveys conducted from 1999-2001. Thus, it is critical to improve our surveillance for learning disabilities and behavioral problems in US children and adolescents.

Multiple risk factors, including both genetic and environmental influences, interact in complex and often unknown ways to cause disease and disability in children. But efforts can be undertaken to prevent or reduce environmental exposures linked to disease without full elucidation of the underlying mechanism [71]. Thus, conducting some sort of test to identify pesticides and industrial chemicals that could cause reproductive or neurobehavioral toxicity before the chemical reaches widespread use is essential to protect pregnant women and children.

## **The European Framework: “REACH”**

In 2001, the European Commission affirmed that the European Union’s legislative framework did not provide adequate information about the adverse effects of chemicals on human health, and that when hazards were identified the regulatory agencies were slow to assess risks and to introduce measures to reduce those risks [78]. Indeed, chemical manufacturers are not required to “prove” that a chemical is safe before marketing it. The European Commission proposed a new regulatory framework for chemicals, REACH (Registration, Evaluation, and Authorization of Chemicals) [78, 79].

Under REACH, chemical manufacturers would have to assume a much greater burden for showing the lack of harm from use of their products. Specifically, REACH would require both European and non-European manufacturers doing business in Europe to submit more extensive toxicity data for about 30,000 chemicals on the market, including reproductive and DNT data for those chemicals produced in highest quantity. Chemicals found to be hazardous would be subject to an authorization procedure to show that they can be used safely or that there are no safer alternatives. This registration process would not guarantee that chemicals are safe, but it is a step in the right direction.

## **Steps to Protect Children from Environmental Toxins**

Children must be better protected from both new and existing chemicals that are known or possible toxins [49]. To protect children from existing toxins, such as lead, mercury, and tobacco, the US EPA and FDA need more authority and resources to regulate and reduce

emissions and exposures. Under our current system, efforts to enhance regulations to protect children from confirmed toxins are costly and protracted. Indeed, countless communities across the globe suffer from widespread environmental contamination with lead, PCBs, arsenic and other environmental chemicals. If there is any lesson from our experience with environmental toxins, it is that we need to identify environmental chemicals that are toxic before they are marketed or widely disseminated.

For new commercial chemicals, toxicity testing in animals should be required before they are marketed. For all new chemicals, including pesticides, extensive premarket testing should be required in multiple animal species of both sexes and at different developmental stages. These tests should be designed to have adequate statistical power to detect subtle differences within the ranges of exposure that occur in human populations. If implemented, these testing requirements would represent a dramatic departure from existing regulations, while providing a powerful incentive for industry to develop less toxic chemicals.

## **Conclusion**

It is time to acknowledge that the existing regulations for toxicity testing of environmental chemicals are inadequate to safeguard pregnant women and children. Until a formal regulatory system is developed to effectively screen and identify new and existing chemicals that are toxic to pregnant women and children, we are left to await the next epidemic to warn us about an environmental disaster. Unfortunately, by then we will have once again fouled our nest [81].

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